Children Health and Air Pollution

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The developmental origins of health and disease (DOHaD) hypothesis links adverse fetal exposures with developmental mal-adaptations and morbidity later in life. Short- and long-term exposures to air pollutants are known contributors to health outcomes. Air pollution is an established risk factor for morbidity and mortality that affects the general population. The developmental origins of health and disease (DOHaD) hypothesis states that adverse fetal, infant, and childhood growth patterns are causally linked to disease development in adulthood. The literature on cardiovascular and metabolic, respiratory, allergic, and neuropsychological health outcomes, from prenatal development through early childhood, associated with early-life exposures to outdoor air pollutants, including traffic-related and wildfire-generated air pollutants are summarized.

Keywords: air pollution ; wildfire smoke ; prenatal ; early life ; developmental health

1. Introduction

Air pollution is an established risk factor for morbidity and mortality that affects the general population [1][2]. The developmental origins of health and disease (DOHaD) hypothesis states that adverse fetal, infant, and childhood growth patterns are causally linked to disease development in adulthood [3][4]. Prenatal or early-childhood environmental exposures predispose the fetus or child to such mal-adaptations in growth and increase the risk of disease in adulthood, in accordance with the DOHaD hypothesis [5][6]. Two recent reviews on ambient and traffic-related air pollution have linked air pollution exposures in neonates and children with increased cardiovascular morbidity [7] and asthma development [8]. Additionally, prenatal exposure to particulate matter (PM) has been associated with higher odds of respiratory and all-cause infant mortality [9]. However, the literature on early-life air pollution exposures has not been reviewed comprehensively with respect to a broad spectrum of fetal and child health outcomes. Additionally, most reviews have focused on outdoor, ambient air pollution without specific source apportionment [7][8]. No reviews have been conducted on emerging sources of air pollution, such as traffic-related air pollution from vehicle emissions or wildfire-generated air pollution. This entry reviewed and summarized the literature on cardiovascular and metabolic, respiratory, allergic, and neuropsychological health outcomes, from prenatal development through early childhood, associated with early-life exposures to outdoor air pollutants, including traffic-related and wildfire-generated air pollutants.

2. Cardiovascular and Metabolic Outcomes

Researchers' search yielded 81 studies on cardiovascular and metabolic outcomes, and the results support that prenatal and postnatal air pollution exposures are both associated with an increased risk of adverse outcomes. Prenatal exposure to ambient air pollution, including particular matter with an aerodynamic diameter less than 2.5 or 10 μm ($PM_{2.5}$ and $PM_{10,}$ respectively), sulfur dioxide (SO_2), nitrogen dioxide (NO_2), or ozone (O_3), has been consistently associated with reduced or low birth weight across various populations and geographic locations [10][11][12][13][14][15][16][17][18][19][20][21][22][23][24][25][26][27][28][29][30][31][32][33][34][35][36][37][38][39][40]. Studies that have estimated traffic-related air pollution (TRAP) or roadway proximity using geographic information system or land use regression models similarly report an association between prenatal TRAP exposure and low birth weight [12][38][41][42][43]. Prenatal exposures to ambient $PM_{2.5}$, PM_{10}, SO_2, and O_3 have also been associated with an elevated risk of macrosomia [44]. Although the results differ in the direction of birth weight deviation, low and high birth weight similarly reflect abnormal metabolism or nutritional transfer to the fetus, and they are both risk factors for developing cardiometabolic disorders [45][46][47][48]. Some studies have examined specific constituents of particulate matter and found that birth weight is inversely correlated with prenatal exposures to constituents, including zinc, sulfur, elemental carbon, silicon, titanium, and aluminum. [12][13][49][50] Basu et al. reported that the strongest associations were found with constituents that are common markers of traffic pollution, industrial pollution, oil combustion, and alloy production [12]. In addition to birth weight, some studies have reported that ultrasound measures of fetal growth during gestation are negatively associated with prenatal exposures to particular matter with an aerodynamic diameter less than 1 μm (PM_1), $PM_{2.5}$, $PM_{10,}$ SO_2, NO_2, or O_3 [22][51][52][53][54][55]. Exposures to traffic-related

and ambient air pollutants, such as $PM_{2.5}$, $PM_{10,}$ O_3, and NO_x, have been consistently associated with increased odds of preterm birth [16][18][25][34][56][57][58]. However, one study did not find significant associations between NO_2 exposure during pregnancy and preterm birth or low birth weight [59]. Early-life wildfire smoke exposure has also been associated with preterm birth and birth weight. Evidence from three studies demonstrates that pregnant women with addresses in wildfire-affected areas during gestation were at a greater risk of preterm birth or low newborn birth weight [60][61][62], while one study found a higher average birth weight in exposed male infants [63].

The results are limited on critical exposure time windows because many studies averaged air pollution exposure across an entire pregnancy or only examined exposure at one time point. Of the studies that did analyze trimester-specific associations, most found that exposures during the second [11][15][19][21][25][36][40][58][60][61] or third [11][14][16][17][31][32][33][34][36][37][39][49][58][61] trimesters had statistically significant associations with birth weight or preterm birth. A few studies report susceptibility during the first trimester to carbon dioxide (CO_2), NO_2, or O_3 exposures, particularly within the first month of pregnancy [16][21][28][52]. One study that associated prenatal PM_{10} exposure with term low birth weight attributed the association to conception month and first trimester exposures [27]. One study on wildfire-related $PM_{2.5}$ exposure found that full gestation and second trimester exposures were associated with preterm birth, while first trimester exposure was associated with decreased birth weight [60].

The literature also supports a link between prenatal air pollution exposure and abnormal weight and growth trajectory after birth. Prenatal and early postnatal exposures to ambient $PM_{2.5,}$ $PM_{10,}$ NO_2, O_3, SO_2, and carbon monoxide (CO) have been associated with deviant growth trajectories, represented by anthropometric measures, in infancy and childhood [40][64][65][66][67][68][69][70][71]. Obesity-related parameters (higher BMI Z-score, levels of adipokines, and higher risk of obesity development) in newborns and children have been positively associated with prenatal highway proximity, TRAP exposure, or ambient PM, NO_2, O_3, and polycyclic aromatic hydrocarbons (PAH) exposures [67][71][72][73][74][75][76], as well as childhood exposures to TRAP and ambient $PM_{2.5}$ and NO_2 [77][78]. However, one study did not find an association between ambient air pollution or nearby traffic load during the first four years of life and childhood obesity, waist circumference, or cholesterol at ages four or eight [79].

Epidemiological studies also support a link between air pollution levels and the childhood risk of metabolic disorder, including diabetes and hypertension. Several studies reported that $PM_{2.5}$ exposure during pregnancy was associated with systolic hypertension in newborns [80], and microvascular changes [81][82] and elevated blood pressure in children [83][84][85]. Prenatal TRAP, $PM_{2.5}$, PM_{10}, and NO_2 exposures have been associated with a significant increase in cord blood insulin, adiponectin, and leptin levels, [74][75][86] with second trimester exposures having the largest effect [86]. Similarly, proximity to a major road and higher traffic-related PM_{10} and NO_2 levels at the birth address, estimated by land use regression models, have been positively associated with childhood insulin resistance [87]. Prenatal $PM_{2.5}$ exposure [88] and childhood TRAP exposure [89] have been positively associated with childhood development of risk factors for metabolic syndrome, such as increased hemoglobin A1c and systolic blood pressure. A study on diabetic and healthy children that were randomly selected from a pediatric database at Loma Linda University found that childhood O_3 exposure prior to diagnosis was significantly higher in children with type 1 diabetes than in healthy controls, and pre-diagnosis PM_{10} exposure was significantly higher in children with diabetes diagnosed before age five, when compared with healthy controls [90]. In summary, prenatal and childhood exposures to ambient and traffic-related air pollution have been consistently associated with preterm birth, deviant birth weight, childhood obesity, and insulin resistance, all of which have long-term impacts on cardiometabolic health in adults. Researchers did not find any studies investigating early-life wildfire exposures in association with cardiometabolic outcomes in infants and children.

3. Respiratory and Allergic Outcomes

Researchers' search resulted in 57 studies on respiratory outcomes, and the results support a link between prenatal and early-childhood air pollution exposures and respiratory morbidity. Prenatal air pollution exposure has been associated with decreased lung function during infancy and childhood [91]. Higher PM_{10} exposure during pregnancy—especially during the second [92] or third [93] trimester—was associated with worsened infant lung function, represented by increased minute ventilation, higher respiratory rate, and tidal breathing flow; in addition, preterm infants showed greater susceptibility to PM_{10}-associated lung inflammation [92]. A different study reported an inverse association between CO exposure during pregnancy and infant lung function [94]. A number of studies have examined the relationship between prenatal exposure to ambient air pollution and pulmonary outcomes in childhood: prenatal exposures to ambient $PM_{2.5}$, $PM_{10,}$ NO_2, $NO_{3,}$ and benzene have been associated with worsened childhood lung function parameters, including forced expiratory volume, forced expiratory flow, airway reactance, and peak expiratory flow [95][96][97][98][99][100][101][102][103][104][105]. There is also evidence that proximity to major roads, childhood $PM_{2.5}$ and black carbon exposures [106], and childhood NO_2 exposure [101] is associated with worsened lung function in mid-childhood (median age 7).

The current literature presents strong evidence that prenatal air pollution exposure also increases the risk of respiratory and allergic disorders. The risk of newborn tachypnea, asphyxia, and respiratory distress has been associated with increased prenatal exposures to ambient PM, CO, NO, and O_3 [107]. Epidemiological studies have demonstrated that prenatal exposures to ambient NO_2, SO_2, $PM_{2.5}$, PM_{10}, and ultrafine particles (with aerodynamic diameter < 0.1 µm) were associated with increased respiratory tract infections in infancy [108][109][110] and asthma, wheezing, and rhinitis in childhood [109][111][112][113][114][115][116][117][118][119][120]. One study that assessed respiratory health at 6 or 18 months found no association between prenatal land use regression-modeled NO_2 exposure and the incidence of lower respiratory tract infections or wheeze [121]. However, a different study that similarly used NO_2 exposure estimates to quantify traffic-related air pollution reported that TRAP exposure during the third-trimester of pregnancy or first year of life was significantly associated with allergic rhinitis, and the association was strongest for male children aged 3 or 4 years old [122].

The literature also presents a consistent relationship between childhood asthma or wheeze and early-childhood exposures to ambient air pollution [102][114][119][120][123][124][125][126][127][128][129][130][131] or traffic-related air pollution, estimated by a land use regression model or road proximity [132][133][134][135]. Postnatal exposures to ambient PM_{10}, NO_2, and O_3 have been associated with eczema and allergic symptoms in children [126][129]. Furthermore, several studies demonstrated that the risk of respiratory infection, such as pneumonia, rhinitis, or bronchitis in infants and children, was associated with increased short-term exposure to ambient PM_{10}, O_3, NO_x, and SO_2 [130][136][137][138], and long-term exposure to TRAP and ambient $PM_{2.5}$, PM_{10}, NO_x, and PAH [115][129][139][140][141][142][143][144][145][146]. Still, one study did not find an association between childhood asthma incidence in kindergarten-aged children and exposure to ambient air toxics at two years, using estimates from the 2002 National Air Toxics Assessment [147]. Researchers found only one study that examined early-life respiratory outcomes in association with wildfire-generated air pollution. This entry reports an increase in respiratory visits for children aged 0–5 in association with acute $PM_{2.5}$ exposure during a wildfire event [148]. In summary, prenatal and early-childhood exposures to TRAP and ambient air-pollution have been consistently associated with worsened lung function and asthma, wheeze, and respiratory infections in infancy or childhood. More research is needed on early-life respiratory and allergic outcomes in association with wildfire exposures.

4. Neuropsychological Outcomes

Researchers' literature search yielded 26 studies on neuropsychological outcomes. While early-life air pollution exposure has been less studied in children with respect to neuropsychological health, the current data suggest there is an association with adverse neurodevelopment. Prenatal and neonatal exposures to both ambient and traffic-related air pollutants, including PM, NO_2, SO_2, and black carbon, have been associated with impaired cognitive, motor, behavioral, and language development during infancy and early childhood [149][150][151][152][153][154][155][156][157][158][159][160]. Prenatal exposures to ambient $PM_{2.5}$, PM_{10}, and PAH have been associated with lower IQ [161][162][163] and worsened attention and memory [162] in children aged 4–7 years old. Several studies found greater odds of autism spectrum disorders (ASD) in children with higher prenatal and perinatal exposures to ambient NO, NO_2, $PM_{2.5}$, PM_{10}, O_3 and near-roadway air pollution, or TRAP [164][165][166][167][168][169][170][171][172]. TRAP and ambient $PM_{2.5}$ and O_3 exposures in the first two years of life have also been associated with an increased ASD risk [165][169][170]. Childhood exposures to near-residence traffic density, as well as the traffic-related air pollutants NO_2, black carbon or elemental carbon, and fine and ultrafine PM, have been positively associated with cognitive and behavioral deficits, hyperactivity, and changes in white matter volume among children [156][173][174]. In summary, ambient and traffic-related air pollution exposures during pregnancy and the first two years of life have been consistently associated with ASD and worsened neuropsychological parameters, including motor and cognitive development. Fewer studies have examined the neuropsychological outcomes associated with childhood air-pollution exposures, and no studies have examined the neuropsychological outcomes in association with early-life wildfire exposure.

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