



Air pollution and childhood obesity

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Childhood obesity is a global health concern. Air pollution is also a crucial health threat, especially in developing countries. Over the past decade, a number of epidemiologic and animal studies have suggested a possible role of pre- or postnatal exposure to air pollutants on childhood obesity. Although no clear mechanism has been elucidated, physical inactivity, oxidative stress, and epigenetic modifications have been suggested as possible mechanisms by which obesity develops due to air pollution. In this review, we summarize and review previous epidemiologic studies linking air pollution and childhood obesity and discuss the possible mechanisms underlying air pollution-induced obesity based on *in vivo* and *in vitro* evidence.

Key words: Air pollution, Particulate matter, Pediatric obesity

Key message

Questions: What are the possible effects of air pollution on the occurrence of childhood obesity and what are the mechanisms?

Finding: Epidemiologic studies suggest that air pollutants might act as obesogens in the pediatric population, and their possible mechanisms include oxidative stress, physical inactivity, and epigenetic modulation.

Meaning: This paper reviews updated information on air pollution, one of the modifiable environmental factors in childhood obesity.

Introduction

The global prevalence of childhood obesity has increased almost 8–10 times over the last 30–40 years.^{1,2)} Childhood obesity can lead to various comorbidities, including type 2 diabetes, hypertension, nonalcoholic fatty liver disease, cardiovascular disease, and even cancer in later life.³⁾ A rapid increase in the prevalence of obesity has occurred with the markedly increased production of industrial chemicals, suggesting potential causative links.⁴⁾ In particular, as more than 90% of children worldwide live in an environment with air pollution levels above the World Health Organization guideline, the link between air pollution and childhood obesity is drawing increasing attention.⁵⁾ A number of

studies on the effects of air pollutants on childhood obesity were reported in the 2010s. Here we review epidemiologic studies on the association between air pollution and childhood obesity and speculate on the underlying mechanisms.

Air pollutants and major sources of exposure

Ambient air pollution is mainly caused by the combustion of fossil fuels, waste incineration, industrial/agricultural processes, and natural processes including thunderstorms and volcanic eruptions.⁶⁾ Household air pollution is primarily generated by the incomplete combustion of fossil fuels during cooking, heating, and lighting. Other household air pollutants include tobacco smoke, mold spores, building materials, and volatile organic compounds (VOCs).⁶⁾ The air pollution sources vary among regions according to industrialization degree. In urban areas, the combustion of fossil fuels for energy production is the primary source of air pollution, while in rural areas, the main sources of air pollution are pollutants generated in the household and from incineration for heating, cooking, and waste disposal.⁷⁾

Major air pollutants include particulate matter (PM), ozone (O₃), carbon monoxide (CO), sulfur dioxide (SO₂), nitrogen dioxide (NO₂), and polycyclic aromatic hydrocarbons (PAHs). Primary air pollutants are emitted from direct sources (e.g., factories, construction sites, fires, cars, and roads) and secondary pollutants are formed by chemical reactions with other substances in the air.⁸⁾ Possible sources of air pollutants are presented in Table 1.

Among air pollutants, PM poses the greatest health concern since it is a complex heterogeneous mixture of all kinds of air pollutants (e.g., nitrates, sulfates, elemental and organic carbon, VOCs, and PAHs), biological compounds (e.g., endotoxin, mold, pollen), and metals (e.g., iron, nickel, copper, and zinc).⁹⁾ There is increasing evidence that the health impact of PM is dependent on its chemical composition.¹⁰⁾

Depending on the aerodynamic diameter of the particles, PM is classified as coarse (PM₁₀), with a diameter of <10 microns, or fine (PM_{2.5}), with a diameter of <2.5 microns.⁸⁾ Fine particles generally penetrate the lower respiratory tract more easily, while

Table 1. Common sources of major air pollutants

Pollutant	Ambient	Household
Primary pollutants		
CO	Incomplete combustion of carbon-containing fuels, vehicular exhaust, and photochemical reactions in the atmosphere	Gas stoves and tobacco smoke
SO ₂	Industrial activities that process sulfur-containing fuels, and motor vehicles emissions	Combustion of coal/oil
NO ₂	Power plants and motor vehicles emissions	Gas and kerosene heaters, and tobacco smoke
PAHs	Incomplete combustion of organic materials (e.g., coal, oil, petrol, and wood)	Wood stoves, barbecues, and tobacco smoke
VOCs	Fossil fuels and motor vehicles emissions	Paints, disinfectant, air-fresheners, and photocopy machines
Secondary pollutants		
PM	Directly emission or transformation of gaseous emissions (e.g., SO ₂ , NO ₂ , VOCs) by motor vehicle combustion of solid/fossil fuels, and forest fires	Gas/wood stoves, and gas space heaters
O ₃	The reaction of NO ₂ and VOCs in the presence of sunlight	None

CO, carbon monoxide; NO₂, nitrogen dioxide; O₃, ozone; PAHs, polycyclic aromatic hydrocarbons; PM, particulate matter; SO₂, sulfur dioxide; VOCs, volatile organic compounds.

coarse particles tend to lodge in the upper respiratory tract.⁸⁾ Most epidemiologic studies have consistently reported that PM_{2.5} is the most harmful fraction.¹¹⁾

Association of air pollution and childhood obesity in epidemiologic studies

1. Prenatal exposure to air pollutants

Human epidemiologic studies on the relationship between prenatal exposure to air pollutants and childhood obesity are listed in Table 2. Longitudinal studies from the U.S. Project Viva cohort consistently reported that traffic density and roadway proximity during pregnancy or at delivery are associated with obesity parameters including body mass index (BMI) and fat mass in children aged 6 months to 10 years.¹²⁻¹⁴⁾ This study group also suggested a possible impact of prenatal air pollution exposure on reduced birth weight. However, other studies on the association between individual traffic-related air pollutants and childhood obesity have not verified this association. Exposure to nitrogen oxides (NO_x) and elemental carbon was not associated with childhood obesity in cohort studies from Sweden, the USA, and Hong Kong.¹⁵⁻¹⁷⁾ SO₂ exposure was even negatively related to BMI in adolescent boys.¹⁷⁾

A relatively large number of studies have examined the effects of exposure to PM versus other air pollutants on childhood obesity. Most cohort studies from the USA and Hong Kong reported a null impact of PM_{2.5} and PM₁₀ exposure during pregnancy on obesity parameters in children and adolescents.^{12-14,16,17)} Only 2 cohort studies from Boston, Massachusetts, reported a weak association between PM during pregnancy and obesity parameters including BMI at 2-9 years of age and waist-to-hip ratio at 4 years of age.^{18,19)}

Prenatal exposure to tobacco smoke, a representative source of household air pollution, is reportedly related to an increased risk of overweight at ages 3 and 7 years in large-scale national cohort studies from the USA and the UK.^{20,21)} A small-scale study from

Canada reported a null effect of tobacco smoke on BMI in 5-year-old children.²²⁾ PAHs, which are known to be highly correlated to tobacco smoke exposure, were also associated with childhood BMI in 2 cohort studies from New York. In these studies, exposure to PAHs measured by personal air monitoring during pregnancy was positively correlated with BMI in children aged 5-14 years in African-American and Hispanic children.^{23,24)}

2. Postnatal exposure of air pollutants

The impacts of postnatal air pollutant exposure on childhood obesity identified through human epidemiologic studies are presented in Table 3. As with prenatal exposure studies, a positive correlation between residential traffic density/roadway proximity in childhood and BMI at 4-8 years was demonstrated by 2 large cohort studies conducted in Southern California.^{25,26)} However, a school-based cross-sectional study reported that the positive association between the presence of arterial roads around school and the obesity rates in elementary schoolers was not statistically significant after the adjusting for crime rates and economic levels around the schools.²⁷⁾ However, other studies on the association between individual traffic-related air pollutants and childhood obesity have not confirmed this association.

Of note, most studies of the association between NO_x, a major traffic-related air pollutant, and childhood obesity reported a statistically significant positive correlation. In 3 cohort studies performed in the USA, teenage exposure to NO_x concentrations was positively correlated with BMI gain until 18 years of age, while 1-year-old exposure to NO_x concentrations was positively associated with BMI gain until 10 years of age.^{16,28,29)} Recent studies published in the Netherlands and Spain have also reported that residential NO₂ concentrations are associated with a higher risk of overweight in childhood and adolescence.^{30,31)} However, in an Italian cohort study, residential NO₂ concentrations measured at birth and age 4 were not associated with obesity at 4 and 8 years of age.³²⁾

Study results on the link between postnatal PM exposure and childhood obesity are inconsistent. In 2 cohort studies from

Table 2. Association between prenatal exposure to air pollution and childhood obesity in human epidemiologic studies

Study	Country	Study design	Study population			Obesity parameter	Air pollutant	Direction of relationship with obesity (+/-/0)	Findings
			Sample size (n)	Age at exposure (yr)	Age at outcome (yr)				
Traffic density/proximity									
Fleisch et al. ¹⁴ 2015	USA	Cohort	2,115	3rd trimester	0-0.5	Weight-for-length	Traffic density	(+)	Negative association with fetal growth, but a positive association with obesity at the age of 6 mo
Fleisch et al. ¹³ 2017	USA	Cohort	1,418	At delivery	3.3 7.7	BMI, skinfold thickness, WC, fat mass	Roadway proximity	(+)	Positive association with fat mass at early & mid-childhood
Fleisch et al. ¹² 2019	USA	Cohort	1,649	3rd trimester	0.5-10	BMI	Traffic density	(+)	Positive association with obesity at the age of 0.5-10
NO_x/SO₂/elemental carbon									
Frondelius et al. ¹⁵ 2018	Sweden	Cohort	5,815	Entire pregnancy	4	BMI	NO _x	(0)	No association with overweight/obesity at the age of 4
Kim et al. ¹⁶ 2018	USA	Cohort	2,318	Entire pregnancy	10	BMI	NO _x	(0)	No association with attained BMI at the age of 10
Huang et al. ¹⁷ 2019	Hong Kong	Cohort	8,298	Entire pregnancy	9-15	BMI	NO _x SO ₂	(0) (-)	No association with BMI at the age of 9-15 Negative association with BMI at the age of 12-15 (only in boys)
Sears et al. ⁴⁹ 2019	USA	Cohort	657	Entire pregnancy	7-8	BMI	Elemental carbon	(0)	No association with BMI at the age of 7-8
PM									
Fleisch et al. ¹⁴ 2015	USA	Cohort	2,115	3rd trimester	0-0.5	Weight-for-length	PM _{2.5}	(0)	No association with obesity at the age of 6 mo
Fleisch et al. ¹³ 2017	USA	Cohort	1,418	3rd trimester	3.3 7.7	BMI, Skinfold thickness, WC, fat mass	PM _{2.5}	(-)	Negative association with BMI and fat mass at early & mid-childhood
Chiu et al. ¹⁹ 2017	USA	Cohort	239	10-29 weeks gestation	4	WHR BMI, skinfold thickness, fat mass	PM _{2.5}	(+)	Positive association with WHR at the age of 4 (only in girls) No association with other adiposity measures
Mao et al. ¹⁸ 2017	USA	Cohort	1,446	Entire pregnancy	2-9	BMI	PM _{2.5}	(+)	Positive association with childhood overweight or obesity
Kim et al. ¹⁶ 2018	USA	Cohort	2,318	Entire pregnancy	10	BMI	PM _{2.5}	(0)	No association with attained BMI at the age of 10
Fleisch et al. ¹² 2019	USA	Cohort	1,649	3rd trimester	0.5-10	BMI	PM _{2.5}	(0)	No association between prenatal PM _{2.5} exposure and BMI outcomes at any age
Huang et al. ¹⁷ 2019	Hong Kong	Cohort	8,298	Entire pregnancy	9-15	BMI	PM ₁₀	(0)	No association with BMI at the age of 9-15
Smoking									
Hawkins et al. ²⁰ 2009	UK	Cohort	13,188	3-Month gestation	3	BMI	Smoking	(+)	Positive association with overweight at the age of 3
Dancause et al. ²² 2012	Canada	Cohort	111	Preconception, entire pregnancy	5.5	BMI	Smoking	(0)	No association with obesity at the age of 5.5
Wen et al. ²¹ 2013	USA	Cohort	21,063	3rd trimester	7	BMI, body weight	Smoking	(+)	Positive association with overweight at the age of 7
PAH									
Rundle et al. ²³ 2012	USA	Cohort	702	3rd trimester	7	BMI, fat mass	PAHs	(+)	Positive association with obesity at the age of 7
Rundle et al. ⁵⁰ 2019	USA	Cohort	535	3rd trimester	5-14	BMI	PAHs	(+)	Positive association with childhood BMI at the age of 5-10

Direction of relationship with obesity: (+), factor related to greater childhood obesity; (-), factor related to decreased childhood obesity; (0), no relationship with childhood obesity

BMI, body mass index; NO_x, nitrogen oxides; PAHs, polycyclic aromatic hydrocarbons; PM_{2.5}, particulate matter <2.5 microns in diameter; PM₁₀, particulate matter <10 microns in diameter; SO₂, sulfur dioxide; WC, waist circumference; WHR, waist-to-hip ratio.

Table 3. Association between postnatal exposure to air pollution and childhood obesity in human epidemiologic studies

Study	Country	Study design	Study population			Obesity parameter	Air pollutant	Direction of relationship with obesity (+/-/0)	Findings
			Sample size (n)	Age at exposure (yr)	Age at outcome (yr)				
Traffic density/proximity									
Jerrett et al. ²⁵⁾ 2010	USA	Cohort	2,889	9–10	18	BMI	Traffic density	(+)	Positive association with attained BMI over 8 study years
Jerrett et al. ²⁶⁾ 2014	USA	Cohort	4,550	5–7	10	BMI	Traffic density	(+)	Positive association with attained BMI at the age of 10
Amram et al. ²⁷⁾ 2019	USA	Cross-sectional	10,327	10–11	10–11	BMI	Arterial road exposure	(0)	No association with overweight
NO_x/elemental carbon									
McConnell et al. ²⁸⁾ 2015	USA	Cohort	3,318	10	18	BMI	NO _x	(+)	Synergism between tobacco smoke and NO _x exposure on attained BMI at the age of 18
Alderete et al. ²⁹⁾ 2017	USA	Cohort	314	8–15	18	BMI Body fat% SAAT IAAT	NO ₂	(+)	Positive association with BMI and SAAT at the age of 18
Kim et al. ¹⁶⁾ 2018	USA	Cohort	2,318	1	10	BMI	NO _x	(+)	Positive association with attained BMI at the age of 10
Fioravanti et al. ³²⁾ 2018	Italy	Cohort	719	0, 4	4, 8	BMI, WC, WHR	NO ₂	(0)	No association with childhood obesity
Bloemsmas et al. ³⁰⁾ 2019	Netherlands	Cohort	3,680	12–14	3–17	BMI	NO ₂	(+)	Positive association with overweight
de Bont et al. ³¹⁾ 2019	Spain	Cross-sectional	2,660	7–10	7–10	BMI	NO ₂	(+)	Positive association with overweight/obese
PM									
Kim et al. ³³⁾ 2016	Korea	Cohort	1,129	0.5–1	1–5	Weight-for-age	PM ₁₀	(-)	Negative association with weight at the age of 12–60 mos\
Mao et al. ¹⁸⁾ 2017	USA	Cohort	1,446	0–2	2–9	BMI	PM _{2.5}	(+)	Positive association with childhood overweight or obesity
Alderete et al. ²⁹⁾ 2017	USA	Cohort	314	8–15	18	BMI Body fat% SAAT IAAT	PM _{2.5}	(+)	Positive association with BMI and SAAT at the age of 18
Kim et al. ¹⁶⁾ 2018	USA	Cohort	2,318	1	10	BMI	PM _{2.5}	(0)	No association with attained BMI at the age of 10
Fioravanti et al. ³²⁾ 2018	Italy	Cohort	719	0, 4	4, 8	BMI, WC, WHR	PM _{2.5} PM ₁₀	(0)	No association with childhood obesity
Bloemsmas et al. ³⁰⁾ 2019	Netherlands	Cohort	3,680	12–14	3–17	BMI	PM _{2.5} PM ₁₀	(0)	No association with overweight
de Bont et al. ³¹⁾ 2019	Spain	Cross-sectional	2,660	7–10	7–10	BMI	PM _{2.5}	(+)	Positive association with overweight/obese
Smoking									
McConnell et al. ²⁸⁾ 2015	USA	Cohort	3,318	10	18	BMI	Smoking	(+)	Synergism between tobacco smoke and NO _x exposure on attained BMI at the age of 18
PAH									
Scinicariello et al. ³⁵⁾ 2014	USA	Cross-sectional	3,189	6–19	6–19	BMI, WC	PAHs	(+)	Positive association with obesity parameters in children

The direction of relationship with obesity: (+), factor related with greater childhood obesity; (-), factor related with decreased childhood obesity; (0), no relationship with childhood obesity.

BMI, body mass index; IAAT, cross-sectional area of intra-abdominal adipose tissue; NO₂, nitrogen dioxide; NO_x, nitrogen oxides; PAHs, polycyclic aromatic hydrocarbons PM_{2.5}, particulate matter <2.5 microns in diameter; PM₁₀, particulate matter <10 microns in diameter; SAAT, cross-sectional area of subcutaneous abdominal adipose tissue; WC, waist circumference; WHR, waist-to-hip ratio.

the USA, PM_{2.5} concentrations in infancy and at 8–15 years of age were positively associated with childhood obesity and BMI at 18 years of age.^{18,29)} Another cross-sectional study from Spain supported this relationship in children aged 7–10 years.³¹⁾

Meanwhile, 3 other cohort studies from the USA, Italy, and the Netherlands reported a null association between PM_{2.5}/PM₁₀ exposure and childhood obesity.^{16,27,32)} In contrast, a negative association between PM₁₀ exposure in infancy and subsequent

poor weight gain during toddlerhood was reported by a cohort study from Korea.³³⁾

Like prenatal smoking exposure, childhood tobacco smoke exposure is positively correlated with increased adolescent BMI.³⁴⁾ In particular, NO_x exposure reportedly has a synergistic effect with tobacco smoke on increasing obesity risk.³⁴⁾ The relationship between postnatal PAH exposure and obesity development was similar to that between prenatal exposure and obesity development. A cross-sectional study in the USA reported a relatively strong positive correlation between urinary PAH levels and obesity parameters in children aged 6–11 years.³⁵⁾

Plausible mechanisms by which air pollution affects childhood obesity

1. Physical inactivity

Air pollution increases the likelihood of obesity by inducing sedentary behaviors. Exposure to air pollution can cause cardio-respiratory symptoms such as coughing, shortness of breath, and high blood pressure, impeding outdoor activity by impairing athletic performance.^{36,37)} Air pollution alarms through various media also influence people's decisions regarding physical activity.³⁸⁾

2. Oxidative stress and systematic inflammation

One of the most important mechanisms of interest is the systemic inflammatory reactions that occur through the stimulation of oxidative stress processes. Several animal and human studies demonstrated an increase in proinflammatory cytokines (e.g., interleukin-6 and tumor necrosis factor- α) in the systemic circulation following inhalation exposure to diesel exhaust particles or PM.³⁹⁻⁴²⁾ Oxidative stress and mitochondrial damage in adipose tissue caused by air pollutant exposure increase the differentiation of white adipocytes, which store extra energy in the form of triglycerides, and increase the differentiation of brown adipocytes, which release energy as heat.^{43,44)} This change adversely affects the energy balance in adipose tissue, predisposing the person to obesity and metabolic abnormalities. Further, *in utero* exposure to air pollutants such as diesel exhausts increases the likelihood of obesity in offspring by causing fetal brain inflammation and a subsequent increase in appetite.⁴⁵⁾

3. Hypothalamic-pituitary-adrenal axis

Chronic psychological stress and the subsequent activation of the hypothalamic-pituitary-adrenal (HPA) axis is a well-known risk factor for obesity development and metabolic dysfunction. Recent studies have demonstrated that adrenocorticotropic hormone and glucocorticoid concentrations increase after the inhalation of ozone or PM in animal models. Therefore, chronic activation of the stress response system is also emerging as a possible mechanism of obesity following air pollution exposure.⁴⁶⁾

4. Epigenetic modulation

Epigenetic modulation might be a plausible mechanism, especially in cases of prenatal exposure to air pollution. Prenatal long-term exposure to air pollutants reported exerting epigenetic effects including alterations of DNA methylation, microRNAs, and noncoding RNAs and regulation of chromatin.⁴⁷⁾ These changes may cause the derangement of the mitochondrial machinery, which is closely related to the control of energy metabolism and inflammation. For instance, a recent study showed that *in utero* exposure to PAHs induced offspring obesity by hypomethylation of peroxisome proliferator-activated receptor- γ and activation of various genes associated with adipogenesis in the offspring's adipose tissue.⁴⁸⁾

Conclusions

Previous *in vitro* and *in vivo* studies indicated that air pollutants might act as obesogens by inducing physical inactivity and epigenetic modulation and promoting oxidative stress and HPA axis. The effects of air pollution on childhood obesity seem to vary according to pollutant type and components, exposed region and area, exposure measurement methods, and exposure duration. Further, exposure timing, observation duration, sex, and ethnicity may be important variables in the study of the effects of air pollution on obesity. Although longitudinal human studies on the possible effects of air pollution on the development of obesity are increasing, most focus on the effects of individual air pollutants, not the mixed effects of various air pollutants. Future large-scale and long-term follow-up studies considering all these factors are required to determine the effects of air pollution on childhood obesity.

Conflicts of interest

No potential conflict of interest relevant to this article was reported.

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