Review Article:

The Association Between Exposure to Ambient Particulate Matter and Childhood Obesity: A Systematic Review and Meta-analysis

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ABSTRACT

Context: Physical environment contamination and in particular, air pollution might cause long-term adverse effects in child growth and a higher risk of catching non-communicable diseases later in life.

Objective: This study aimed to overview the human studies on the association of exposure to ambient Particulate Matter (PM) with childhood obesity.

Data Sources: We systematically searched human studies published until March 2018 in PubMed, Scopus, Ovid, ISI Web of Science, Cochrane library, and Google Scholar databases.

Study Selection: All studies that explored the association between PM exposure and childhood obesity were assessed in the present study, and finally, 5 studies were used in the meta-analysis.

Data Extraction: Two independent researchers performed the data extraction procedure and quality assessment of the studies. The papers were qualitatively assessed by STROBE (Strengthening the Reporting of Observational studies in Epidemiology) statement checklist.

Results: The pooled analysis of PM exposure was significantly associated with increased Body Mass Index (BMI) (Fisher's z-distribution=0. 028; 95% CI=0. 017, 0. 038) using the fixed effects model. We also used a random-effect model because we found a significant high heterogeneity of the included studies concerning the PM (I^2=94. 4%; P<0. 001). PM exposure was associated with increased BMI (Fisher's z-distribution=0. 022; 95% CI=-0. 057, 0. 102). However, the overall effect size was not significant, and heterogeneity of the included studies was similar to the fixed effect model.

Discussion: Our findings on the significant association between PM10 exposure and the increased BMI (r=0. 034; 95%CI=0. 007, 0. 061) without heterogeneity (I^2=16. 6%, P=0. 274) (in the studies with PM10) suggest that the PM type might account for the heterogeneity among the studies.

Conclusion: The findings indicate that exposure to ambient PM10 might have significant effects on childhood obesity.

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1. Context

Childhood obesity is a growing public health problem, even in developing countries (1, 2). It is associated with several health complications during childhood, which will usually extend to adulthood (3). It has several underlying causes, both genetic and environmental factors (4, 5).

Recently, researchers have paid attention to the association between air pollution and obesity, and some studies suggest that ambient air pollution may increase the risk of catching Non-communicable Diseases (NCDs) in adults, diseases such as cardiovascular diseases, diabetes and cancer (6-8). However, little epidemiological evidence is available on the association of exposure to ambient air pollution with the development of childhood obesity (9-11). Physical environment contamination and in particular, air pollution might cause long-term adverse effects in child growth and a higher risk of developing NCDs later in life. The "Obesogenic Environment" hypothesis discusses the impact of environmental chemicals with endocrine disruption properties that can change child growth patterns and result in weight gain, obesity, and obesity-related NCDs (12).

Some previous studies reveal a positive association between exposure to Polycyclic Aromatic Hydrocarbons (PAHs) and childhood obesity (11, 13). A recent study conducted in China reports that long-term exposure to air pollutants, including Particulate Matter $(PM)_{10}$, NO_2, SO_2, and O_3 is associated with higher risk of childhood obesity and hypertension (14). Moreover, the association of residential traffic density and roadway proximity with rapid infant weight gain and childhood obesity has been documented in some previous studies (15-18). A study on Latino children living in the US shows that higher exposure to NO_2 and $PM_{2.5}$ is related to higher Body Mass Index (BMI) at the age of 18 (19). However, some other studies report no association between exposure to vehicular traffic and pollutants and the risk of obesity and dyslipidemia in children (20, 21). Therefore, the overall evidence on obesogenic properties of air pollutants is controversial.

2. Objectives

Due to the high prevalence of childhood obesity, its multifactorial nature, and the importance of conducting preventive strategies, we aimed to provide a systematic review and meta-analysis on the association of exposure to ambient PM and childhood obesity.

3. Data Sources

We performed a systematic review and meta-analysis of human studies that explored the association between PM exposure and childhood obesity. We considered PECO as the following: Population (P): Children and adolescents; Exposure (E): PM exposure; Comparison (C) (There is no comparison between exposed and non-exposed groups because we have reported the correlations of PM exposure and BMI); and Outcome (O): Childhood obesity (BMI).

We systematically searched human studies available on the study subject until March 2018 in PubMed, Scopus, Ovid, ISI Web of Science, Cochrane library, and Google Scholar databases. All cross-sectional and cohort studies were selected. We used the search terms of "Air Pollution" OR "Pollutants" OR "Particulate Matter" in combination with "Obesity" OR "Weight" OR "Body Mass Index" OR "BMI" OR "Overweight" OR "Cardiometabolic" OR "Metabolic Syndrome" OR "Metabolic Syndrome X" OR "Mets" OR "Adiposity" AND "Child" OR "Adolescent" OR "School-aged" OR "Youth" OR "Teenager" OR "Boy" OR "Girl" OR "Student" OR "Pediatrics" in the form of Medical Subject Headings (MeSH) and truncations. The relevant articles were examined without any language restriction.

4. Study Selection

After removing the duplicates, the relevant papers were selected in three phases. In the first and the second phases, titles and abstracts of papers were screened, and the irrelevant papers were excluded. In the third phase, the full texts of the remaining papers were explored carefully to select only the relevant papers. To find any additional pertinent study, the reference list of all reviews and related papers were screened as well.

The included studies had the following criteria: 1. Observational cross-sectional design; 2. Longitudinal cohort studies which report the study association; 3. Measurement of PM concentration as an index for air pollution exposure; and 4. Reporting the Odds Ratio (OR), Relative Risk (RR), and β-coefficient of PM with child obesity. In the final step, all statistics were changed to the correlation coefficient values.

5. Data Extraction

Two reviewers extracted the data independently using a data collection form, including the first author's name, publication year, sample size, study design, as well as

age, exposure measurement, statistical analysis, and the variables adjusted in the analyses.

5. 1. Quality assessment

Two independent reviewers (MB and MKH) evaluated the methodological quality of each study. The Strengthening the Reporting of Observational studies in Epidemiology (STROBE) checklist was used for the quality assessment of the papers. According to STROBE (22 questions), the included studies were divided into three groups of high, medium, and low-quality. The studies scored one to eight were ranked as low-quality studies, 9-16 as medium-quality ones, and 17-22 as high-quality papers. The two reviewers agreed on (80%) of cases. The remaining discrepancies were resolved by consultation and consensus.

5. 2. Statistical analysis

The effect sizes of RR, OR, and β-coefficient from all articles were extracted directly from the original reports. All effect sizes were transformed into (r: correlation), and Fisher z-transformation of the r value was applied for the pooled analysis (22, 23). The potential heterogeneity across studies was evaluated using the Cochran's Q test and was expressed using the I^2 index. The pooled results for Fisher z-transformation were calculated by the fixed-effects model (for low heterogeneity) or the random-effects model (for high heterogeneity). Publication bias was evaluated by the Egger's and the Begg's tests. Subgroup analyses and meta-regression were performed to seek the sources of heterogeneity. The sensitivity analyses were performed by omitting one study at a time to gauge the robustness of our results. All statistical analyses were conducted in STATA V. 14. 0.

6. Results

We initially retrieved 4391 articles from the databases. Figure 1 represents the search results. After the initial study of the titles and abstracts, the duplicate papers were omitted, and out of 4276 papers, five articles remained. No additional references were identified through checking the reference lists of selected papers.

The main characteristics of the studies included in the systematic review are presented in Appendix 1. Overall, the studies reported data on 33825 subjects, and they were published between 2010 and 2018.

6. 1. Meta-analysis of the correlations

Figure 2 showed the pooled results using random effect model. It showed that PM exposure was associated with the increased BMI (Fisher-z= 0. 022; 95% CI (-0. 057, 0. 102)) that overall effect size was not significant and heterogeneity of the included studies was as same fixed effect model.

Table 1 presents the results of the meta-regression analysis. The univariate meta-regression analyses indicated that none of the factors, including mean age, sample size, study location (Europe, Asia, and the USA), study type (cross-sectional and cohort), and PM type (2. 5 and 10) contributed to the heterogeneity of meta-analysis (P>0. 05 for all).

Table 2 presents the results of subgroup analysis according to the study location, study type, and PM type. We observed significant association between PM_{10} exposure and the increased BMI (Fisher's z=0. 034; 95% CI=0. 007, 0. 061) with no apparent heterogeneity (I^2=16. 6%, P=0. 274) in the studies with PM_{10}. It suggests that the PM type may partially account for the heterogeneity among the studies on BMI (Figure 3).

Begg's test and Egger's test revealed no obvious publication bias among these studies. The P-values for these tests were higher than 0. 05 (P=0. 661 and 1. 0, respectively). The results of sensitivity analyses showed that with excluding the study of Fleisch AF et al. (7. 7 years), the pooled Fisher's z for the subgroup $PM_{2.5}$ increased. Although this change was not significant, it decreased the overall heterogeneity (I^2=83. 1%, P<0. 001) (Figure 4).

Figure 4 Forest plot of Fisher's z values for the correlation between PM and BMI by PM type after excluding the study of Fleisch AF et al. (7. 7 years) Table 3 presents the results of converting Fisher's z values into correlation values. We found a significant relationship between PM_{10} and BMI (r=0. 034, P=0. 015), but the association of $PM_{2.5}$ and BMI was not statistically significant (r=0. 035, P=0. 606).

7. Discussion

This systematic review and meta-analysis revealed a weak positive association between ambient PM_{10} and child obesity. However, the results for $PM_{2.5}$ was not significant. A few meta-analysis or large sample size studies have explored the association of ambient PM with adult obesity or birth weight, but with childhood obesity (24-26).

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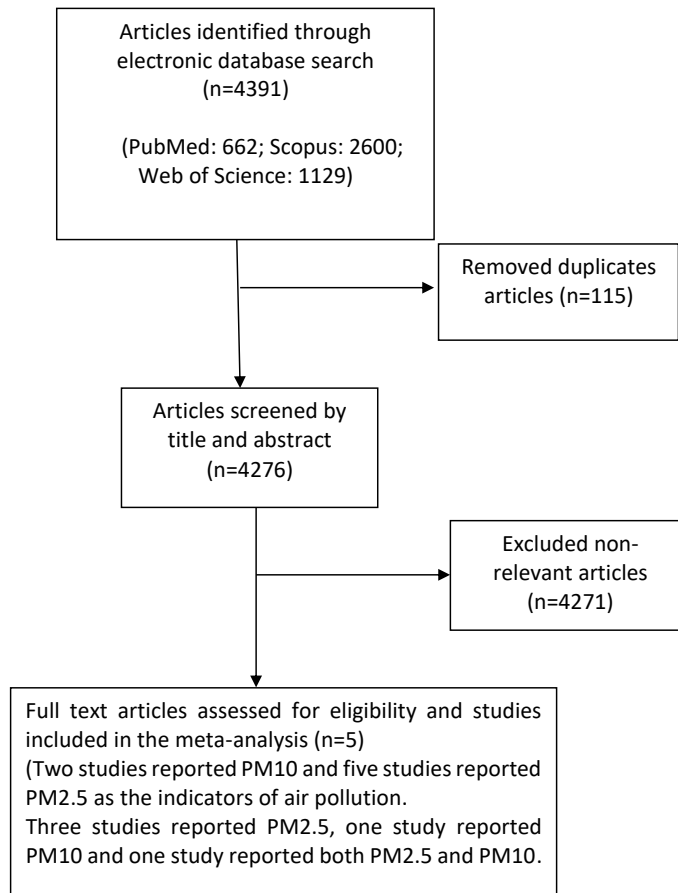
Figure 1. The flowchart of the search results

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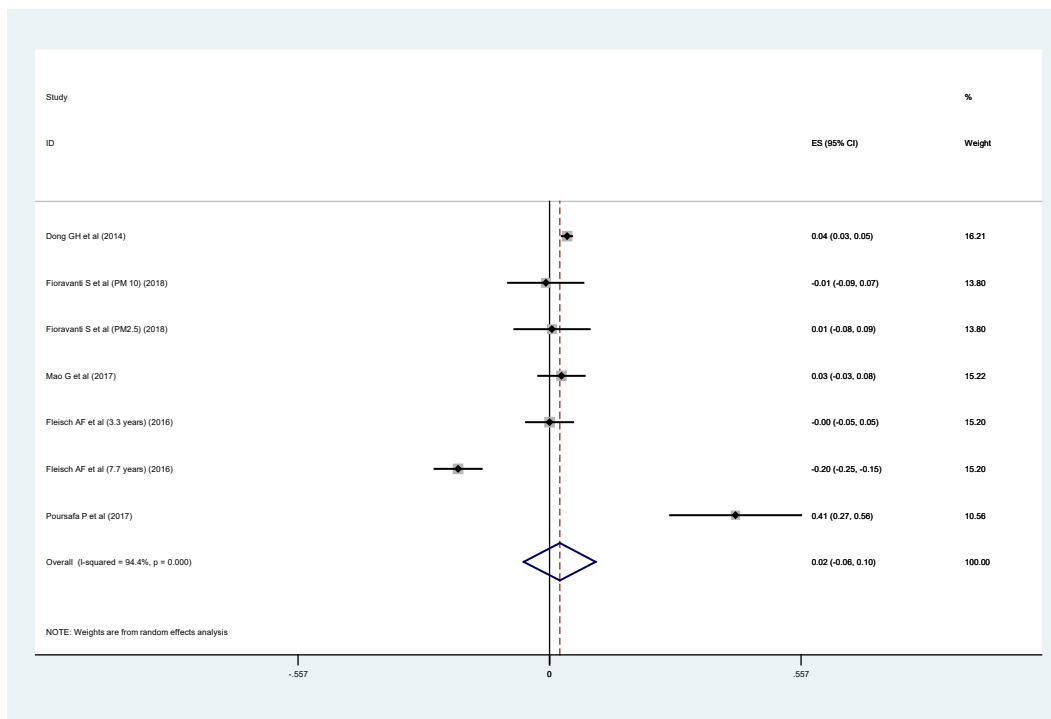
Figure 2. Forest plot of Fisher's z values indicating the correlation between PM and BMI

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Table 1. Results of meta-regression analyses for the potential source of heterogeneity

Covariate		B	SE	P	95% CI
Year of publication		0. 016	0. 056	0. 790	(-0. 128, 0. 159)
Mean age		0. 040	0. 030	0. 243	(-0. 038, 0. 118)
PM2. 5 (Ref, PM10)		0. 024	0. 162	0. 889	(-0. 392, 0. 440)
Sample size of study		-0. 0000002	0. 000007	0. 974	(-0. 00002, 0. 00002)
Study location: (Ref. : Asia)	USA	-0. 264	0. 144	0. 107	(-0. 664, 0. 136)
	Europe	-0. 207	0. 159	0. 109	(-0. 649, 0. 235)
	Study type: Cohort (Ref: Case control)	-0. 238	0. 121	0. 107	(-0. 550, 0. 074)

SE: Standard Error; CI: Confidence Interval

Table 2. Results of subgroup analysis on the association between PM and BMI

Variables	Groups	NO. of Study	Effect Size (Fisher' z) 95% CI	P	Heterogeneity	
					I² (%)	P
PM type	10	2	0. 034 (0. 007, 0. 061)	0. 015	16. 60	0. 274
	2. 5	3	0. 035 (-0. 099, 0. 169)	0. 606	95. 30	< 0. 001
Study type	Cross-sectional	2	0. 218 (-0. 148, 0. 583)	0. 243	96. 10	< 0. 001
	cohort	3	-0. 037 (-0. 132, 0. 057)	0. 442	91. 60	< 0. 001
Study location	Asia	2	0. 218 (-0. 148, 0. 583)	0. 243	96. 10	< 0. 001
	Europe	1	-0. 001 (-0. 06, 0. 057)	0. 961	0. 00	0. 818
	USA	2	-0. 059 (-0. 2, 0. 083)	0. 416	95. 50	< 0. 001
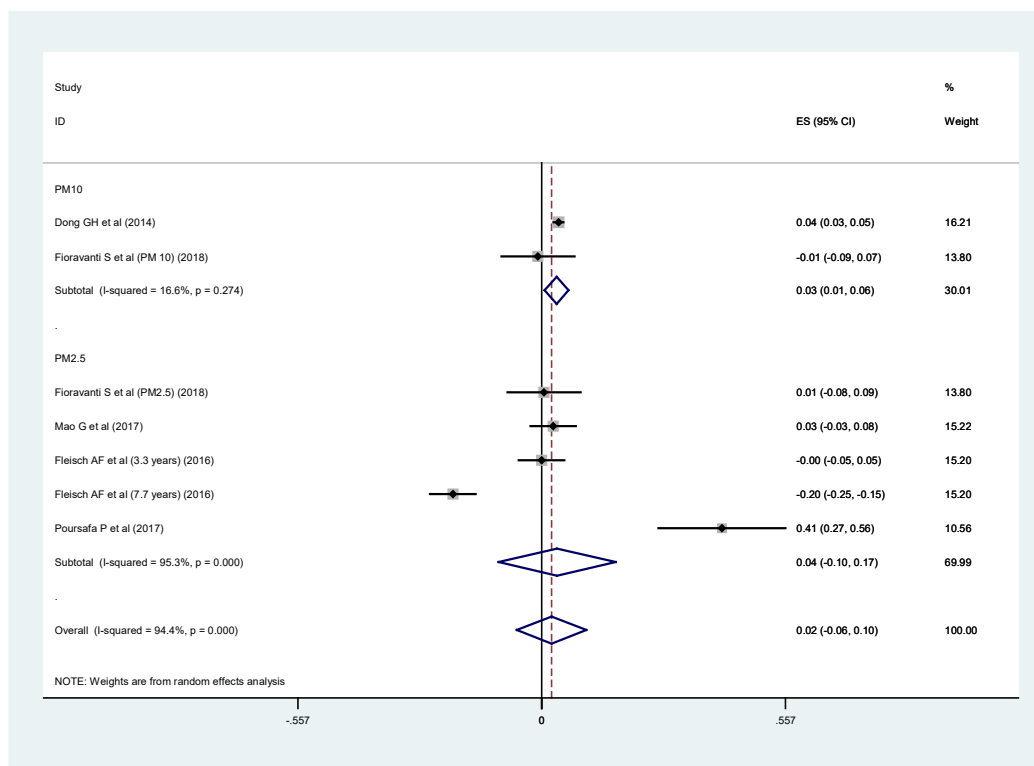
Figure 3. Forest plot of Fisher's z values indicating the correlation between PM and BMI by PM type

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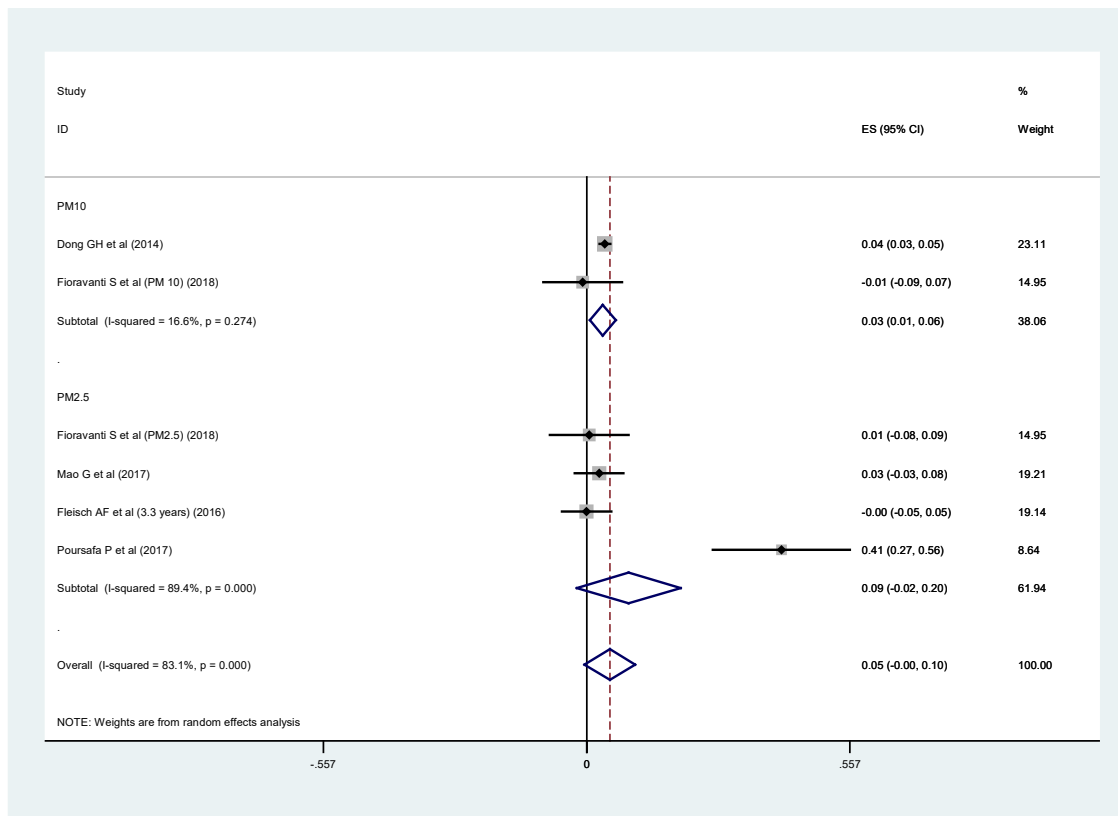
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Figure 4. Forest plot of Fisher's z values, indicating the correlation between PM and BMI by PM type after excluding the study of Fleisch AF et al (7. 7 years)

Table 3. The correlation between PM exposure and BMI

Variables	Effect Size			Heterogeneity		
	Pooled r	95% CI	P	I^2	P	τ^2
PM10	0. 034	(0. 007, 0. 061)	0. 015	16. 60%	0. 0002	0. 0002
PM2. 5	0. 035	(-0. 099, 0. 167)	0. 606	95. 30%	0. 0216	0. 022
overall	0. 022	(-0. 057, 0. 102)	0. 579	94. 40%	0. 0101	0. 010

τ^2: Between-studies variance

These five studies investigated more than 33000 participants. The current literature provides conflicting results on the association between air pollution and childhood obesity (19-21, 27). We found a relatively weak positive relationship between exposure to PM_{10} and childhood BMI, consistent with most previous studies findings. Five of the seven studies included in the current meta-analysis reported the direct association of air pollution and child weight, whereas two cohort studies did not report such association (20, 21).

Such discrepancies among these studies results might be due to confounding factors like age, gender, ethnicity, physical activity, level of exposures, and some other factors. These findings might be confounded by heterogeneity due to multiple dispersions between studies such as study design, different techniques to measure PM concentration, the way PM levels is reported, and other various confounders which were adjusted in the analysis.

Only a few cross-sectional studies have investigated the relation of air pollution and obesity in children (16, 17, 20, 27-29). In a longitudinal study, higher exposure to NO_2 and

$PM_{2.5}$ was associated with higher BMI, body fat percentage, and abdominal obesity during follow up and at the age of 18 in children who were overweight or obese at the study baseline (19). Another study conducted on overweight and obese minority youths found that higher exposures to NO_2 and $PM_{2.5}$ during one year before the study was not associated with obesity, but it was related to lower insulin sensitivity and higher acute insulin response to glucose, which might contribute to obesity (19, 30).

The mechanisms linking air pollution to obesity risk and type-2 diabetes are not entirely determined. The effects of air pollutants on immune response, oxidative stress, and insulin resistance might explain the results (31).

Air pollutants such as PM might increase the infiltration and activation of immune-competent cells, including monocyte and macrophages, in body tissues (32). Previous findings also indicated that early life exposure to $PM_{2.5}$ might result in insulin resistance and obesity later in life, through NAD(P)H oxidase-derived superoxide anions, which might cause changes in adipocyte numbers and size (33).

Animal studies suggest that higher exposure to air pollutants might result in increased adipose tissue inflammation, accumulation of glucose in skeletal muscles, and therefore it might contribute to metabolic dysfunction and obesity (34, 35). Furthermore, previous studies indicate that long-term exposure to combustion-related air pollutants can increase systemic inflammation and oxidative stress (34).

Little information is available about the biological basis of the relationship between exposure to air pollutants and childhood obesity. There may be a potential for residual confounders, including socioeconomic status and physical activity, which can be associated with both air pollution exposure and children's weight. Therefore, residual confounding may affect the study results due to the associations of poor diet and low physical activity with child overweight and metabolic disruption. Also, these factors may be related to residential proximity to sources of air pollution (36, 37). For example, children living in areas with higher levels of air pollution usually belong to lower socioeconomic families who often consume higher amounts of total or saturated fats (27, 38).

Lack of physical activity among the children living in polluted regions (because of their parental control to restrict the children's exposure to air pollution) may be another reason for the excess weight in children. It is documented that overweight children usually have less frequent and shorter periods of activities compared to their normal weight peers (39, 40). However, the findings on the associations of exposure to air pollutants and childhood obesity are unlikely to be confounded by these factors, because many of these studies had adjusted these associations for socioeconomic status, as a strong predictor of dietary intake and physical activity (36).

Furthermore, misclassification of exposure to air pollutants might have occurred with residential-based estimates of pollutant exposure, which might decrease the observed effects (41). Some studies also lack information about other potential confounders such as active and passive smoking as well as exposure to noise pollution (19). Previous studies suggest that tobacco exposure and near roadway air pollution contribute to synergistic effects on the development of child obesity (18).

The findings of the current study concerning the association of exposure to ambient PM with childhood obesity should be considered with caution. The cross-sectional design of some studies used for this meta-analysis might preclude any causality. Another limitation is the high heterogeneity between studies. Other potential risk factors like child physical activity, familial socioeconomic status, and climate conditions were not available in some studies.

8. Conclusions

This systematic review and meta-analysis indicate that exposure to ambient PM_{10} has a weak positive association with childhood obesity. This finding should be considered in future studies and preventive programs. Our results are also useful for health policymakers and health care providers to design health promotion interventions and preventive strategies. More research is needed to clarify the effect of other ambient air pollutants on child health status.

Ethical Considerations

Compliance with ethical guidelines

All ethical principles were considered in this article.

Funding

Authors contribution's

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Conflicts of interest

The authors declared no conflict of interests.

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Appendix 1. Summary of exposure and outcome assessment strategies and an estimate of effect size in included studies

Author(s)/Date	Study Location	Study Design	Follow Up Duration	Sample Size	Age (y)	Outcome	Exposure Assessment Method	Exposure	Group / Subgroups	Effect Size CI (95%)	Adjustment Factors
Michael Jerrett, 2010	Southern California, USA	Cohort	8-year follow-up	2889	10–18 years	Body Mass Index (BMI)	Traffic-related air pollution	Annual average daily traffic (AADT) 150 m	B (SE): 0. 0039 (0. 0008)	Gender, cohorts variables parental education, personal weekly smoking, second hand smoke (current + past), ever asthma, buffer population, gamma index, proportion of below poverty people within census block, normalized difference vegetation index (NDVI), foreign-born, community-level violent crime rate, and having no food stores within 500-m road network buffer with random community effects
								AADT 300 m	0. 0013 (0. 0008)	
Pei, 2013	Germany	Cohort	10-year follow-up	3121 Females (N=1114), Males (N=1158)	10 (We predicted standardized body mass index (BMI) at 10 years of age using standardized BMIs from birth to 5 years.)	Maternal smoking during pregnancy			β (CI): 0. 13 (0. 03, 0. 22)	Parental education, family income, and maternal smoking during pregnancy
Michael Jerrett (2014)	Southern California, USA	Cohort	4-year follow-up	N=4550	5–11 years			Traffic density	B (SE) 0. 0002 (0. 0001)	Having asthma, parental education, immigrant status, the measure of green cover, street connectivity, recreational programming within 5 km of the home, and fast food access within 500 m of the home
								Non-Freeway NOx	0. 0861 (0. 0255)	

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Guang-Hui Dong, 2014	China	Cross-sectional	-	30056	2-14	Obesity BMI>95th percentile	Measurements of ambient PM_{10}, SO_2, NO2, and O_3 concentrations from 2006 to 2008 were obtained at municipal air pollution monitoring stations.	PM_{10} (µg/m³) SO_2 (ppb) NO2 (ppb) O_3 (ppb)	OR(CI): 1.19 (1.11–1.26) 1.11 (1.03–1.20) 1.13 (1.04–1.22) 1.14 (1.04–1.24)	Age, gender, parental education, breast feeding, low birth weight, area of residence per person, house decorations, home coal use, ventilation device in the kitchen, air exchange in winter, passive smoking exposure, and districts
Rob McConnel, 2015	Southern California, USA	Cohort	8-year	3318	10-18	BMI	Residential near-road-way pollution exposure (NRP) was estimated based on a line source dispersion model accounting for traffic volume, proximity, and meteorology	Secondhand smoke	BMI growth (95%CI): One smoker at home: 0.48 (0.16, 1.12) Two or more than 2 smokers at home: 1.08 (0.19, 1.97) Maternal smoking during pregnancy: 0.72 (0.14, 1.31) NRP: 1.13 (0.61, 1.65) The difference in the attained BMI (95%CI): One smoker at home: 0.95 (0.42, 1.47) ≥2 smokers at home: 1.77 (1.04, 2.51) Maternal smoking during pregnancy: 1.14 (0.66, 1.62) NRP: 1.27 (0.75, 1.80)	Ethnicity, sex, community, year of enrollment, and age

Author(s)/ Date	Study Location	Study Design	Follow Up Duration	Sample Size	Age (y)	Outcome	Exposure Assessment Method	Exposure	Group / Subgroups	Effect Size CI (95%)	Adjustment Factors
Fleisch, 2016	Boston, USA	Cohort	Early childhood (median: 3.3 years of age) mid-childhood (median: 7.7 years of age)	1418	Mean (standard deviation) of age at early childhood visit 3.3 (0.4), and at mid-childhood visit 8.0 (0.9)	BMI z-score	Spatiotemporal models to estimate prenatal and early life residential PM₂.₅ and black carbon exposure as well as traffic density and roadway proximity.	PM₂.₅ (µg/m³)	3.3 years		Child (age, sex and race/ethnicity), mother (age, education, and smoking during pregnancy), neighborhood (census tract median income), season and date of health outcome
									Third trimester	0.0 (-0.1, 0.1)	
									Year prior to early childhood visit	-0.0 (-0.1, 0.1)	
									Early childhood address	0.0 (-0.0, 0.1)	
							Near-residence traffic density		Early childhood address	0.0 (-0.0, 0.1)	
								Birth address	<50m	0.3 (0.0, 0.7)	
								Proximity to major roadway, birth address Reference:≥200m	[50, 100m]	-0.0 (-0.4, 0.3)	
									[100, 200m]	0.4 (0.1, 0.6)	
								Proximity to major roadway, early childhood address Reference:≥200m	<50 m	0.1 (-0.2, 0.5)	
									[50, 100 m]	-0.0 (-0.4, 0.3)	
									[100, 200 m]	0.1 (-0.2, 0.3)	
Yueh-Hsiu Mathil-da Chiu 2017	Boston, USA	Cohort	4.0±0.7 years	239		BMI z-score	Prenatal daily PM₂.₅ exposure was estimated using a validated satellite-based spatio-temporal model. Prenatal PM₂.₅ level (µg/m³) Median IQR 10.7[9.9–11.4]	PM₂.₅ (µg/m³)	Girls	-0.12 (-0.37,-0.03)	Maternal age, race/ethnicity, education, pre-pregnancy BMI, and child's age at anthropometric measurement
									Boys	0.21 (0.003,-0.37)	

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Tanya Alderete, 2017	Los Angeles, USA	Cohort	3.4 years	314 overweight and obese children	8-15 years	BMI	NO2 and PM$_{2.5}$ were modeled as long-term exposure using cumulative 12-month averaged exposure during the follow-up. Estimated effect estimates were reported for a 5-ppb difference in NO2 and a 4-µg/m³ difference in PM$_{2.5}$	NO$_2$ (ppb) · PM$_{2.5}$ (µg/m³)	2.1 (0.1, 4.1) · 3.8 (1, 6.6)	Sex, Tanner stage, the season of testing (warm/cold), prior year exposure at each follow-up visit, social position, body fat percentage (where appropriate), study wave, and study entry year
Poursafa, 2017	Iran	Cross-sectional	—	186	6-18	BMI	The air quality index (AQI) is used to describe the level of air pollution with adverse health effects. We used PM$_{2.5}$ data.		B=0.34	Age and gender
Sara Fioravant, 2018	Italy	Cohort	8-year follow-up	581	Birth-8 years	Prevalence of over-weight/obesity was 9.3% and 36.9%	Air pollution was assessed at the residential address	NO2 (per 10 µg/m³) · NOX (per 20 µg/m³) · PM$_{10}$ (per 10 µg/m³) · PM$_{2.5}$ (per 5 µg/m³) · PMcoarse (per 5 µg/m³) · PM$_{2.5}$abs (per 1 µg/m³)	0.99 (0.86, 1.12) · 0.98 (0.86, 1.12) · 0.971 (0.77, 1.23) · 1.02 (0.75, 1.40) · 0.91 (0.77, 1.09) · 1.10 (0.88, 1.37)	Maternal age at delivery, race/ethnicity, education level, smoking status, diabetes, marriage status, household income per year, the season of childbirth, preterm birth, birth weight, and breastfeeding
Guangyun Mao, 2017	Boston, USA	Cohort	2-year follow-up	1,446	first 2 years of life		Comparing the highest and lowest quartiles of PM (2.5 µg/m³)		The adjusted Relative Risks (RRs) 1.3 (1.1, 1.5)	Maternal age at delivery, diabetes, maternal pre-pregnancy BMI, maternal smoking during pregnancy, gestational diabetes, maternal age at delivery, gestational age, childbirth weight, breast-feeding duration, age (in months) at weaning

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