Environment International 200 (2025) 109527

Contents lists available at ScienceDirect

Environment International

journal homepage: www.elsevier.com/locate/envint

Full length article

Ambient air pollution and childhood obesity from infancy to late childhood: An individual participant data meta-analysis of 10 European birth cohorts

Sarah Warkentin [a,b,c,1,*], Serena Fossati [a,b,c,1], Sandra Marquez [a,b,c], Anne-Marie Nybo Andersen [d], Sandra Andrusaityte [e], Demetris Avraam [d], Ferran Ballester [f], Tim Cadman [a,g], Maribel Casas [a,b,c], Montserrat de Castro [a,b,c], Leda Chatzi [h], Ahmed Elhakeem [i], Antonio d'Errico [j,k], Mònica Guxens [a,b,c,l,ac], Regina Grazuleviciene [e], Jennifer R. Harris [m], Carmen Iñiguez Hernandez [c,n], Barbara Heude [o], Elena Isaevska [j,l], Vincent W.V. Jaddoe [p,q], Marianna Karachaliou [a,r], Aitana Lertxundi [c,s,t], Johanna Lepeule [u], Rosemary R.C. McEachan [v,w], Johanna L. Thorbjørnsrud Nader [x], Marie Pedersen [d], Susana Santos [p,q,y,z], Mariska Slofstra [g], Euripides G. Stephanou [aa], Morris A. Swertz [g], Tanja Vrijkotte [ab], Tiffany C. Yang [v,w], Mark Nieuwenhuijsen [a,b,c], Martine Vrijheid [a,b,c]

[a] *ISGlobal, Barcelona, Spain*
[b] *Universitat Pompeu Fabra (UPF), Barcelona, Spain*
[c] *Spanish Consortium for Research on Epidemiology and Public Health (CIBERESP), Instituto de Salud Carlos III, Madrid, Spain*
[d] *Department of Public Health, University of Copenhagen, Copenhagen, Denmark*
[e] *Department of Environmental Sciences, Vytautas Magnus University, Kaunas, Lithuania*
[f] *FISABIO-University Jaume I-Universitat de València, Valencia, Spain*
[g] *Department of Genetics and Genomics Coordination Center, University Medical Center Groningen, University of Groningen, Groningen, the Netherlands*
[h] *University of South California, Los Angeles, USA*
[i] *University of Bristol, Bristol, UK*
[j] *Cancer Epidemiology Unit, Department of Medical Sciences, University of Turin, Turin, Italy*
[k] *Institute for Risk Assessment Sciences, Division of Environmental Epidemiology, Utrecht University, Utrecht, the Netherlands*
[l] *Department of Child and Adolescent Psychiatry/Psychology, Erasmus University Medical Center, the Netherlands*
[m] *Centre for Fertility and Health, Norwegian Institute of Public Health, Oslo, Norway*
[n] *Department of Statistics and Operational Research, Universitat de València, Valencia, Spain*
[o] *Université Paris Cité and Université Sorbonne Paris Nord, Inserm, INRAE, Center for Research in Epidemiology and StatisticS (CRESS), F-75004 Paris, France*
[p] *Generation R Study Group, Erasmus MC, University Medical Center Rotterdam, Rotterdam, the Netherlands*
[q] *Department of Pediatrics, Erasmus MC, University Medical Center Rotterdam, Rotterdam, the Netherlands*
[r] *Clinic of Preventive and Social Medicine, Medical School, University of Crete, Greece*
[s] *University of the Basque Country, Bilbao, Spain*
[t] *Biogipuzkoa Health Research Institute, Donostia-San Sebastian, Spain*
[u] *Université Grenoble Alpes, Inserm, CNRS, Team of Environmental Epidemiology applied to Development and Respiratory Health, IAB, Grenoble, France*
[v] *Bradford Institute for Health Research, Bradford Teaching Hospitals NHS Foundation Trust, Bradford, UK*
[w] *Public Health Improvement, UK*
[x] *Department of Genetics and Bioinformatics, Division of Health Data and Digitalisation, Norwegian Institute of Public Health, Oslo, Norway*
[y] *EPIUnit - Instituto de Saúde Pública, Universidade do Porto, Porto, Portugal*
[z] *Laboratório para a Investigação Integrativa e Translacional em Saúde Populacional (ITR), Universidade do Porto, Porto, Portugal*
[aa] *Department of Chemistry, University of Crete, Greece*
[ab] *University of Amsterdam, Amsterdam, the Netherlands*
[ac] *ICREA, Barcelona, Spain*

* Corresponding author at: ISGlobal - Barcelona Institute for Global Health, C/ Doctor Aiguader, 88, 08003 Barcelona, Spain.
E-mail address: sarah.warkentin@isglobal.org (S. Warkentin).
[1] Joint co-authorship.

https://doi.org/10.1016/j.envint.2025.109527
Received 27 November 2024; Received in revised form 6 May 2025; Accepted 9 May 2025
Available online 11 May 2025

ARTICLE INFO

Handling Editor: Hanna Boogaard

Keywords:
Air pollution
Child
Pediatric obesity
Urban
Meta-analysis

ABSTRACT

Ambient air pollution may contribute to childhood obesity through various mechanisms. However, few longitudinal studies examined the relationship between pre- and postnatal exposure to air pollution and obesity outcomes in childhood. We aimed to investigate the association between pre- and postnatal exposure to air pollution and body mass index (BMI) and the risk of overweight/obesity throughout childhood in European cohorts. This study included mother–child pairs from 10 European birth cohorts (n = 37111 (prenatal), 33860 (postnatal)). Exposure to nitrogen dioxide (NO_2) and fine particulate matter with aerodynamic diameter < 2.5 μm ($PM_{2.5}$) was estimated at the home addresses during pre- and postnatal periods (year prior outcome assessment). BMI z-scores (continuous) and overweight/obesity status (categorical: zBMI≥+2 (<5 years) or ≥+1 (≥5 years) standard deviations) were derived at 0–2, 2–5, 5–9, 9–12 years. Associations between air pollution exposure and zBMI were estimated separately for each pollutant and cohort using linear and logistic longitudinal mixed effects models, followed by a random-effects meta-analysis. The overweight/obesity prevalence ranged from 12.3-40.5 % between cohorts at 0–2 years, 16.7–35.3 % at 2–5 years, 12.5–40.7 % at 5–9 years, and 10.7–43.8 % at 9–12 years. Results showed no robust associations between NO_2 exposure and zBMI or overweight/obesity risk. Exposure to $PM_{2.5}$ during pregnancy was associated with 23 % (95%CI 1.05;1.37) higher overweight/obesity risk across childhood, and higher zBMI and overweight/obesity risk at 9–12 years. Heterogeneity between cohorts was considerable (I^2:25–89 %), with some cohort-specific associations; e.g., pre- and postnatal exposure to $PM_{2.5}$ was associated with lower zBMI across age periods in UK cohorts (ALSPAC and BiB), while postnatal exposure to $PM_{2.5}$ and NO_2 was associated with higher zBMI in one Dutch cohort (Generation R). Overall, this large-scale meta-analysis suggests that prenatal $PM_{2.5}$ exposure may be associated with adverse childhood obesity outcomes, but provides no evidence to support an effect of postnatal air pollution exposure, although cohort-specific associations were observed.

1. Introduction

The worldwide prevalence of childhood obesity has quadrupled since the 1990s, when 65.1 million girls and 94.2 million boys in school-age were estimated to have obesity, with rates in constant rise (Phelps et al., 2024). Obesity is a multifactorial condition and consequence of a complex interaction between genes, lifestyle, social and psychological determinants (An et al., 2018a; González-Muniesa et al., 2017). Overweight and obesity in early life is associated with a higher risk of developing adult obesity, poorer health and lower quality of life (World Health Organization, 2016), which may persist into later life if not prevented. The identification of modifiable risk factors of these conditions represents an important opportunity for public health interventions (World Health Organization (WHO), 2021).

Exposure to combustion related pollutants, such as particulate matter with aerodynamic diameter <2.5 μm ($PM_{2.5}$) <10 μm (PM_{10}), as well as nitrogen dioxide (NO_2) is widespread, with approximately 56 % of the population worldwide (4.4 billion people) living in more polluted, urban areas (Worldbank, 2023). Long-term exposure to these pollutants, in particular to nitrogen oxides (NO_x), NO_2 (Malacarne et al., 2022; Wang et al., 2021), and particulate matter ($PM_{2.5}$ and PM_{10}) (Parasin et al., 2021; Zheng et al., 2024), may be associated with increased risk of overweight and obesity in children, as described in four systematic reviews published in the past four years. Several direct and indirect mechanisms have been proposed to explain these associations, including inflammation (Jerrett et al., 2014), decreased glucose utilization in skeletal muscles (Toledo-Corral et al., 2018), endocrine system disruption (Darbre, 2018), respiratory diseases and decreased lung function (Wang et al., 2019), besides changes in basal metabolism and appetite control of the central nervous system (McConnell et al., 2016). Indirectly, areas in which motorized road traffic and air pollution levels are high, with limited safe, recreational areas and/or visible smog, noise, for example, may lead to behavior changes in the population, which may discourage outdoor physical activities and, consequently, increase weight gain (An et al., 2018a; Tainio et al., 2021).

There are critical windows of exposure to air pollution, including pregnancy and birth to 1 year of age, which are associated with development disorders, cancer, obesity and other conditions in childhood (Spencer-Hwang et al., 2023); thus, it is possible that exposure to air pollutants during these periods may also have disproportionate impacts on child growth and adiposity. However, evidence is inconsistent. Prenatal exposure to ambient air pollution has been associated with fetal growth restriction and low birth weight (Bekkar et al., 2020; Health Effects Institute, 2022; Hung et al., 2022; Pedersen et al., 2013), decreased weight and a reduced risk of being in a trajectory with accelerated body mass index (BMI) gain at the age of 4 (Fossati et al., 2020). In addition, prenatal exposure has been associated with variable changes in growth rates, with increases in the growth rate from the 3rd trimester of gestation up to 3 months of infancy and decreases between 6 months and 2 years in a study from the US (Ji et al., 2023). Others, however, did not find significant associations between prenatal traffic-related air pollution exposure and childhood adiposity in the first years of life (Frondelius et al., 2018; Starling et al., 2020). Regarding postnatal air pollution exposure, several longitudinal and cross-sectional studies have reported associations between exposure to pollutants, such as NO_2, and higher weight in children and adolescents from China (Huang et al., 2019), the Netherlands (Bloemsma et al., 2019) and Spain (De Bont et al., 2019; Warkentin et al., 2023). A meta-analysis by Huang et al, using mainly cross-sectional studies, indicated that postnatal exposure to ambient air pollutants, especially particulate matter, was associated with increased risk of childhood obesity and weight gain (Huang et al., 2022); prenatal exposure was not assessed in this meta-analysis. Others have, however, reported null associations between postnatal air pollution exposure and weight outcomes in childhood (Alderete et al., 2017; Fioravanti et al., 2018).

The heterogeneous results in the literature point to the necessity of larger studies incorporating pre- and postnatal exposure windows, obesity outcomes across childhood, and different geographical settings. Previous studies on air pollution and childhood obesity were mainly conducted in single countries within a specific region or city and, up to now, no studies have evaluated and compared associations across multiple longitudinal cohorts using individual participant data. Meta-analysis of individual participant data from each trial may improve the quality and scope of derived information in comparison with meta-analysis using aggregate data from publications (Riley et al., 2023). Additionally, this method enables a better control over confounders included in the models, as these are chosen a priori and harmonized across different cohorts.

For these reasons, the aim of this study was to examine associations of pre- and postnatal air pollution exposure with childhood sex- and age-specific measures of body mass index (BMI) and overweight/obesity status, using harmonized individual participant data from 10 European

birth cohorts.

2. Material and methods

2.1. Study population

This study was part of the European Union-funded project LifeCycle, which aims to investigate the effect of diverse early life stressors on health throughout life. LifeCycle has established the EU Child Cohort Network, a Europe-wide network of pregnancy and childhood cohort studies with harmonized data of more than 250,000 mother–child dyads. More detail on the LifeCycle project and the EU Child Cohort Network can be found elsewhere (Jaddoe et al., 2020; Pinot de Moira et al., 2021).

We analyzed data from 10 European birth cohorts. Cohorts were eligible to participate if they had (i) available harmonized data on air pollution exposure estimates, (ii) harmonized data on weight and height measurements in at least one age period of interest and, (iii) harmonized data uploaded on a local server to undergo federated analysis through the DataSHIELD platform (Gaye et al., 2014). We restricted our study population in each cohort to singletons with at least one exposure estimate (pre- or postnatal) and one outcome at any age between 0 to 12 years. The following cohorts were included: ABCD [Amsterdam Born Children and their Development, Amsterdam, the Netherlands, period of enrolment: 2003–2004] (van Eijsden et al., 2011), ALSPAC [Avon Longitudinal Study of Parents and Children, Bristol, UK, 1991–1992] (Fraser et al., 2013), BiB [Born in Bradford, Bradford, UK, 2007–2011] (McEachan et al., 2024; Wright et al., 2013), DNBC [Danish National Birth Cohort, Copenhagen greater area, Denmark, 1996–2002] (Olsen et al., 2001), EDEN [Etude des Déterminants pré et postnatals précoces du développement et de la santé de l'Enfant, Nancy and Poitiers, France, 2003–2006] (Heude et al., 2016), Gen R [the Generation R Study, Rotterdam, the Netherlands, 2002–2006] (Kooijman et al., 2016), INMA [Infancia y Medio Ambiente, Sabadell, Gipuzkoa, Valencia, Spain, 2003–2008] (Guxens et al., 2012), MoBa [Norwegian Mother, Father and Child Cohort Study, Oslo urban area, Norway, 1999–2008] (Magnus et al., 2016), NINFEA [Nascita e INFanzia: gli Effetti dell'Ambiente, Florence, Rome, Turin, Italy, 2005–2016] (Richiardi et al., 2007) and RHEA [RHEA Mother & Child Cohort Study, Crete, Greece, 2007–2008] (Chatzi et al., 2017) (Fig. 1, Table 1). Before enrolment, all participants signed an informed consent in accordance with each center's ethics committee (cohort-specific ethical approvals can be found in **Supplementary Text 1**). The flowchart of cohorts and participants can be found in **Supplementary Fig. 1**.

Fig. 1. Map of the 10 included cohorts from the EU Child Cohort Network.

2.2. Air pollution exposure assessment

The assessment of air pollution exposure was performed at the mother's residential address during pregnancy and the address at the time of each outcome assessment in each cohort. This was done in accordance with a standardized protocol for harmonized urban environment stressor data across all cohorts, developed as part of the LifeCycle project (Jaddoe et al., 2020).

Mean annual concentrations of NO_2 (in $\mu g/m^3$) and $PM_{2.5}$ (in $\mu g/m^3$) were calculated using spatial land-use regression (LUR) models developed as part of the European Study of Cohorts for Air Pollution Effects (ESCAPE) project (Eeftens et al., 2012; Sellier et al., 2014) for cohorts where local ESCAPE models were available: ABCD, BiB, EDEN (NO_2), Gen R, INMA, MoBa (Oslo region), NINFEA and RHEA cohorts. The median LUR model variance (R^2) for $PM_{2.5}$ in ESCAPE project was 71 %, with variations in different regions (e.g., Oslo, Norway – 74 % and Catalunya, Spain – 86 %), which was considered high to moderate, with differences due to local particularities, especially the data availability at each region. For NO_2 (EDEN) seasonalized LUR models accounted for 99 % of the variance of exposure (Sellier et al., 2014). For cohorts where local ESCAPE models were not available, we used air pollution models developed as part of the Effects of Low-Level Air Pollution: A Study in Europe (ELAPSE) project (de Hoogh et al., 2018): ALSPAC, DNBC (Copenhagen greater area) and EDEN ($PM_{2.5}$) cohorts. In ELAPSE, LUR model variance for $PM_{2.5}$ on all available monitoring sites explained 62 % of spatial variation, and the NO_2 model on all available sites explained 59 % of the spatial variation of the measured air pollution concentrations. The ESCAPE and ELAPSE models followed a very similar procedure and thus were comparable. ESCAPE model was applied directly on the participants locations allowing estimates at point resolution. The ELAPSE model was mapped at a 100 × 100 m resolution across Europe (de Hoogh et al., 2018).

To obtain estimates for each relevant exposure period in each cohort and for each participant, temporal adjustment was conducted using background routine monitoring stations. Briefly, for each participant the nearest routine air quality monitoring from the home address was selected and daily time-series data was retrieved to fully cover the target period. The adjustment factor was calculated as the ratio of the pollutant concentration from the routine monitor of each day and the annual average during 2009 and 2010 (baseline years of ESCAPE and ELAPSE models, respectively), following the common methodology developed within ESCAPE project (Alemany et al., 2021; Ballvé et al., 2024; Cruells et al., 2024; Kogevinas et al., 2023; Pedersen et al., 2013).

In the LifeCycle project, average annual exposure to each pollutant was calculated for the entire pregnancy period (prenatal exposure) and for every year of life, from birth up to 12 years of age. For prenatal exposures, we considered whole-pregnancy exposure, not trimester-specific exposures, since there are no indications in the literature for trimester specific effects (e.g. (Laurent et al., 2013; Ng et al., 2017)) and since not all cohorts had available trimester-specific exposure data at the time of the analysis.

Postnatal exposure was defined as the weighted average exposure in the year preceding the outcome assessment (postnatal exposure), as in **Supplementary Text 2**.

2.3. BMI and weight status outcomes

We used two weight-related outcomes based on BMI: (i) continuous BMI z-scores (zBMI) and (ii) categorical variables indicating weight status (normal weight (including underweight), overweight/obesity). Height (in cm), weight (in kg) and age of the offspring were either measured in the clinic/research center or self-reported by parents (**Supplementary Table 1**). BMI was calculated as weight (kg) divided by height squared $(m)^2$ (Woo & Daniels, 2019), and sex- and age-specific

Table 1
Summary of the cohort characteristics.

Cohort	Country	City/area	Birth years	Age range of included children[a]	Number of included children[b]	
					Prenatal	Postnatal
ABCD [Amsterdam Born Children and their Development]	the Netherlands	Amsterdam	2003–2004	0–12 years	3753 (NO_2) 3753 ($PM_{2.5}$)	2690 (NO_2) 2690 ($PM_{2.5}$)
ALSPAC [Avon Longitudinal Study of Parents and Children]	UK	Bristol	1991–1992	0–12 years	7874 (NO_2) 7874 ($PM_{2.5}$)	7874 (NO_2) 7874 ($PM_{2.5}$)
BiB [Born in Bradford]	UK	Bradford	2007–2011	0–5 years	4029 (NO_2) 4031 ($PM_{2.5}$)	4006 (NO_2) 4020 ($PM_{2.5}$)
DNBC [Danish National Birth Cohort]	Denmark	Copenhagen greater area	1996–2002	0–2, 5–12 years	4340 (NO_2) 4340 ($PM_{2.5}$)	3146 (NO_2) 3146 ($PM_{2.5}$)
EDEN [Etude des Déterminants pré et postnatals précoces du développement et de la santé de l'Enfant]	France	Nancy and Poitiers	2003–2006	0–9 years	1186 (NO_2) 1580 ($PM_{2.5}$)	1185 (NO_2) 1578 ($PM_{2.5}$)
Gen R [the Generation R Study]	the Netherlands	Rotterdam	2002–2006	0–12 years	4345 (NO_2) 4345 ($PM_{2.5}$)	4334 (NO_2) 4334 ($PM_{2.5}$)
INMA [Infancia y Medio Ambiente]	Spain	Sabadell, Gipuzkoa, Valencia	2003–2008	0–12 years	1817 (NO_2) 1812 ($PM_{2.5}$)	1815 (NO_2) 1802 ($PM_{2.5}$)
MoBa [Norwegian Mother, Father and Child Cohort Study]	Norway	Oslo urban area	1999–2008	0–9 years	7028 (NO_2) 7053 ($PM_{2.5}$)	6282 (NO_2) 6282 ($PM_{2.5}$)
NINFEA [Nascita e INFanzia: gli Effetti dell'Ambiente]	Italy	Florence, Rome, Turin	2005–2016	0–12 years	1961 (NO_2) 1961 ($PM_{2.5}$)	1765 (NO_2) 1765 ($PM_{2.5}$)
RHEA [RHEA Mother & Child Cohort Study]	Greece	Crete	2007–2008	0–5 years	357 (NO_2) 357 ($PM_{2.5}$)	356 (NO_2) 356 ($PM_{2.5}$)

[a] Children with available harmonized data on air pollution exposure estimates and harmonized data on weight and height measurements in at least one age period of interest;
[b] Children included in the analyses (complete cases, with no missing covariates), per cohort, by period and pollutant, with some exposure and outcome data.

zBMI were estimated according to the World Health Organization (WHO) reference curves (de Onis, 2007; WHO, 2006). zBMI values ± 5 standard deviations (SD) from the population median were excluded. Weight status was defined according to the WHO as follows: For children < 5 years: Normal weight (inclusive underweight) for zBMI<+2SD, overweight for zBMI≥+2 and zBMI<+3SD, and obesity for zBMI≥+3SD (WHO, 2006). For children ≥ 5 years: Normal weight (inclusive underweight) for zBMI<+1SD, overweight for zBMI≥+1 and zBMI<+2SD, and obesity for zBMI≥+2SD (de Onis, 2007). We considered weight status as overweight/obesity *vs.* normal weight. The age periods used for the outcome assessment were: 0–2, 2–5, 5–9 and 9–12 years, which represent key developmental periods (infancy, pre-school age, adiposity rebound, and early puberty). In cases when the child had multiple zBMI measurements within an age period, the latest available measurement within the period was used.

2.4. Confounders

We defined confounders that were known or a plausible risk factor associated with exposure to air pollution and childhood zBMI. We used Directed Acyclic Graphs (DAGs) to identify these confounders in pre- and postnatal periods along with potential colliders that should not be adjusted for (**Supplementary Fig. 2**). Prenatal analyses included child sex (male/female) and exact age (in months) at follow-up, maternal educational level at birth (categories described below) as an indicator of individual-level socioeconomic status (SES), maternal pre-pregnancy BMI (self-report or clinical measurement, in kg/m^2), maternal smoking during pregnancy (yes/no), and area-level SES measured during pregnancy. In our DAGs, maternal pre-pregnancy BMI was considered to be a confounder rather than a mediator, as is common in studies with a similar research question (De Bont et al., 2020; Fioravanti et al., 2018; Fleisch et al., 2015, 2017; Frondelius et al., 2018; Nieuwenhuijsen et al., 2019). For the postnatal analyses, we further included prenatal exposure to air pollution as a confounder in the case of weak-to-moderate correlation with postnatal exposure (correlation coefficient <0.8). For the multi-city cohorts EDEN, INMA, and NINFEA, we further adjusted the models for city, and for ABCD, BiB, and Gen R, we further included self-reported mother's ethnicity in the models (Western origin/Non-western origin/Mixed). This was done based on previous findings on different levels of air pollution depending on the city in the EDEN, INMA and NINFEA cohorts and the great variability of ethnic origin within the study population of ABCD, BiB and Gen R cohorts, which may have had an effect on zBMI outcomes in these specific populations (De Hoog et al., 2011; Guxens et al., 2014; Schembari et al., 2015; West et al., 2013).

Two SES measures were included in this study: one individual-level (maternal education) and one area-level (area deprivation index). These measures, although correlated, can capture different constructs either of an individual's vulnerability to or potential for exposure to air pollution

(Hajat et al., 2015). Maternal education level was based on the highest ongoing or completed education at the time of delivery, and was coded according to the International Standard Classification of Education 97 (ISCED-97) in three categories: low (no education to lower secondary; ISCED-97 categories 0–2), medium (upper and post-secondary; ISCED-97 categories 3–4) and high (degree and above; ISCED-97 categories 5–6) (UNESCO, 2006). An area deprivation index was used as an indicator of prenatal area-level SES. This index was derived from country-specific indicators categorized into tertiles, where 1 means the least deprived area and 3 means most deprived area (**Supplementary Text 3**). This index was not available in MoBa.

As sensitivity analyses, we further included in the models two urban characteristics: a cohort-specific walkability index and population density. These two measures were selected based on the described association between various urban characteristics on air pollution levels and childhood zBMI. Countries with higher total population exhibit higher air pollution emissions per capita (with the opposite happening when looking at density in urban areas) (Castells-Quintana et al., 2021). City density is also associated with increases in childhood BMI (De Bont et al., 2020) and walkable cities are, on the other hand, related to lower childhood BMI (Vrijheid et al., 2020), as well as lower air pollution levels (Nieuwenhuijsen, 2021). The walkability index used in the current analysis included the average of the following four components capturing differences in the physical environment: (i) Land use Shannon's Evenness Index (calculated as the proportional abundance of each land use type multiplied by that proportion, divided by the logarithm of the number of land use types within a 300 m buffer), (ii) Facility richness (calculated as the number of different facility types (e.g. restaurants, shops, schools, medical centres) within a 300 m buffer divided by the maximum (ten) potential types (range 0–1), (iii) Population density and (iv) Connectivity index (described as the number of street intersections within a 300 m buffer) with a range of 0 (less walkable) to 1 (most walkable) (Binter et al., 2022; Frank et al., 2006). Each component was converted to deciles before entering to formula to have equal weight. These four indices were summed and divided by four, resulting in a walkability index ranging from 0 to 1. Population density was expressed as the number of inhabitants per km^2 at the home address and was calculated using the Global Human Settlement Layer (*Global Human Settlement* Layer, 2015) for all cohorts except MoBa for which local data were used (Robinson et al., 2018).

2.5. Statistical analysis

This study used harmonized data, which were analyzed remotely through DataSHIELD (dsBase package, version 6.1.0) (Gaye et al., 2014).

We used a two-stage individual participant data meta-analysis approach to evaluate associations between pre- and postnatal air pollution exposure and zBMI and overweight/obesity status (overweight/obesity *vs.* normal weight). First, associations were estimated separately for each cohort using linear and logistic mixed effects models with repeated measures of exposures and outcomes (child id was treated as random effect and confounders as fixed effects). Then, cohort-specific coefficients and standard errors were combined using random-effects meta-analysis to obtain overall association estimates. Common and random effects estimates were described for adjusted models; in the common effects model it is assumed that there is one true effect size underlying all the studies in the meta-analysis, and in the random effects model, it is allowed that true effect sizes differ from study to study (Borenstein et al., 2010). Given the heterogeneity between studies in the current meta-analysis, we have focused our results on the random effects estimates. All analyses were adjusted for the main confounders selected by the DAGs. Results are described as beta estimates (95 % confidence intervals (CI)), for zBMI for every 10 $\mu g/m^3$ increase in air pollution levels, and odds ratios (OR) (95 % CI) for overweight/obesity status (*vs.* normal weight). Additionally, we examined between-study heterogeneity using the I^2 statistic (%). We performed longitudinal analyses on the associations on overall zBMI across all age periods and stratified by age period (0–2, 2–5, 5–9 and 9–12 years). We conducted only single-pollutant analysis; due to the moderate-to-very-strong correlations between pollutants (**Supplementary Fig. 3**), two-pollutant models were not performed.

We performed several sensitivity analyses. First, in order to assess potential effect modification by sociodemographic characteristics, we repeated analyses stratifying by maternal education (low/medium/high), as indicator of individual-level SES, and by residential area-level deprivation index (low/medium/high) as indicator of area-level SES. Second, aiming to explore if associations between air pollution exposure and child weight outcomes are independent of urban characteristics, we further adjusted the models for walkability and population density (separately and together in the same model). Third, to detect whether highly influential cohorts affected the overall effect estimates or heterogeneity we excluded each cohort in turn using a "leave-one-out analysis" approach (Viechtbauer & Cheung, 2010).

3. Results

A total of 37111 (prenatal) and 33860 (postnatal) mother–child dyads in the 10 cohort studies from eight European countries were included in this study. As described in Table 2, mother–child pairs from the BiB cohort (UK) were most likely to live in a high deprivation area (84.3 %), and mother–child pairs from the INMA cohort (Spain) were least likely to live in a high deprivation area (12 %). Child overweight/obesity status varied between cohorts and age periods, with prevalence ranging between cohorts from 12.3 to 40.5 % at 0–2 years, 16.7–35.3 % at 2–5 years, 12.5–40.7 % at 5–9 years, and 10.7–43.8 % at 9–12 years. Full characteristics of cohorts are described in **Supplementary Table 2**. Full cohort characteristics showed to be very similar to the study population, with exception to maternal education, in which we see participants with lower educational level in the full cohorts of NINFEA, MoBa and DNBC, compared to the study population. This might be explained by the fact that the study population included mainly the urban areas of each cohort, aiming to estimate urban exposures (three major Italian cities (Florence, Rome, Turin) in the NINFEA cohort, Oslo urban area in the MoBa cohort, and Copenhagen greater area in the DNBC cohort). Also, in the full cohort of EDEN we observe a lower proportion of participants from Western origin, compared to the study population (study population included the urban areas of Nancy and Poitiers). Cohort-specific information on prenatal and postnatal air pollution weighted average exposure throughout the age periods, and other urban exposures, are described in Table 3**.** During pregnancy, higher NO_2 and $PM_{2.5}$ levels were seen in the Italian cohort (NINFEA), and lower levels were seen in Greece (RHEA) and Norway (MoBa), compared to the other cohorts (Table 3). Correlation plots between air pollutants, by cohort and time of exposure (pre- and postnatal), are displayed in **Supplementary Fig. 3**.

Meta-analyses of the estimates from the 10 cohorts showed no evidence of an association between exposure to NO_2 during pre- and postnatal periods, and zBMI and overweight/obesity across childhood with overall estimates close to null (e.g., prenatal exposure to NO_2 and zBMI per increment of 10 $\mu g/m^3$: random effects β(95 %CI) 0.01(−0.01; 0.02); postnatal exposure to NO_2 and zBMI per increment of 10 $\mu g/m^3$: β(95 %CI) −0.01(−0.04; 0.02) (Fig. 2). The association of pre- and postnatal NO_2 exposure with zBMI and overweight/obesity status showed considerable heterogeneity between cohorts (I^2 range 25–74 %) (Fig. 2). Some cohort-specific associations were observed: For prenatal NO_2 exposure, a positive association was observed in ABCD (zBMI and overweight/obesity status) and negative, but non-significant, associations were observed for ALSPAC (zBMI and overweight/obesity status). For postnatal NO_2 exposure, negative associations were observed in BiB, EDEN and INMA (zBMI), ALSPAC (overweight/obesity) and RHEA (zBMI and overweight/obesity). In contrast, the Dutch Gen R cohort

Table 2
Study population characteristics – outcomes and covariates.

	ABCD	ALSPAC	BiB	DNBC	EDEN	Gen R	INMA	MoBa	NINFEA	RHEA
Total N										
Prenatal	3753	7874	4029 (NO_2), 4031 ($PM_{2.5}$)	4340	1186 (NO_2), 1580 ($PM_{2.5}$)	4345	1817 (NO_2), 1812 ($PM_{2.5}$)	7028 (NO_2), 7053 ($PM_{2.5}$)	1961	357
Postnatal	2690		4006 (NO_2), 4020 ($PM_{2.5}$)	3146	1185 (NO_2), 1578 ($PM_{2.5}$)	4334	1815 (NO_2), 1802 ($PM_{2.5}$)	6282	1765	356
Area-level deprivation[a] – n (%)										
Low	1178 (22.34)	2711 (29.98)	302 (3.05)	3459 (54.46)	654 (38.88)	1302 (15.22)	939 (48.25)	–	774 (39.25)	161 (42.04)
Medium	571 (10.83)	3107 (34.35)	1255 (12.69)	1576 (24.81)	429 (25.51)	1786 (20.87)	773 (39.72)	–	662 (33.57)	122 (31.85)
High	3524 (66.83)	3226 (35.67)	8336 (84.26)	1316 (20.72)	599 (35.61)	5469 (63.91)	234 (12.02)	–	536 (27.18)	100 (26.11)
Missing	*341 (6.07)*	*33 (0.36)*	*5 (0.05)*	*1863 (22.68)*	*40 (2.32)*	*72 (0.83)*	*6 (0.31)*		*481 (19.61)*	*515 (57.35)*
Maternal education at birth – n (%)										
High	2909 (52.22)	1235 (14.46)	2528 (27.73)	4174 (68.63)	957 (55.87)	3473 (45.2)	668 (34.96)	7486 (85.71)	1665 (69.78)	283 (31.55)
Medium	1512 (27.14)	5906 (69.16)	1416 (15.53)	1070 (17.59)	656 (38.3)	3420 (44.51)	784 (41.03)	1169 (13.38)	641 (26.87)	458 (51.06)
Low	1150 (20.64)	1399 (16.38)	5174 (56.74)	838 (13.78)	100 (5.84)	790 (10.28)	459 (24.02)	79 (0.9)	80 (3.35)	156 (17.39)
Missing	*43 (0.77)*	*537 (5.92)*	*780 (7.88)*	*2132 (25.96)*	*9 (0.52)*	*946 (10.96)*	*41 (2.1)*	*582 (6.25)*	*67 (2.73)*	*1 (0.11)*
Maternal pre-pregnancy BMI (kg/m^2)[b] – Median (P5, P95)	22.2 (18.59, 30.85)	–	27.73 (20.57, 33.51)	21.72 (18.41, 28.71)	22.1 (18.14, 31.99)	22.66 (18.69, 32.35)	22.53 (18.68, 31.9)	–	21.34 (17.92, 29.06)	23.51 (18.81, 34.59)
Missing	*433 (7.71)*	*7874 (100.0)*	*5537 (55.94)*	*559 (6.81)*	*33 (1.92)*	*2088 (24.2)*	*18 (0.92)*	*9316 (100)*	*41 (1.67)*	*6 (0.67)*
Maternal smoking during pregnancy – n(%)										
Yes	617 (10.99)	2024 (25.0)	1588 (16.07)	2083 (25.6)	433 (25.22)	1910 (25.95)	605 (31.43)	1561 (16.76)	196 (8.08)	283 (33.41)
Missing	*2 (0.04)*	*980 (10.8)*	*19 (0.19)*	*76 (0.93)*	*5 (0.29)*	*1270 (14.72)*	*27 (1.38)*	*1 (0.01)*	*26 (1.06)*	*51 (5.68)*
Maternal ethnicity - n(%)										
Western origin	3193 (57.26)	8329 (98.38)	4130 (41.7)	–	1474 (85.6)	4643 (56.98)	1829 (95.51)	–	–	891 (99.66)
Non-western origin	1650 (29.59)	137 (1.62)	5571 (56.3)	–	8 (0.47)	2704 (33.19)	86 (4.49)	–	–	3 (0.34)
Mixed	733 (13.15)	0 (0)	185 (1.87)	–	6 (0.35)	801 (9.83)	0 (0)	–	–	0 (0)
Missing	*38 (0.68)*	*611 (6.73)*	*12 (0.12)*	*8214 (100)*	*234 (13.6)*	*481 (5.57)*	*37 (1.90)*	*9316 (100)*	*2453 (100)*	*4 (0.45)*
Child sex – n (%)										
Male	2787 (49.64)	4579 (50.45)	5094 (51.46)	4194 (51.06)	898 (52.15)	4350 (50.41)	1006 (51.54)	4727 (50.74)	1264 (51.53)	478 (53.23)
Child age (months) – Median (P5, P95)										
0–2 years	14 (12, 20)	12 (12, 12)	0 (0, 23)	12 (12, 14)	12 (0, 15)	14 (4, 19)	14 (12, 18)	12 (1, 16)	12 (3, 18)	14 (2, 20)
2–5 years	44 (26, 54)	49 (31, 49)	51 (25, 59)	–	48 (35, 57)	37 (24, 47)	51 (48, 55)	36 (24, 36)	48 (48, 48)	49 (48, 57)
5–9 years	69 (61, 85)	99 (69, 103)	–	85 (79, 88)	66 (60, 70)	72 (68, 89)	93 (75, 106)	84 (60, 84)	84 (75.35, 93)	–
9–12 years	128 (120, 141)	140 (127, 141)	–	133 (130, 138)	–	116 (113, 122)	131 (111, 140)	–	122 (119, 129)	–
Child zBMI[c] – Median (P5, P95)										
0–2 years	0.48 (−1.14, 2.04)	0.79 (−0.68, 2.29)	−0.37 (−2.31, 1.57)	0.3 (−1.47, 2.12)	0.36 (−1.57, 1.94)	0.55 (−1.15, 2.08)	0.42 (−1.2, 2.03)	0.25 (−1.32, 1.82)	0.05 (−2.11, 2.12)	0 (−1.77, 1.77)
2–5 years	0.27 (−1.26, 1.95)	0.61 (−0.84, 2.17)	0.46 (−1.18, 2.44)	–	0.14 (−1.37, 1.66)	0.37 (−1.19, 2.06)	0.51 (−0.95, 2.45)	0.43 (−1.31, 2.1)	0.08 (−1.85, 2.06)	0.59 (−1.07, 2.81)
5–9 years	0.11 (−1.34, 1.97)	0.24 (−1.56, 2.42)	–	−0.06 (−1.61, 1.51)	−0.01 (−1.39, 1.52)	0.36 (−1.04, 2.29)	0.71 (−1.03, 2.9)	−0.02 (−1.74, 1.67)	−0.02 (−1.9, 2.09)	–
9–12 years	0.02 (−1.66, 2.16)	0.36 (−1.52, 2.4)	–	−0.28 (−1.97, 1.42)	–	0.29 (−1.3, 2.31)	0.76 (−1.29, 2.71)	–	0.07 (−2.23, 1.91)	–
Child overweight/obesity status[c] – n (%)										
0–2 years	1357 (28.7)	477 (40.49)	1003 (12.33)	1122 (25.1)	418 (25.32)	2107 (31.08)	510 (26.32)	1797 (20.96)	444 (21.38)	147 (17.21)

(*continued on next page*)

Table 2 (*continued*)

	ABCD	ALSPAC	BiB	DNBC	EDEN	Gen R	INMA	MoBa	NINFEA	RHEA
2–5 years	1014 (22.05)	358 (32.05)	2315 (29.96)	–	254 (16.67)	1646 (25.5)	502 (30.91)	1577 (28.4)	374 (19.63)	281 (35.26)
5–9 years	782 (18.57)	2111 (25.66)	–	644 (12.49)	162 (13.81)	1709 (25.58)	581 (40.74)	1072 (15.38)	247 (21.15)	–
9–12 years	726 (22.39)	2504 (31.7)	–	463 (10.65)	–	1466 (26.39)	412 (43.83)	–	95 (20.08)	–

ABCD: Amsterdam Born Children and their Development; ALSPAC: Avon Longitudinal Study of Parents and Children; BiB: Born in Bradford; DNBC: Danish National Birth Cohort; EDEN: Etude des Déterminants pré et postnatals précoces du développement et de la santé de l'Enfant; Gen R: Generation R Study; INMA: Infancia y Medio Ambiente; MoBa: Norwegian Mother, Father and Child Cohort Study; NINFEA: Nascita e INFanzia: gli Effetti dell'Ambiente; RHEA: Rhea Mother & Child Cohort Study; P: Percentile; BMI: Body mass index, zBMI: Body mass index z-score.

[a] Area-level deprivation defined in each cohort, as described in Supplementary Text 3;

[b] Maternal pre-pregnancy BMI defined as weight/height^2;

[c] Sex- and age- specific zBMI and child weight status were estimated according to the World Health Organization (WHO) reference curves (de Onis, 2007; WHO, 2006).

reported a positive association between postnatal NO_2 exposure with zBMI (β(95 %CI) 0.04(0.02; 0.07)) and overweight/obesity status (OR (95 %CI) 1.14(1.02; 1.27)).

For prenatal $PM_{2.5}$ exposure, increased exposure was associated with an increased risk of overweight/obesity (OR(95 %CI) 1.23 (1.01; 1.51), whereas a small non-significant increase in zBMI was observed (β(95 % CI) 0.04 (−0.03; 0.10) (Fig. 3). There were no associations with postnatal $PM_{2.5}$ exposure. Associations for $PM_{2.5}$ exposure also showed considerable heterogeneity between cohorts, with I^2 values ranging from 43 to 89 % (Fig. 3). We observed cohort-specific associations in both positive and negative directions: For prenatal $PM_{2.5}$ exposure, negative associations were found for ALSPAC (zBMI), and positive associations were seen for Gen R (zBMI and overweight/obesity). For postnatal $PM_{2.5}$ exposure, negative associations were seen in ALSPAC, INMA and RHEA (zBMI and overweight/obesity), BiB (zBMI, only) and positive associations were seen for Gen R (zBMI and overweight/obesity).

Fig. 4 shows results of the meta-analyses stratified by age period of the outcome assessment. Associations between air pollution exposures and zBMI and overweight/obesity status were close to null in most age periods and did not reach statistical significance. In the period between 9–12 years, higher exposure to $PM_{2.5}$ during pregnancy was associated with a statistically significant increase in zBMI per increments of 10 μg/m^3 (β(95 %CI) 0.13(0.03; 0.24) and in the risk of overweight/obesity (OR(95 %CI) 1.29(1.04; 1.60)).

Analyses stratified by individual- and area-level SES showed little difference in associations by SES, with overlapping confidence intervals between the SES strata. Nevertheless, in the most deprived area-level SES group (T3) a statistically significant association was observed between prenatal $PM_{2.5}$ and increased zBMI per increments of 10 μg/m^3 (β(95 %CI) 0.10(0.01; 0.18)). A weaker positive association was found between prenatal NO_2 exposure and zBMI per increments of 10 μg/m^3 in this SES group (β(95 %CI) 0.03(0.00; 0.06)) (**Supplementary Figs. 4 and 5**). Some negative associations in specific SES groups were seen in the postnatal exposure period (e.g., in the medium deprivation group of area-level SES for postnatal $PM_{2.5}$ exposure and zBMI per increments of 10 μg/m^3 (β(95 %CI) −0.19(−0.34 to −0.04)).

Models with further adjustments by walkability and population density showed similar effect estimates to our main models without these adjustments (**Supplementary Fig. 6**). Sensitivity analyses excluding one cohort at the time mostly showed minor changes in β and OR estimates that do not change the main conclusions. An exception was the association between prenatal NO_2 and overweight/obesity risk which increased from 1.01 (95 %CI 0.96; 1.07) (overall random-effect OR) to 1.28 (95 %CI 1.04;1.58) when removing the DNBC cohort. Decreases in the heterogeneity (I^2) were observed mainly for prenatal NO_2 exposure, especially when removing the ABCD cohort, and for prenatal $PM_{2.5}$ exposure, especially when removing the Generation R cohort. (**Supplementary Tables 3 and 4**).

4. Discussion

In this meta-analysis of individual participant data from 10 European cohorts and a sample size of 37111 (prenatal) and 33860 (postnatal) mother–child pairs, pre- and postnatal exposure to NO_2 and $PM_{2.5}$ showed few associations with obesity-related outcomes across key developmental periods during childhood. Exposure to $PM_{2.5}$ during pregnancy was associated with higher overweight/obesity risk across childhood, and specifically with higher zBMI and overweight/obesity risk in one age period (9–12 years). Considerable heterogeneity between cohorts was noted, with some cohorts showing increased zBMI or overweight/obesity risk with increased air pollution exposure, and others showing negative or null associations.

This study investigated both pre- and postnatal ambient air pollution exposure and obesity-related outcomes during childhood in a multi-cohort study using individual participant data, rather than aggregate estimates from publications. Associations between pre- and postnatal air pollution exposures and childhood obesity-related outcomes have been studied in a number of single cohort studies, but results are inconsistent. Regarding prenatal air pollution exposure, various studies have shown that exposure to pollutants during pregnancy, such as $PM_{2.5}$, may be associated with decreased weight and BMI at birth and in first years of life (Bekkar et al., 2020; Fossati et al., 2020; Hung et al., 2022). Two studies in the US suggested a complicated and dynamic exposure-weight relationship between the exposure to air pollution in prenatal life and weight outcomes. Infants in the Project Viva cohort exposed to higher prenatal traffic-related air pollution ($PM_{2.5}$ and black carbon) exhibited reduced fetal growth at birth and rapid postnatal weight-for-length gain between 0–6 months of age (Fleisch et al., 2015). More recently, Ji and colleagues found that those children exposed to higher levels of NO_2 had faster "catch-up growth" rates in early infancy, which may lead to increased risk of obesity later in life (Ji et al., 2023). However, other studies that investigated maternal prenatal particulate matter exposure and child weight-related outcomes, did not show significant associations with childhood BMI (Huang et al., 2019; Sears et al., 2019), cardiometabolic health (Fleisch et al., 2017) nor BMI trajectories (Fleisch et al., 2019). In the current study, we observed an association between prenatal $PM_{2.5}$ exposure and greater overweight/obesity risk across childhood, and specifically with higher zBMI and overweight/obesity risk in one age period (9–12 years). However, this age-specific analysis was based on less cohorts than the repeated outcome analysis due to less cohorts having data in this age group, and this result needs to be interpreted with caution. Experimental studies in animals have shown that $PM_{2.5}$ exposure during pregnancy led to decreased body and tail length, birth weight, and survival rates of the young mice (Huang et al., 2023), the development of increased body weight, altered blood pressure, and increased susceptibility to cardiovascular disease and heart failure in offspring's adulthood (Weldy et al., 2014), besides increased risk for metabolic syndrome in the offspring (Zhang et al., 2019).

Table 3
Study population characteristics – prenatal and postnatal air pollution estimates[a] and other urban characteristics.

	ABCD	ALSPAC	BiB	DNBC	EDEN	Gen R	INMA	MoBa	NINFEA	RHEA
Total N										
Prenatal	3753	7874	4029 (NO_2), 4031 ($PM_{2.5}$)	4340	1186 (NO_2), 1580 ($PM_{2.5}$)	4345	1817 (NO_2), 1812 ($PM_{2.5}$)	7028 (NO_2), 7053 ($PM_{2.5}$)	1961	357
Postnatal	2690		4006 (NO_2), 4020 ($PM_{2.5}$)	3146	1185 (NO_2), 1578 ($PM_{2.5}$)	4334	1815 (NO_2), 1802 ($PM_{2.5}$)	6282	1765	356
Prenatal air pollution exposure (µg/m^3)[b] – Median (P5, P95)										
NO_2	36.62 (27.53, 48.16)	27.21 (19.22, 33.38)	23.04 (18.16, 29.98)	47.27 (38.72, 57.91)	21.28 (11.3, 48.92)	38.35 (30.41, 49.36)	26.23 (10.13, 51.65)	20.66 (10.87, 37.17)	49.25 (19.49, 76.25)	11.39 (7.9, 21.89)
Missing	*118 (2.1)*	*193 (2.13)*	*15 (0.15)*	*634 (7.72)*	*464 (27)*	*37 (0.43)*	*108 (5.53)*	*962 (10.33)*	*352 (14.35)*	*1 (0.11)*
$PM_{2.5}$	18.47 (16.78, 21.02)	13.33 (11.92, 14.67)	10.43 (8.62, 12.66)	18.22 (14.89, 20.9)	17.11 (14.17, 21.1)	19.62 (16.88, 23.82)	14.72 (10.89, 18.89)	10.8 (7.73, 14.46)	24.39 (9.64, 35.71)	14.18 (11.02, 18.67)
Missing	*118 (2.1)*	*193 (2.13)*	*5 (0.05)*	*634 (7.72)*	*2 (0.12)*	*37 (0.43)*	*114 (5.84)*	*937 (10.06)*	*352 (14.35)*	*1 (0.11)*
Postnatal NO_2 – weighted average (Median (P5, P95))										
0–2 years	35.01 (26.67, 45.04)	27.18 (19.69, 33.11)	24.21 (18.76, 34.79)	45.12 (37.14, 53.34)	20.57 (11.06, 46.91)	37.36 (30.29, 47.23)	27.53 (9.54, 50.79)	35.16 (24.62; 44.15)	48.67 (18.04, 75.18)	13.65 (10.59, 24.8)
2–5 years	34.32 (26.27, 43.86)	27.09 (19.72, 32.76)	25.94 (13.71, 33.54)	–	17.92 (10.48, 39.5)	35.08 (27.94, 43.68)	22.25 (8.54, 46.68)	35.19 (24.4, 45.44)	45.41 (16.99, 61.07)	13.92 (10.71, 25.03)
5–9 years	32.09 (25.01, 41.43)	23.57 (16.17, 29.57)	–	40.28 (32.12, 50.9)	15.99 (10.12, 33.73)	32.9 (24.08, 41.05)	22.75 (8.15, 46.85)	33.28 (23.27, 44.47)	44.15 (19.98, 59.05)	–
9–12 years	15.24 (11.15, 32.29)	22.08 (15.17, 26.9)	–	33.39 (26.78, 39.8)	–	29.03 (17.8, 36.67)	26.8 (3.04, 43.76)	–	40.99 (22.61, 51.48)	–
Postnatal $PM_{2.5}$ – weighted average (Median (P5, P95))										
0–2 years	17.56 (16.69, 19.48)	13.37 (12.18, 14.41)	10.7 (8.9, 14.75)	18.44 (15.19, 21.04)	16.67 (14.25, 18.93)	17.99 (16.96, 22.78)	14.89 (10.72, 18.72)	9.84 (6.80; 13.7)	24.44 (8.68, 36.61)	17.29 (14.95, 20.23)
2–5 years	17.71 (16.81, 19.34)	13.39 (12.23, 14.48)	11.87 (6.12, 14.1)	–	15.86 (13.5, 18.39)	17.49 (16.31, 19.28)	13.31 (10.53, 15.72)	9.14 (6.45; 12.1)	22.77 (8.32, 26.92)	17.83 (15.37, 20.69)
5–9 years	17.02 (16.22, 18.37)	11.62 (10.22, 13.59)	–	16.07 (13.12, 19.37)	15.37 (13.44, 17.81)	16.18 (15.53, 17.38)	13.69 (10.61, 15.85)	7.66 (5.67; 10.57)	21.74 (12.15, 26.76)	–
9–12 years	7.42 (7.01, 15.3)	11.04 (9.8, 11.99)	–	14.34 (11.93, 16.58)	–	14.94 (12.9, 17.42)	12.65 (9.16, 14.65)	–	20.13 (14.4, 22.84)	–
Population density during pregnancy (persons/km^2 in 100 m) – Median (P5, P95)	6752.25 (3132.1, 6842.3)	4320 (775.45, 11616)	5552 (1225.04, 19312)	8233.43 (2047.07, 19800.6)	2092.98 (114.51, 7595.57)	4136.41 (2457.38, 4136.41)	6121.41 (608.85, 12049.58)	6412.18 (1934.02, 25572.41)	10202.16 (5321.16, 10202.16)	5458.87 (333.25, 6887.29)
Missing	*126 (2.24)*	*210 (2.31)*	*49 (0.5)*	*503 (6.12)*	*147 (8.54)*	*952 (11.03)*	*165 (8.45)*	*1038 (11.14)*	*363 (14.8)*	*19 (2.12)*
Walkability during pregnancy (0–1 range; 300 m buffer) – Median (P5, P95)	0.38 (0.25, 0.48)	0.28 (0.2, 0.42)	0.32 (0.22, 0.45)	0.38 (0.25, 0.55)	0.28 (0.2, 0.4)	0.35 (0.28, 0.45)	0.32 (0.22, 0.48)	0.3 (0.17, 0.45)	0.38 (0.3, 0.48)	0.38 (0.2, 0.5)
Missing	*115 (2.05)*	*301 (3.32)*	*9 (0.09)*	*503 (6.12)*	*404 (23.46)*	*957 (11.09)*	*133 (6.81)*	*2224 (23.87)*	*353 (14.39)*	*237 (26.39)*

ABCD: Amsterdam Born Children and their Development; ALSPAC: Avon Longitudinal Study of Parents and Children; BiB: Born in Bradford; DNBC: Danish National Birth Cohort; EDEN: Etude des Déterminants pré et postnatals précoces du développement et de la santé de l'Enfant; Gen R: Generation R Study; INMA: Infancia y Medio Ambiente; MoBa: Norwegian Mother, Father and Child Cohort Study; NINFEA: Nascita e INFanzia: gli Effetti dell'Ambiente; RHEA: Rhea Mother & Child Cohort Study; P: Percentile.

[a] Values are described as weighted average air pollution levels in the different age periods;

[b] Ambient air pollution levels were estimated using land-use regression (LUR) models developed within the European Study of Cohorts for Air Pollution Effects (ESCAPE) project (Eeftens et al., 2012) and through the Effects of Low-Level Air Pollution: A Study in Europe (ELAPSE) project (de Hoogh et al., 2018).

Further studies are necessary to better understand how prenatal air pollution exposure may affect adiposity-related outcomes in different key developmental periods during childhood.

We found no evidence for an association between postnatal air pollution exposure and childhood zBMI and overweight/obesity status. The literature has also been inconsistent with respect to findings on the association between postnatal air pollution exposure and weight outcomes in childhood. In China, exposure to PM_1, $PM_{2.5}$, PM_{10} and NO_2 was associated with increased zBMI, waist circumference and waist-to-height ratio, and higher prevalence of both general and central obesity, in children and adolescents (Zhang et al., 2021). In a recent natural experiment study in Catalonia, Spain, those children and

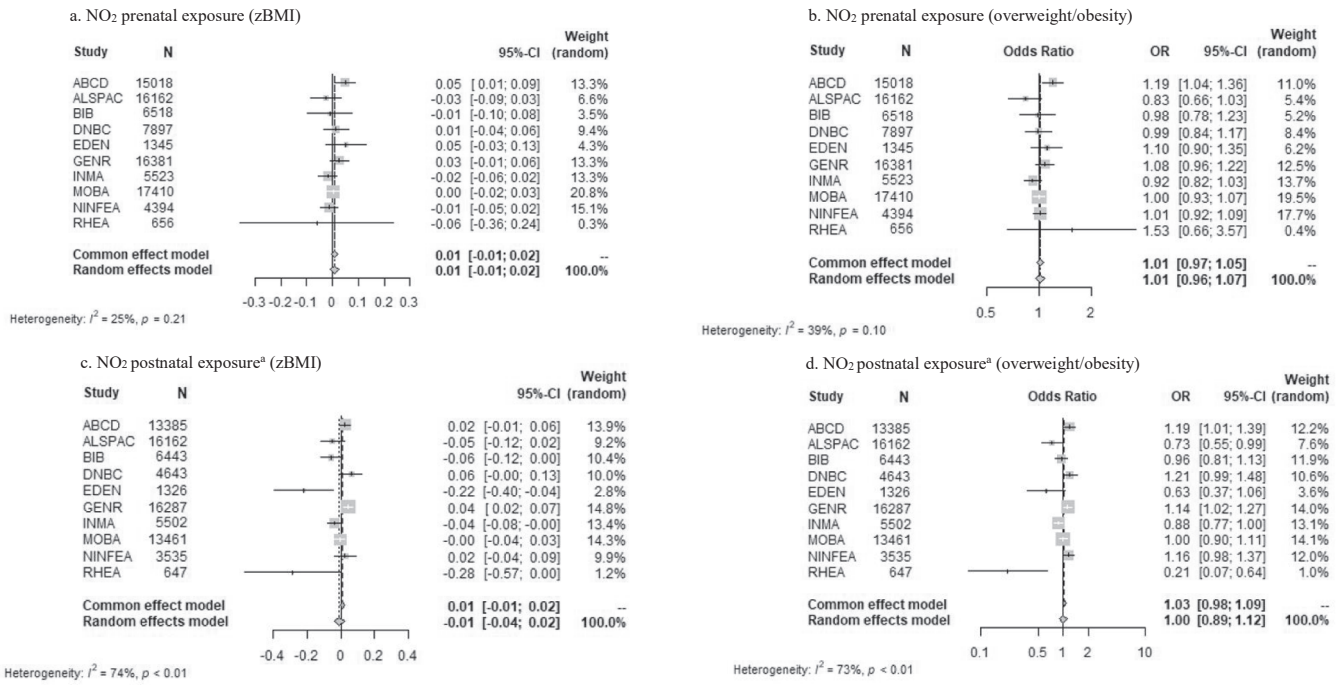


Fig. 2. Forest plot of associations between pre- and postnatal exposure[a] to NO_2 and childhood zBMI and overweight/obesity using two-stage meta-analysis. ABCD: Amsterdam Born Children and their Development; ALSPAC: Avon Longitudinal Study of Parents and Children; BiB: Born in Bradford; DNBC: Danish National Birth Cohort; EDEN: Etude des Déterminants pré et postnatals précoces du développement et de la santé de l'Enfant; Gen R: Generation R Study; INMA: Infancia y Medio Ambiente; MoBa: Norwegian Mother, Father and Child Cohort Study; NINFEA: Nascita e INFanzia: gli Effetti dell'Ambiente; RHEA: Rhea Mother & Child Cohort Study; zBMI: Body mass index z-score; OR: Odds ratio; CI: Confidence intervals. Prenatal exposure models adjusted for sex, child age (in months), individual-level SES (maternal education), pre-pregnancy BMI, smoking during pregnancy, and area-level SES. Postnatal exposure models were further adjusted for prenatal air pollution exposure. Further adjustments: INMA, NINFEA and EDEN: for city, in Gen R, ABCD and BiB: for ethnicity. I^2: Test for heterogeneity (%). a Values are described as weighted average postnatal air pollution levels in the year before zBMI measurement, in the different age periods.

adolescents who moved to more polluted areas (NO_2, PM_{10} and $PM_{2.5}$) showed prospective increases in zBMI (Warkentin et al., 2023). The study by De Bont and colleagues, also in Catalonia, highlighted that ambient air pollution exposure in childhood, especially at school, was associated with 30 % increased odds of having overweight or obesity (De Bont et al., 2019). Another study, using the PIAMA birth cohort in the Netherlands, showed that NO_2 exposure throughout childhood was associated with an increased odds (adjusted OR = 1.40) of overweight in children and adolescents aged 3–17 years (Bloemsma et al., 2019). However, others reported null findings. A study in Italy did not find a significant association between long-term exposure to air pollution ($PM_{2.5}$, PM_{10}, NO_2, NO_x, coarse particles, and $PM_{2.5abs}$) and traffic density, and several body weight measures including BMI, waist circumference, waist-to-height ratio and prevalence of overweight/obesity in 4–8-year-olds (Fioravanti et al., 2018). In another study in the south of the UK, PM_{10} exposure in preschool-age (4–5 years) was associated with small relative risk of overweight/obesity at 10 years, however not the other pollutants ($PM_{2.5}$, NO_x) (Wilding et al., 2020). A recent systematic review concluded that there is strong evidence of association between NO_2 and NO_x (which are traffic-related pollutants) and childhood obesity, but evidence is still weak for PM_{10} and $PM_{2.5}$ (which are driven to a lesser degree by local traffic). It is argued that the biochemical mechanisms in which NO_2 is involved in the human body might also impact the onset of obesity (Malacarne et al., 2022). Besides variability of associations between air pollution and weight by specific air pollutants, this link may also vary by sex, age group (children *vs.* adults) (An et al., 2018a), study design (cross-sectional *vs.* longitudinal), methodological disparities in body weight and air pollution measurements, and heterogeneity of study populations (Zhang et al., 2021).

Our study finds prenatal, but not postnatal, exposure to $PM_{2.5}$ to be associated with obesity-related outcomes in childhood. From the previous epidemiological studies, described above, there is no clear indication of which time window of exposure would be expected to be more sensitive for the development of childhood obesity. Our study results, if causal, support evidence for mechanisms during pregnancy, rather than during childhood, to be of most concern. Further studies into the critical time windows for the potential effects of air pollution on childhood obesity are needed to support this. Our study also suggests that prenatal $PM_{2.5}$, but not NO_2, exposure is associated with obesity-related outcomes in children. Previous studies do not provide clear evidence on whether $PM_{2.5}$ may be more likely to be associated with obesity than NO_2. We note that NO_2 and $PM_{2.5}$ share common sources and are mainly driven by combustion-related activities, such as vehicle exhaust and agricultural emissions (Cai et al., 2020). Differences between the pollutants include that NO_2 has been described to be a more local, short-lived trace gas (Matandirotya and Burger, 2021), whereas $PM_{2.5}$ can be transported over regional and long-range transboundary distances (Jun & Gu, 2023). Our study included mainly urban areas and traffic would be the main source of both pollutants in these study areas. The two pollutants showed moderate to strong correlations in the current study, and differences in study areas can partly explain differences in correlations between cohorts.

Possible explanations for the inconsistencies and heterogeneity between cohorts found in the association between air pollution exposure, both during pregnancy and in childhood, and childhood BMI may reflect a number of factors including: (i) different levels of air pollution exposure in the different cohorts; (ii) possible differences in lifestyle behaviors of children from different countries, which may alter their exposure to ambient air pollution and have an effect on weight; and (iii) possible residual confounding by SES and other factors specific to each city environment. Residual confounding may, for example, explain the association between higher air pollution levels and lower BMI we observed in the British BiB cohort. This cohort is characterized by a high level of socioeconomic deprivation and ethnic diversity, with half of the sample

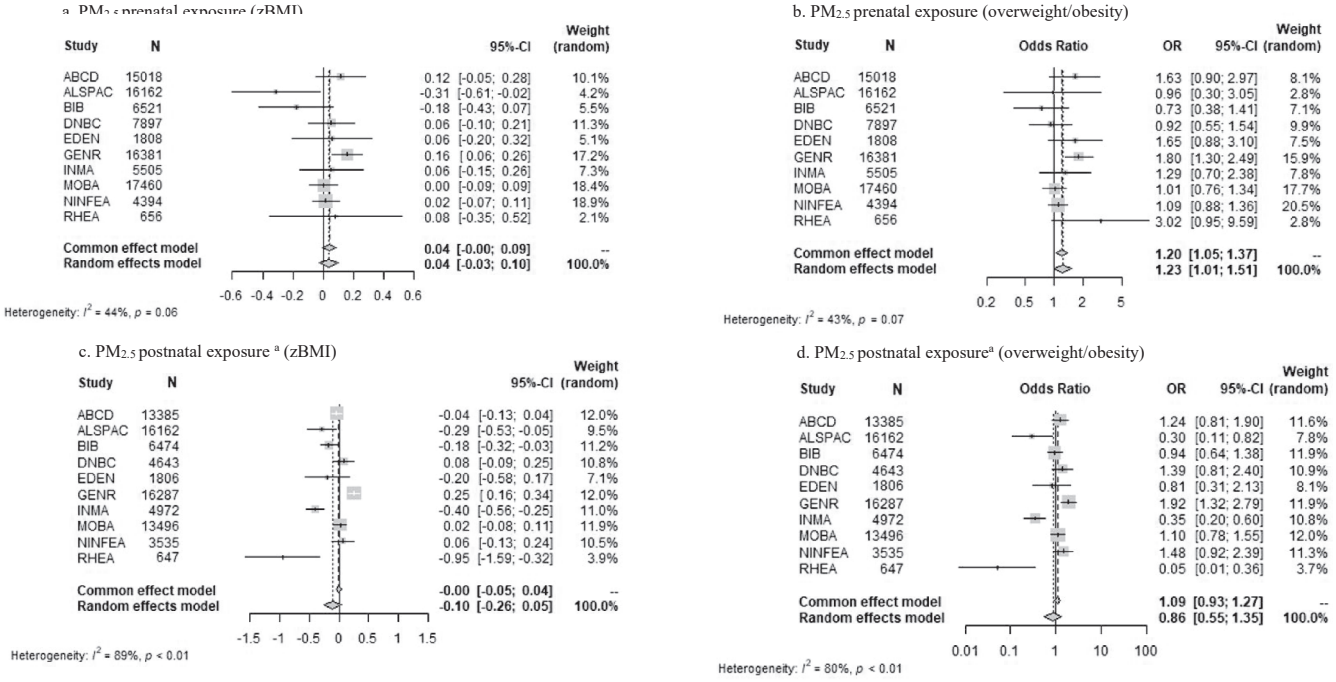


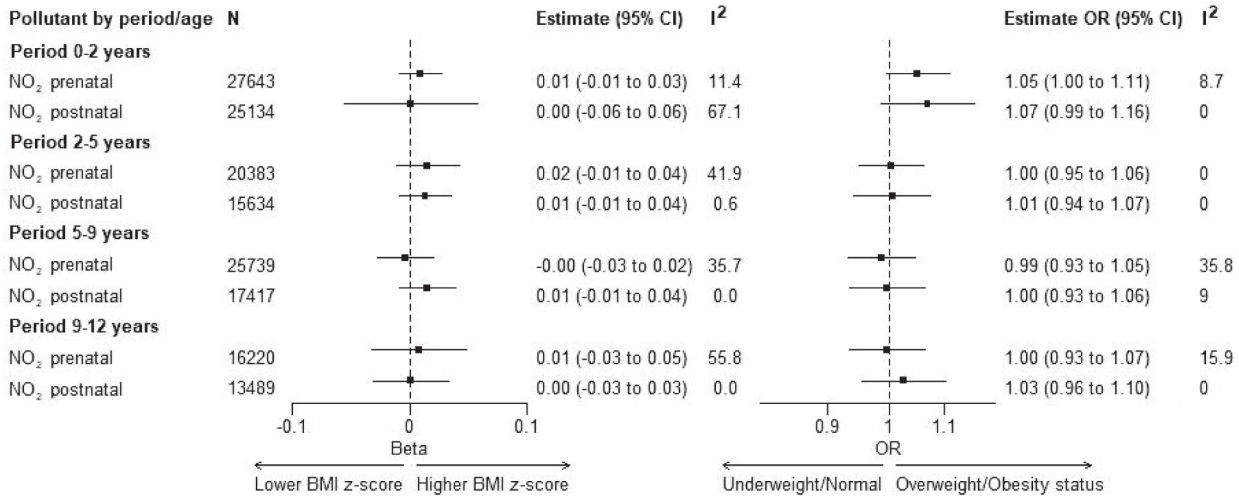
Fig. 3. Forest plot of associations between pre- and postnatal exposure[a] to $PM_{2.5}$ and childhood zBMI and overweight/obesity using two-stage meta-analysis. ABCD: Amsterdam Born Children and their Development; ALSPAC: Avon Longitudinal Study of Parents and Children; BiB: Born in Bradford; DNBC: Danish National Birth Cohort; EDEN: Etude des Déterminants pré et postnatals précoces du développement et de la santé de l'Enfant; Gen R: Generation R Study; INMA: Infancia y Medio Ambiente; MoBa: Norwegian Mother, Father and Child Cohort Study; NINFEA: Nascita e INFanzia: gli Effetti dell'Ambiente; RHEA: Rhea Mother & Child Cohort Study; zBMI: Body mass index z-score; OR: Odds ratio; CI: Confidence intervals. Prenatal exposure models adjusted for sex, child age (in months), individual-level SES (maternal education), pre-pregnancy BMI, smoking during pregnancy, and area-level SES. Postnatal exposure models were further adjusted for prenatal air pollution exposure. Further adjustments: In INMA, NINFEA and EDEN: for city, in Gen R, ABCD and BiB: for ethnicity. I^2: Test for heterogeneity (%). [a] Values are described as weighted average postnatal air pollution levels in the year before zBMI measurement, in the different age periods.

of Pakistani origin, a population that lives in the most polluted and deprived parts of the city, whilst at the same time having a lower BMI (West et al., 2018) and lower susceptibility to air pollution exposure for some outcomes, such as birth weight, compared to their White British peers (Schembari et al., 2015). Our adjustment for ethnicity and SES may not have completely controlled for these effects. Also, we note that the cohorts studied were very socially diverse, and that the relationship between SES and air pollution exposure can vary between cities, with some low SES communities facing higher exposure to air pollution and other environmental hazards and others lower exposures (Hajat et al., 2015; Robinson et al., 2018). In the Spanish cohort INMA, for example, contrasting patterns were seen among cities, with highly educated families being exposed to higher levels of air pollution in Sabadell, and the opposite pattern in Valencia. In Oslo (MoBa), however, high income families were exposed to higher levels of NO_2 and in Bradford (BiB) the opposite was found (i.e., high family education and high family income were exposed to lower levels of air pollutants) (Robinson et al., 2018). To summarize, these are some examples of factors that might explain the high heterogeneity found in the current study and the complex and dynamic relation between air pollution exposure and childhood weight.

The major strength of this study is the large geographical coverage including eight countries and 10 cohorts in Europe, which enabled the study of culturally diverse populations. We were also able to include different urban environments, besides urban characteristics that could be confounders in our analyses. Additionally, we meta-analyzed individual participant data from different cohorts, which may have improved the quality and scope of derived information (Riley et al., 2023), increased our statistical power and enabled more precise estimates compared to single cohort studies. Finally, one important practicality of the current study is the use of federated analyses, which enabled the analysis of data from multiple studies/cohorts without the need of data transfer, decreasing the administrative burden of data transfer agreements, and governance issues of physical data sharing (Cadman et al., 2023).

This study also has limitations that need to be mentioned. First, two different spatial models (ESCAPE and ELAPSE) were used to estimate the exposures. Although they were developed using sampling data from the same period and a common methodology, some differences might occur. Model year development (2009–2010) may be seen as a limitation to represent a scenario several years later than the pregnancy exposure time period and earlier than the childhood exposure periods. However, for most of our cohorts, exposure estimation and health outcome data collection fell within a time period of 10 years either side of the spatial modelling and many predictor variables in LUR models (such as building density, green spaces, distance to roads, etc.) have shown to be stable over time (Beelen et al., 2007; Eeftens et al., 2011; Gulliver et al., 2013; Johnson et al., 2010). Besides, stability of the spatial structure of the ESCAPE and ELAPSE models was confirmed and agreement in spatial variation was reported to be generally high in Europe (de Hoogh et al., 2018). One outlier is the ALSPAC cohort with years of birth in 1991–1992 and a 12-year follow-up included in this study leading to longer term back-extrapolation of the spatial models. Our sensitivity analysis leaving this cohort out showed that main conclusions remain the same. Secondly, missing data were addressed using complete cases, as imputation methods were not available within DataSHIELD at the time of the study. We recognize that missing data on covariates might introduce bias into our findings, and determining the direction of this bias presents a challenge. Aiming to explore if any of the cohorts with more missings (such as cohort BiB and cohort DNBC) could have had an effect on the overall pooled estimate, we performed sensitivity analyses using the "leave-one-out" approach, which showed no meaningful cohort-specific effect on the pooled estimates in the majority of cohorts, except when excluding the DNBC cohort. The observed changes in pooled estimates when excluding DNBC cohort might be explained by

a. NO_2



b. $PM_{2.5}$

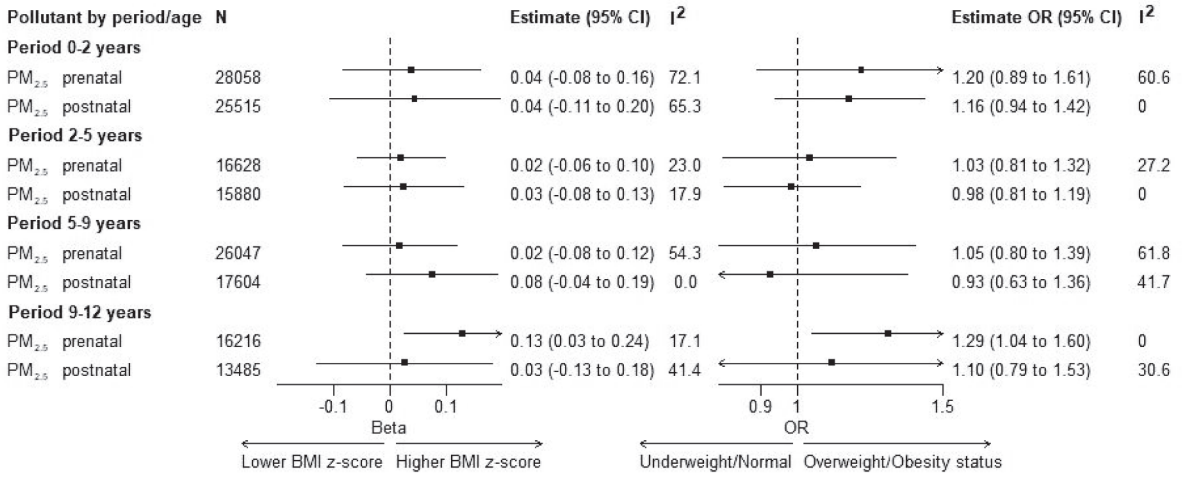


Fig. 4. Pre- and postnatal air pollution exposure[a] and childhood overall zBMI and overweight/obesity using two-stage meta-analysis, stratified by age period. BMI: Body mass index; OR: Odds ratio; CI: Confidence intervals. Prenatal exposure models are adjusted for sex, child age (in months), individual-level SES (maternal education), pre-pregnancy BMI, smoking during pregnancy, and area-level SES. Postnatal exposure models were further adjusted for prenatal air pollution exposure. Further adjustments: In INMA, NINFEA and EDEN: for city, in Gen R, ABCD and BiB: for ethnicity. I^2: Test for heterogeneity (%). [a] Values are described as weighted average postnatal air pollution levels in the year before zBMI measurement, in the different age periods.

the fact that this cohort showed higher proportion of missing values, in variables such as area-level SES (22.7 %) and maternal education (26 %), compared to the other cohorts, and no zBMI data in the age-group 2–5 years. Besides, DNBC is one of the biggest cohorts included in our analyses, so with higher weight in our analysis compared to smaller sample size cohorts. As discussed previously, considerable heterogeneity was found in this study with included cohorts, which might explain the changes in the "leave-one-out" analysis. Third, the use of different cohorts includes harmonization challenges, with variables being harmonized in different ways and in different levels of detail, which may have resulted in between-study heterogeneity that could affect the interpretation of results. More specifically, not all cohorts had data covering all age periods by the end date of data inclusion of this study (e.g., BiB, EDEN and MoBa). Also, not all cohorts had objectively measured anthropometric data (as described in Supplementary Table 2), so we had to rely on parent-reported information for some cohorts (e.g., DNBC, MoBa and NINFEA). In addition, we had to harmonize variables with the least detail, which could have increased the risk of residual confounding leading to greater heterogeneity of estimates. For example, maternal education was harmonized in three categories from more granular detail variables within many of the cohorts. Similarly, maternal smoking during pregnancy was used as a yes/no answer in the current study, however was asked in more detail in several cohorts (which included, for example, the number of cigarettes and the timing of smoking). Fourth, although models were adjusted for both individual-level and area-level SES indicators, we cannot rule out the possibility of bias in our effect estimates due to residual confounding of SES. Also, regarding SES, we used as a proxy of individual-level SES maternal education, which is

commonly used in epidemiological studies (Hajat et al., 2015), since household income was not available in all included cohorts. Fifth, we were not able to take into account other personal and environmental factors that may influence pediatric obesity, such as physical activity and noise levels (An et al., 2018b; González-Muniesa et al., 2017; Jebeile et al., 2022; Wu et al., 2017) due to high missingness and absence of harmonized variables for these factors. We therefore took advantage of availability of family characteristics (e.g., maternal education, BMI, smoking, SES) which influence childhood weight outcomes (Jebeile et al., 2022), along with alternative urban environment features, such as walkability and population density, which may be seen as noise sources due to human activities emission in the neighborhood (Yuan et al., 2019). Lastly, both indoor and outdoor air pollution may have important health effects in the population. Since we did not have data on indoor air pollution, we relied on data of outdoor air pollution only. It is worth noting that a considerable proportion of indoor air pollution comes from outdoors (Amato et al., 2014). Future studies should take into account both indoor and outdoor air pollution exposures, especially in indoor places where children spend a lot of time, such as schools (De Bont et al., 2019), aiming to increase robustness of estimates.

5. Conclusions

In conclusion, this large-scale meta-analysis suggest that prenatal $PM_{2.5}$ exposure may be associated with adverse childhood obesity outcomes, but provides little evidence to support an effect of postnatal air pollution exposure although cohort-specific associations were observed.

CRediT authorship contribution statement

Sarah Warkentin: Writing – review & editing, Writing – original draft. **Serena Fossati:** Writing – review & editing, Methodology, Conceptualization. **Sandra Marquez:** Writing – review & editing, Methodology, Formal analysis. **Anne-Marie Nybo Andersen:** Writing – review & editing, Funding acquisition. **Sandra Andrusaityte:** Writing – review & editing, Funding acquisition. **Demetris Avraam:** Writing – review & editing, Data curation. **Ferran Ballester:** Writing – review & editing, Funding acquisition. **Tim Cadman:** Writing – review & editing. **Maribel Casas:** Writing – review & editing. **Montserrat de Castro:** Writing – review & editing, Data curation. **Leda Chatzi:** Writing – review & editing, Funding acquisition. **Ahmed Elhakeem:** Writing – review & editing, Funding acquisition. **Antonio d'Errico:** Writing – review & editing, Funding acquisition. **Mònica Guxens:** Writing – review & editing, Funding acquisition. **Regina Grazuleviciene:** Writing – review & editing, Funding acquisition. **Jennifer R. Harris:** Writing – review & editing, Funding acquisition. **Carmen Iñiguez Hernandez:** Writing – review & editing, Funding acquisition. **Barbara Heude:** Writing – review & editing, Funding acquisition. **Elena Isaevska:** Writing – review & editing. **Vincent W.V. Jaddoe:** Writing – review & editing, Funding acquisition. **Marianna Karachaliou:** Writing – review & editing. **Aitana Lertxundi:** Writing – review & editing, Funding acquisition. **Johanna Lepeule:** Writing – review & editing. **Rosemary R.C. McEachan:** Writing – review & editing, Funding acquisition. **Johanna L. Thorbjørnsrud Nader:** Writing – review & editing. **Marie Pedersen:** Writing – review & editing, Funding acquisition, Conceptualization. **Susana Santos:** Writing – review & editing. **Mariska Slofstra:** Writing – review & editing. **Euripides G. Stephanou:** Writing – review & editing. **Morris A. Swertz:** Writing – review & editing. **Tanja Vrijkotte:** Writing – review & editing, Funding acquisition. **Tiffany C. Yang:** Writing – review & editing. **Mark Nieuwenhuijsen:** Writing – review & editing, Funding acquisition, Conceptualization. **Martine Vrijheid:** Writing – review & editing, Writing – original draft, Supervision, Methodology, Funding acquisition, Conceptualization.

Funding

The LifeCycle project received funding from the European Union's Horizon 2020 research and innovation programme (Grant Agreement No. 733,206 LifeCycle). This study was funded by the European Union's Horizon 2020 research and innovation programme under grant agreement No 874583 (ATHLETE project).

ABCD: Core funding of the ABCD-study was provided by the Amsterdam University Medical center, Amsterdam, the Public Health Services, Amsterdam, and the Dutch Organization for Health Research and Development (ZonMw).

ALSPAC: Core funding for the Avon Longitudinal Study of Parents and Children (ALSPAC) is provided by the UK Medical Research Council and Wellcome (217065/Z/19/Z) and the University of Bristol. A comprehensive list of grants funding is available on the ALSPAC website (https://www.bristol.ac.uk/alspac/external/documents/grant-acknowledgements.pdf).

BiB: Born in Bradford receives funding from by a joint grant from the UK Medical Research Council (MRC) and UK Economic and Social Science Research Council (ESRC) [MR/N024391/1]; the British Heart Foundation [CS/16/4/32482]; a Wellcome Infrastructure Grant [WT101597MA]; the National Institute for Health Research under its Applied Research Collaboration for Yorkshire and Humber [NIHR200166]; and was supported by UK Research and Innovation for the Healthy Urban Places consortium (grant reference MR/Y022785/1) as part of Population Health Improvement UK (PHI-UK), a national research network which works to transform health and reduce inequalities through change at the population level. The views expressed are those of the author(s), and not necessarily those of the NHS, the NIHR or the Department of Health and Social Care.

DNBC: The Danish National Birth Cohort was established with a significant grant from the Danish National Research Foundation. Additional support was obtained from the Danish Regional Committees, the Pharmacy Foundation, the Egmont Foundation, the March of Dimes Birth Defects Foundation, the Health Foundation and other minor grants. The DNBC Biobank has been supported by the Novo Nordisk Foundation and the Lundbeck Foundation. Follow-up of mothers and children have been supported by the Danish Medical Research Council (SSVF 0646, 271-08-0839/06-066023, O602-01042B, 0602-02738B), the Lundbeck Foundation (195/04, R100-A9193), The Innovation Fund Denmark 0603-00294B (09-067124), the Nordea Foundation (02-2013-2014), Aarhus Ideas (AU R9-A959-13-S804), University of Copenhagen Strategic Grant (IFSV 2012), and the Danish Council for Independent Research (DFF – 4183-00594 and DFF – 4183-00152).

EDEN: The EDEN study was supported by Foundation for medical research (FRM), National Agency for Research (ANR), National Institute for Research in Public health (IRESP: TGIR cohorte santé 2008 program), French Ministry of Health (DGS), French Ministry of Research, INSERM Bone and Joint Diseases National Research (PRO-A) and Human Nutrition National Research Programs, Paris-Sud University, Nestlé, French National Institute for Population Health Surveillance (InVS), French National Institute for Health Education (INPES), the European Union FP7 programmes (FP7/2007-2013, HELIX, ESCAPE, ENRIECO, Medall projects), Diabetes National Research Program (through a collaboration with the French Association of Diabetic Patients (AFD)), French Agency for Environmental Health Safety (now ANSES), Mutuelle Générale de l'Education Nationale a complementary health insurance (MGEN), French national agency for food security, French speaking association for the study of diabetes and metabolism (ALFEDIAM).

Gen R: The general design of the Generation R Study is made possible by financial support from the Erasmus MC, University Medical Center, Rotterdam, Erasmus University Rotterdam, Netherlands Organization for Health Research and Development (ZonMw), Netherlands Organisation for Scientific Research (NWO), Ministry of Health, Welfare and Sport and Ministry of Youth and Families. This project received

funding from the European Union's Horizon 2020 research and innovation programme (LIFECYCLE, grant agreement No 733206, 2016; EUCAN-Connect grant agreement No 824989; ATHLETE, grant agreement No 874583; LongITools, grant agreement No 874739). VJ received funding from a Consolidator Grant from the European Research Council (ERC-2014-CoG-648916).

INMA: This study was funded by grants from the Instituto de Salud Carlos III (Red INMA G03/176) and the Generalitat de Catalunya-CIRIT (1999SGR 00241). INMA-Valencia was funded by Grants from UE (FP7-ENV-2011 cod 282,957 and HEALTH.2010.2.4.5-1), Spain: ISCIII (G03/176; FIS-FEDER: PI09/02647, PI11/01007, PI11/02591, PI11/02038, PI13/1944, PI13/2032, PI14/00891, PI14/01687, and PI16/1288; Miguel Servet-FEDER CP11/00178, CP15/00025, and CPII16/00051), and Generalitat Valenciana: FISABIO (UGP 15-230, UGP-15-244, and UGP-15-249). INMA-Gipuzkoa was funded by grants from the Instituto de Salud Carlos III (FISFIS PI06/0867, FISPS09/0009) 0867, Red INMA G03/176) and the Departamento de Salud del Gobierno Vasco (2005111093 and 2009111069) and the Provincial Government of Guipúzcoa (DFG06/004 and FG08/001). INMA-Sabadell was funded by grants from Instituto de Salud Carlos III (Red INMA G03/176; CB06/02/0041; PI041436; PI081151 incl. FEDER funds; PI12/01890 incl. FEDER funds; CP13/00054 incl. FEDER funds; PI15/00118 incl. FEDER funds; CP16/00128 incl. FEDER funds; PI16/00118 incl. FEDER funds; PI16/00261 incl. FEDER funds; PI17/01340 incl. FEDER funds; PI18/00547 incl. FEDER funds), CIBERESP, Generalitat de Catalunya-CIRIT 1999SGR 00241, Generalitat de Catalunya-AGAUR (2009 SGR 501, 2014 SGR 822), Fundació La marató de TV3 (090430), Spanish Ministry of Economy and Competitiveness (SAF2012-32991 incl. FEDER funds), Agence Nationale de Securité Sanitaire de l'Alimentation de l'Environnement et du Travail (1262C0010; EST-2016 RF-21), EU Commission (261357, 308333, 603,794 and 634453). Mònica Guxens was funded by a Miguel Servet II fellowship (CPII18/00018) awarded by the Spanish Institute of Health Carlos III. We acknowledge support from the grant CEX2023-0001290-S funded by MCIN/AEI/https://doi.org/10.13039/501100011033, and support from the Generalitat de Catalunya through the CERCA Program.

MoBa: The Norwegian Mother, Father and Child Cohort Study is supported by the Norwegian Ministry of Health and Care Services and the Ministry of Education and Research. We are grateful to all the participating families in Norway who take part in this on-going cohort study. This work was partly supported by the Research Council of Norway through its Centres of Excellence funding scheme, project number 262700. This study includes data from the Norwegian Mother, Father and Child Cohort Study (MoBa) conducted by the Norwegian Institute of Public Health.

NINFEA: The NINFEA cohort was initially funded by the Compagnia SanPaolo Foundation and the Piedmont Region. It received funding from European projects: CHICOS (FP7 grant number HEALTH-FP7-2009-241604, LifeCycle (H2020 grant number 733206), ATHLETE (H2020 grant number 874583).

RHEA: The "Rhea" project was financially supported by European projects (EU FP6-2003-Food-3-NewGeneris, EU FP6. STREP Hiwate, EU FP7 NV.2007.1.2.2.2. Project No 211,250 Escape, EU FP7-2008-ENV-1.2.1.4 Envirogenomarkers, EU FP7-HEALTH-2009- single stage CHICOS, EU FP7 ENV.2008.1.2.1.6. Proposal No 226,285 ENRIECO, EU-FP7- HEALTH-2012 Proposal No 308,333 HELIX) and the Greek Ministry of Health (Program of Prevention of obesity and neurodevelopmental disorders in preschool children, in Heraklion district, Crete, Greece: 2011-2014; "Rhea Plus": Primary Prevention Program of Environmental Risk Factors for Reproductive Health, and Child Health: 2012-15).

SW acknowledges receiving the funding by the Generalitat de Catalunya (Agency for Management of University and Research Grants) with a Beatriu de Pinós post-doctoral fellowship (Ref: 2021 BP 00058).

SS is supported by the European Unioń s Horizon Europe Research and Innovation Programme under the Marie Sklodowska-Curie Postdoctoral Fellowship Grant Agreement No. 101,109,136 (URBANE).

The funders had no role in the design and conduct of the study; management, analysis, and interpretation of the data; preparation, review, or approval of the manuscript; and decision to submit the manuscript for publication.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgments

Data harmonisations received funding from the European Union's Horizon 2020 research and innovation programme (Grant Agreement No. 733206 LifeCycle project and No. 874739 LongITools project). We are grateful to all the participating children, parents, practitioners and researchers in the cohorts who took part in this study.

ABCD: We are grateful to all participating hospitals, obstetric clinics, and general practitioners for their assistance in implementing the ABCD study and thank all of the women who participated for their cooperation.

ALSPAC: We are extremely grateful to all of the families who took part in ALSPAC, the midwives for their help in recruiting them, and the whole ALSPAC team, which includes interviewers, computer and laboratory technicians, clerical workers, research scientists, volunteers, managers, receptionists and nurses.

BiB: BiB is only possible because of the enthusiasm and commitment of the children and parents in BiB. We are grateful to all the participants, health professionals, schools and researchers who have made BiB happen.

DNBC: The authors would like to thank the participants, the previous Principal Investigators of DNBC Prof. Jørn Olsen and Prof Anne-Marie Nybo Andersen, the scientific managerial team, and DNBC secretariat for being, establishing, developing and consolidating the Danish National Birth Cohort.

EDEN: The authors thank all the participants of the EDEN study and the EDEN mother-child cohort study group, whose members are I. Annesi-Maesano, J.Y. Bernard, M.A. Charles, P. Dargent-Molina, B. de Lauzon-Guillain, P. Ducimetière, M. de Agostini, B. Foliguet, A. Forhan, X. Fritel, A. Germa, V. Goua, R. Hankard, B. Heude, M. Kaminski, B. Larroquey, N. Lelong, J. Lepeule, G. Magnin, L. Marchand, C. Nabet, F Pierre, R. Slama, M.J. Saurel-Cubizolles, M. Schweitzer, and O. Thiebaugeorges.

Gen R: The authors gratefully acknowledge the contribution of participants, research collaborators, general practitioners, hospitals, midwives, and pharmacies in Rotterdam.

INMA Gipuzkoa, Sabadell and Valencia: The authors would particularly like to thank all the participants for their generous collaboration. A full roster of the INMA Project Investigators can be found at https://www.proyectoinma.org/en/inma-project/inma-project-researchers/.

MoBa: We are grateful to all the participating families in Norway who take part in this on-going cohort study.

NINFEA: The authors thank all families participating in the NINFEA cohort.

RHEA: We are grateful to all the participating families who took part in this cohort study.

Appendix A. Supplementary material

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envint.2025.109527.

Data availability

The data that has been used is confidential.

References

Alderete, T.L., Habre, R., Toledo-Corral, C.M., Berhane, K., Chen, Z., Lurmann, F.W., Weigensberg, M.J., Goran, M.I., Gilliland, F.D., 2017. Longitudinal associations between ambient air pollution with insulin sensitivity, β-cell function, and adiposity in los angeles latino children. *Diabetes* 66 (7), 1789–1796. https://doi.org/10.2337/db16-1416.

Alemany, S., Crous-Bou, M., Vilor-Tejedor, N., Milà-Alomà, M., Suárez-Calvet, M., Salvadó, G., Cirach, M., Arenaza-Urquijo, E.M., Sanchez-Benavides, G., Grau-Rivera, O., Minguillon, C., Fauria, K., Kollmorgen, G., Domingo Gispert, J., Gascón, M., Nieuwenhuijsen, M., Zetterberg, H., Blennow, K., Sunyer, J., Luis Molinuevo, J., 2021. Associations between air pollution and biomarkers of Alzheimer's disease in cognitively unimpaired individuals. *Environ. Int.* 157, 106864. https://doi.org/10.1016/j.envint.2021.106864.

Amato, F., Rivas, I., Viana, M., Moreno, T., Bouso, L., Reche, C., Àlvarez-Pedrerol, M., Alastuey, A., Sunyer, J., Querol, X., 2014. Sources of indoor and outdoor PM2.5 concentrations in primary schools. *Sci. Total Environ.* 490, 757–765. https://doi.org/10.1016/j.scitotenv.2014.05.051.

An, R., Ji, M., Yan, H., Guan, C., 2018a. Impact of ambient air pollution on obesity: a systematic review. *Int. J. Obes. (Lond)* 42 (6), 1112–1126. https://doi.org/10.1038/s41366-018-0089-y.

An, R., Zhang, S., Ji, M., Guan, C., 2018b. Impact of ambient air pollution on physical activity among adults: A systematic review and meta-analysis. *Perspect. Public Health* 138 (2), 111–121. https://doi.org/10.1177/1757913917726567.

Ballvé, A., Pizarro, J., Maisterra, O., Riba-Llena, I., Pujadas, F., Jiménez-Balado, J., Palasi, A., Cirach, M., Turner, M.C., Sunyer, J., Delgado, P., 2024. Ambient air pollution, covert cerebrovascular disease and cognition: results from the ISSYS study. *Eur. J. Neurol.* 31 (10), e16404. https://doi.org/10.1111/ene.16404.

Beelen, R., Hoek, G., Fischer, P., Brandt, P.A.V.D., Brunekreef, B., 2007. Estimated long-term outdoor air pollution concentrations in a cohort study. *Atmos. Environ.* 41 (7), 1343–1358. https://doi.org/10.1016/j.atmosenv.2006.10.020.

Bekkar, B., Pacheco, S., Basu, R., DeNicola, N., 2020. Association of air pollution and heat exposure with preterm birth, low birth weight, and stillbirth in the US. *JAMA Netw. Open* 3 (6), e208243. https://doi.org/10.1001/jamanetworkopen.2020.8243.

Binter, A.-C., Bernard, J.Y., Mon-Williams, M., Andiarena, A., González-Safont, L., Vafeiadi, M., Lepeule, J., Soler-Blasco, R., Alonso, L., Kampouri, M., Mceachan, R., Santa-Marina, L., Wright, J., Chatzi, L., Sunyer, J., Philippat, C., Nieuwenhuijsen, M., Vrijheid, M., Guxens, M., 2022. Urban environment and cognitive and motor function in children from four European birth cohorts. *Environ. Int.* 158, 106933. https://doi.org/10.1016/j.envint.2021.106933.

Bloemsma, L.D., Wijga, A.H., Klompmaker, J.O., Janssen, N.A.H., Smit, H.A., Koppelman, G.H., Brunekreef, B., Lebret, E., Hoek, G., Gehring, U., 2019. The associations of air pollution, traffic noise and green space with overweight throughout childhood: the PIAMA birth cohort study. *Environ. Res.* 169, 348–356. https://doi.org/10.1016/j.envres.2018.11.026.

Borenstein, M., Hedges, L.V., Higgins, J.P.T., Rothstein, H.R., 2010. A basic introduction to fixed-effect and random-effects models for meta-analysis. *Res. Synth. Methods* 1 (2), 97–111. https://doi.org/10.1002/jrsm.12.

Cadman, T., Elhakeem, A., Vinther, J.L., Avraam, D., Carrasco, P., Calas, L., Cardo, M., Charles, M.-A., Corpeleijn, E., Crozier, S., de Castro, M., Estarlich, M., Fernandes, A., Fossatti, S., Gruszfeld, D., Gurlich, K., Grote, V., Haakma, S., Harris, J.R., Lawlor, D., 2023. Associations of Maternal educational level, proximity to greenspace during pregnancy, and gestational diabetes with body mass index from infancy to early adulthood: a proof-of-concept federated analysis in 18 birth cohorts. *Am. J. Epidemiol.* https://doi.org/10.1093/aje/kwad206.

Cai, J., Ge, Y., Li, H., Yang, C., Liu, C., Meng, X., Wang, W., Niu, C., Kan, L., Schikowski, T., Yan, B., Chillrud, S.N., Kan, H., Jin, L., 2020. Application of land use regression to assess exposure and identify potential sources in $PM_{2.5}$, BC, NO_2 concentrations. *Atmos. Environ.* 223, 117267. https://doi.org/10.1016/j.atmosenv.2020.117267.

Castells-Quintana, D., Dienesch, E., Krause, M., 2021. Air pollution in an urban world: a global view on density, cities and emissions. *Ecol. Econ.* 189, 107153. https://doi.org/10.1016/j.ecolecon.2021.107153.

Chatzi, L., Leventakou, V., Vafeiadi, M., Koutra, K., Roumeliotaki, T., Chalkiadaki, G., Karachaliou, M., Daraki, V., Kyriklaki, A., Kampouri, M., Fthenou, E., Sarri, K., Vassilaki, M., Fasoulaki, M., Bitsios, P., Koutis, A., Stephanou, E.G., Kogevinas, M., 2017. Cohort Profile: the mother-child cohort in Crete, Greece (Rhea Study). *Int. J. Epidemiol.* 46 (5), 1392–1393k. https://doi.org/10.1093/ije/dyx084.

Cruells, A., Cabrera-Rubio, R., Bustamante, M., Pelegrí, D., Cirach, M., Jimenez-Arenas, P., Samarra, A., Martínez-Costa, C., Collado, M.C., Gascon, M., 2024. The influence of pre- and postnatal exposure to air pollution and green spaces on infant's gut microbiota: results from the MAMI birth cohort study. *Environ. Res.* 257, 119283. https://doi.org/10.1016/j.envres.2024.119283.

Darbre, P., 2018. Overview of air pollution and endocrine disorders. Int. J. General Med. 11 (May 2018), 191–207. https://doi.org/10.2147/IJGM.S102230.

De Bont, J., Casas, M., Barrera-Gómez, J., Cirach, M., Rivas, I., Valvi, D., Álvarez, M., Dadvand, P., Sunyer, J., Vrijheid, M., 2019. Ambient air pollution and overweight and obesity in school-aged children in Barcelona, Spain. Environ. Int. 125 (September 2018), 58–64. https://doi.org/10.1016/j.envint.2019.01.048.

De Bont, J., Hughes, R., Tilling, K., Díaz, Y., De Castro, M., Cirach, M., Fossati, S., Nieuwenhuijsen, M., Duarte-Salles, T., Vrijheid, M., 2020. Early life exposure to air pollution, green spaces and built environment, and body mass index growth trajectories during the first 5 years of life: a large longitudinal study. *Environ. Pollut.* 266, 115266. https://doi.org/10.1016/j.envpol.2020.115266.

De Hoog, M.L., Van Eijsden, M., Stronks, K., Gemke, R.J., Vrijkotte, T.G., 2011. Overweight at age two years in a multi-ethnic cohort (ABCD study): the role of prenatal factors, birth outcomes and postnatal factors. *BMC Public Health* 11 (1), 611. https://doi.org/10.1186/1471-2458-11-611.

de Hoogh, K., Chen, J., Gulliver, J., Hoffmann, B., Hertel, O., Ketzel, M., Bauwelinck, M., van Donkelaar, A., Hvidtfeldt, U.A., Katsouyanni, K., Klompmaker, J., Martin, R.V., Samoli, E., Schwartz, P.E., Stafoggia, M., Bellander, T., Strak, M., Wolf, K., Vienneau, D., Brunekreef, B., Hoek, G., 2018. Spatial $PM_{2.5}$, NO_2, O_3 and BC models for Western Europe – Evaluation of spatiotemporal stability. *Environ. Int.* 120, 81–92. https://doi.org/10.1016/j.envint.2018.07.036.

de Onis, M., 2007. Development of a WHO growth reference for school-aged children and adolescents. *Bull. World Health Organ.* 85 (9), 660–667. https://doi.org/10.2471/BLT.07.043497.

Eeftens, M., Beelen, R., de Hoogh, K., Bellander, T., Cesaroni, G., Cirach, M., Declercq, C., Dėdelė, A., Dons, E., de Nazelle, A., Dimakopoulou, K., Eriksen, K., Falq, G., Fischer, P., Galassi, C., Gražulevičienė, R., Heinrich, J., Hoffmann, B., Jerrett, M., Hoek, G., 2012. Development of land use regression models for $PM_{2.5}$, $PM_{2.5}$ absorbance, PM_{10} and PM_{coarse} in 20 European study areas; results of the ESCAPE project. *Environ. Sci. Tech.* 46 (20), 11195–11205. https://doi.org/10.1021/es301948k.

Eeftens, M., Beelen, R., Fischer, P., Brunekreef, B., Meliefste, K., Hoek, G., 2011. Stability of measured and modelled spatial contrasts in NO_2 over time. *Occup. Environ. Med.* 68 (10), 765–770. https://doi.org/10.1136/oem.2010.061135.

Fioravanti, S., Cesaroni, G., Badaloni, C., Michelozzi, P., Forastiere, F., Porta, D., 2018. Traffic-related air pollution and childhood obesity in an Italian birth cohort. *Environ. Res.* 160 (January 2018), 479–486. https://doi.org/10.1016/j.envres.2017.10.003.

Fleisch, A.F., Aris, I.M., Rifas-Shiman, S.L., Coull, B.A., Luttmann-Gibson, H., Koutrakis, P., Schwartz, J.D., Kloog, I., Gold, D.R., Oken, E., 2019. Prenatal exposure to traffic pollution and childhood body mass index trajectory. *Front. Endocrinol.* 9. https://doi.org/10.3389/fendo.2018.00771.

Fleisch, A.F., Luttmann-Gibson, H., Perng, W., Rifas-Shiman, S.L., Coull, B.A., Kloog, I., Koutrakis, P., Schwartz, J.D., Zanobetti, A., Mantzoros, C.S., Gillman, M.W., Gold, D. R., Oken, E., 2017. Prenatal and early life exposure to traffic pollution and cardiometabolic health in childhood. *Pediatr. Obes.* 12 (1), 48–57. https://doi.org/10.1111/ijpo.12106.

Fleisch, A.F., Rifas-Shiman, S.L., Koutrakis, P., Schwartz, J.D., Kloog, I., Melly, S., Coull, B.A., Zanobetti, A., Gillman, M.W., Gold, D.R., Oken, E., 2015. Prenatal exposure to traffic pollution: associations with reduced fetal growth and rapid infant weight gain. *Epidemiology* 26 (1), 43–50. https://doi.org/10.1097/EDE.0000000000000203.

Fossati, S., Valvi, D., Martinez, D., Cirach, M., Estarlich, M., Fernández-Somoano, A., Guxens, M., Iñiguez, C., Irizar, A., Lertxundi, A., Nieuwenhuijsen, M., Tamayo, I., Vioque, J., Tardón, A., Sunyer, J., Vrijheid, M., 2020. Prenatal air pollution exposure and growth and cardio-metabolic risk in preschoolers. *Environ. Int.* 138, 105619. https://doi.org/10.1016/j.envint.2020.105619.

Frank, L.D., Sallis, J.F., Conway, T.L., Chapman, J.E., Saelens, B.E., Bachman, W., 2006. Many pathways from land use to health: associations between neighborhood walkability and active transportation, body mass index, and air quality. *J. Am. Plann. Assoc.* 72 (1), 75–87. https://doi.org/10.1080/01944360608976725.

Fraser, A., Macdonald-Wallis, C., Tilling, K., Boyd, A., Golding, J., Davey Smith, G., Henderson, J., Macleod, J., Molloy, L., Ness, A., Ring, S., Nelson, S.M., Lawlor, D.A., 2013. Cohort profile: the avon longitudinal study of parents and children: ALSPAC mothers cohort. *Int. J. Epidemiol.* 42 (1), 97–110. https://doi.org/10.1093/ije/dys066.

Frondelius, K., Oudin, A., Malmqvist, E., 2018. Traffic-related air pollution and child BMI—A study of prenatal exposure to nitrogen oxides and body mass index in children at the age of four years in Malmö, Sweden. *Int. J. Environ. Res. Public Health* 15 (10), 2294. https://doi.org/10.3390/ijerph15102294.

Gaye, A., Marcon, Y., Isaeva, J., LaFlamme, P., Turner, A., Jones, E.M., Minion, J., Boyd, A.W., Newby, C.J., Nuotio, M.-L., Wilson, R., Butters, O., Murtagh, B., Demir, I., Doiron, D., Giepmans, L., Wallace, S.E., Budin-Ljøsne, I., Oliver Schmidt, C., Burton, P.R., 2014. DataSHIELD: taking the analysis to the data, not the data to the analysis. *Int. J. Epidemiol.* 43 (6), 1929–1944. https://doi.org/10.1093/ije/dyu188.

Global Human Settlement Layer, 2015. https://ghsl.jrc.ec.europa.eu/.

González-Muniesa, P., Mártinez-González, M.-A., Hu, F.B., Després, J.-P., Matsuzawa, Y., Loos, R.J.F., Moreno, L.A., Bray, G.A., Martinez, J.A., 2017. Obesity. *Nat. Rev. Dis. Primers* 3 (1), 17034. https://doi.org/10.1038/nrdp.2017.34.

Gulliver, J., De Hoogh, K., Hansell, A., Vienneau, D., 2013. Development and back-extrapolation of NO_2 land use regression models for historic exposure assessment in Great Britain. *Environ. Sci. Tech.* 47 (14), 7804–7811. https://doi.org/10.1021/es4008849.

Guxens, M., Ballester, F., Espada, M., Fernández, M.F., Grimalt, J.O., Ibarluzea, J., Olea, N., Rebagliato, M., Tardón, A., Torrent, M., Vioque, J., Vrijheid, M., Sunyer, J., 2012. Cohort Profile: The INMA—INfancia y Medio Ambiente—(Environment and Childhood) Project. *Int. J. Epidemiol.* 41 (4), 930–940. https://doi.org/10.1093/ije/dyr054.

Guxens, M., Garcia-Esteban, R., Giorgis-Allemand, L., Forns, J., Badaloni, C., Ballester, F., Beelen, R., Cesaroni, G., Chatzi, L., De Agostini, M., De Nazelle, A., Eeftens, M., Fernandez, M.F., Fernández-Somoano, A., Forastiere, F., Gehring, U., Ghassabian, A., Heude, B., Jaddoe, V.W.V., Sunyer, J., 2014. Air pollution during pregnancy and

childhood cognitive and psychomotor development: six european birth cohorts. *Epidemiology* 25 (5), 636–647. https://doi.org/10.1097/EDE.0000000000000133.
Hajat, A., Hsia, C., O'Neill, M.S., 2015. Socioeconomic disparities and air pollution exposure: a global review. *Curr. Environ. Health Rep.* 2 (4), 440–450. https://doi.org/10.1007/s40572-015-0069-5.
Health Effects Institute, 2022. Systematic Review and Meta-analysis of Selected Health Effects of Long-Term Exposure to Traffic-Related Air Pollution: Traffic Pollution and Your Health (HEI Special Report 23). https://www.healtheffects.org/publication/systematic-review-and-meta-analysis-selected-health-effects-long-term-exposure-traffic.
Heude, B., Forhan, A., Slama, R., Douhaud, L., Bedel, S., Saurel-Cubizolles, M.-J., Hankard, R., Thiebaugeorges, O., De Agostini, M., Annesi-Maesano, I., Kaminski, M., Charles, M.-A., 2016. Cohort Profile: the EDEN mother-child cohort on the prenatal and early postnatal determinants of child health and development. *Int. J. Epidemiol.* 45 (2), 353–363. https://doi.org/10.1093/ije/dyv151.
Huang, C., Li, C., Zhao, F., Zhu, J., Wang, S., Sun, G., 2022. The association between childhood exposure to ambient air pollution and obesity: a systematic review and meta-analysis. *Int. J. Environ. Res. Public Health* 19 (8), 4491. https://doi.org/10.3390/ijerph19084491.
Huang, J.V., Leung, G.M., Schooling, C.M., 2019. The association of air pollution with body mass index: evidence from Hong Kong's "Children of 1997" birth cohort. *Int. J. Obes. (lond)* 43 (1), 62–72. https://doi.org/10.1038/s41366-018-0070-9.
Huang, Lu., Du, J., Zhang, L., Wei, J., Huang, Q., Wu, S., Zhou, X., Ren, L., 2023. Effects of exposure to $PM_{2.5}$ during pregnancy on the multigenerational reproductive outcomes of male mouse offspring and the role of Sertoli cells. *Environ. Sci. Pollut. Res.* 30 (47), 103823–103835. https://doi.org/10.1007/s11356-023-29751-8.
Hung, T.-H., Chen, P.-H., Tung, T.-H., Hsu, J., Hsu, T.-Y., Wan, G.-H., 2022. Risks of preterm birth and low birth weight and maternal exposure to NO_2/$PM_{2.5}$ acquired by dichotomous evaluation: a systematic review and meta-analysis. *Environ. Sci. Pollut. Res.* 30 (4), 9331–9349. https://doi.org/10.1007/s11356-022-24520-5.
Jaddoe, V.W.V., Felix, J.F., Andersen, A.-M.-N., Charles, M.-A., Chatzi, L., Corpeleijn, E., Donner, N., Elhakeem, A., Eriksson, J.G., Foong, R., Grote, V., Haakma, S., Hanson, M., Harris, J.R., Heude, B., Huang, R.-C., Inskip, H., Järvelin, M.-R., Koletzko, B., Rauschert, S., 2020. The LifeCycle Project-EU child cohort network: a federated analysis infrastructure and harmonized data of more than 250,000 children and parents. *Eur. J. Epidemiol.* 35 (7), 709–724. https://doi.org/10.1007/s10654-020-00662-z.
Jebeile, H., Kelly, A.S., O'Malley, G., Baur, L.A., 2022. Obesity in children and adolescents: epidemiology, causes, assessment, and management. *Lancet Diabetes Endocrinol.* 10 (5), 351–365. https://doi.org/10.1016/S2213-8587(22)00047-X.
Jerrett, M., McConnell, R., Wolch, J., Chang, R., Lam, C., Dunton, G., Gilliland, F., Lurmann, F., Islam, T., Berhane, K., 2014. Traffic-related air pollution and obesity formation in children: a longitudinal, multilevel analysis. *Environ. Health: A Global Access Sci. Source* 13 (1), 1–9. https://doi.org/10.1186/1476-069X-13-49.
Ji, N., Johnson, M., Eckel, S.P., Gauderman, W.J., Chavez, T.A., Berhane, K., Faham, D., Lurmann, F., Pavlovic, N.R., Grubbs, B.H., Lerner, D., Habre, R., Farzan, S.F., Bastain, T.M., Breton, C.V., 2023. Prenatal ambient air pollution exposure and child weight trajectories from the 3rd trimester of pregnancy to 2 years of age: a cohort study. *BMC Med.* 21 (1), 341. https://doi.org/10.1186/s12916-023-03050-y.
Johnson, M., Isakov, V., Touma, J.S., Mukerjee, S., Özkaynak, H., 2010. Evaluation of land-use regression models used to predict air quality concentrations in an urban area. *Atmos. Environ.* 44 (30), 3660–3668. https://doi.org/10.1016/j.atmosenv.2010.06.041.
Jun, M.-J., Gu, Y., 2023. Effects of transboundary PM2.5 transported from China on the regional PM2.5 concentrations in South Korea: a spatial panel-data analysis. *PLoS One* 18 (4), e0281988. https://doi.org/10.1371/journal.pone.0281988.
Kogevinas, M., Karachaliou, M., Espinosa, A., Aguilar, R., Castaño-Vinyals, G., Garcia-Aymerich, J., Carreras, A., Cortés, B., Pleguezuelos, V., Papantoniou, K., Rubio, R., Jiménez, A., Vidal, M., Serra, P., Parras, D., Santamaría, P., Izquierdo, L., Cirach, M., Nieuwenhuijsen, M., Tonne, C., 2023. Long-term exposure to air pollution and COVID-19 vaccine antibody response in a general population cohort (COVICAT Study, Catalonia). *Environ. Health Perspect.* 131 (4), 047001. https://doi.org/10.1289/EHP11989.
Kooijman, M.N., Kruithof, C.J., van Duijn, C.M., Duijts, L., Franco, O.H., van IJzendoorn, M.H., de Jongste, J.C., Klaver, C.C.W., van der Lugt, A., Mackenbach, J. P., Moll, H.A., Peeters, R.P., Raat, H., Rings, E.H.H.M., Rivadeneira, F., van der Schroeff, M.P., Steegers, E.A.P., Tiemeier, H., Uitterlinden, A.G., Jaddoe, V.W.V., 2016. The generation R study: design and cohort update 2017. *Eur. J. Epidemiol.* 31 (12), 1243–1264. https://doi.org/10.1007/s10654-016-0224-9.
Laurent, O., Wu, J., Li, L., Chung, J., Bartell, S., 2013. Investigating the association between birth weight and complementary air pollution metrics: a cohort study. *Environ. Health* 12 (1), 18. https://doi.org/10.1186/1476-069X-12-18.
Magnus, P., Birke, C., Vejrup, K., Haugan, A., Alsaker, E., Daltveit, A.K., Handal, M., Haugen, M., Høiseth, G., Knudsen, G.P., Paltiel, L., Schreuder, P., Tambs, K., Vold, L., Stoltenberg, C., 2016. Cohort profile update: the Norwegian mother and child cohort study (MoBa). *Int. J. Epidemiol.* 45 (2), 382–388. https://doi.org/10.1093/ije/dyw029.
Malacarne, D., Handakas, E., Robinson, O., Pineda, E., Saez, M., Chatzi, L., Fecht, D., 2022. The built environment as determinant of childhood obesity: a systematic literature review. *Obes. Rev.* 23 (S1), 1–11. https://doi.org/10.1111/obr.13385.
Matandirotya, N.R., Burger, R.P., 2021. Spatiotemporal variability of tropospheric NO_2 over four megacities in Southern Africa: implications for transboundary regional air pollution. *Environ. Challenges* 5, 100271. https://doi.org/10.1016/j.envc.2021.100271.
McConnell, R., Gilliland, F.D., Goran, M., Allayee, H., Hricko, A., Mittelman, S., 2016. Does near-roadway air pollution contribute to childhood obesity? *Pediatr. Obes.* 11 (1), 1–3. https://doi.org/10.1111/ijpo.12016.
McEachan, R.R.C., Santorelli, G., Watmuff, A., Mason, D., Barber, S.E., Bingham, D.D., Bird, P.K., Lennon, L., Lewer, D., Mon-Williams, M., Shire, K.A., Waiblinger, D., West, J., Yang, T.C., Lawlor, D.A., Pickett, K.E., Wright, J., 2024. Cohort profile update: born in Bradford. *Int. J. Epidemiol.* 53 (2), dyae037. https://doi.org/10.1093/ije/dyae037.
Ng, C., Malig, B., Hasheminassab, S., Sioutas, C., Basu, R., Ebisu, K., 2017. Source apportionment of fine particulate matter and risk of term low birth weight in California: Exploring modification by region and maternal characteristics. *Sci. Total Environ.* 605–606, 647–654. https://doi.org/10.1016/j.scitotenv.2017.06.053.
Nieuwenhuijsen, M.J., 2021. New urban models for more sustainable, liveable and healthier cities post covid19; reducing air pollution, noise and heat island effects and increasing green space and physical activity. *Environ. Int.* 157, 106850. https://doi.org/10.1016/j.envint.2021.106850.
Nieuwenhuijsen, M.J., Agier, L., Basagaña, X., Urquiza, J., Tamayo-Uria, I., Giorgis-Allemand, L., Robinson, O., Siroux, V., Maitre, L., de Castro, M., Valentin, A., Donaire, D., Dadvand, P., Aasvang, G.M., Krog, N.H., Schwarze, P.E., Chatzi, L., Grazuleviciene, R., Andrusaityte, S., Dedele, A., McEachan, R., Wright, J., West, J., Ibarluzea, J., Ballester, F., Vrijheid, M., Slama, R., 2019. Influence of the urban exposome on birth weight. *Environ. Health Perspect.* 127 (4). https://doi.org/10.1289/EHP3971.
Olsen, J., Melbye, M., Olsen, S.F., Sørensen, T.I.A., Aaby, P., Nybo Andersen, A.-M., Taxbøl, D., Hansen, K.D., Juhl, M., Schow, T.B., Sørensen, H.T., Andresen, J., Mortensen, E.L., Wind Olesen, A., Søndergaard, C., 2001. The Danish National Birth Cohort—Its background, structure and aim. *Scand. J. Public Health* 29 (4), 300–307. https://doi.org/10.1177/14034948010290040201.
Parasin, N., Amnuaylojaroen, T., Saokaew, S., 2021. Effect of air pollution on obesity in children: a systematic review and meta-analysis. *Children* 8 (5), 327. https://doi.org/10.3390/children8050327.
Pedersen, M., Giorgis-Allemand, L., Bernard, C., Aguilera, I., Andersen, A.-M.-N., Ballester, F., Beelen, R.M.J., Chatzi, L., Cirach, M., Danileviciute, A., Dedele, A., Eijsden, M.V., Estarlich, M., Fernández-Somoano, A., Fernández, M.F., Forastiere, F., Gehring, U., Grazuleviciene, R., Gruzieva, O., Slama, R., 2013. Ambient air pollution and low birthweight: a European cohort study (ESCAPE). *Lancet Respir. Med.* 1 (9), 695–704. https://doi.org/10.1016/S2213-2600(13)70192-9.
Phelps, N.H., Singleton, R.K., Zhou, B., Heap, R.A., Mishra, A., Bennett, J.E., Paciorek, C. J., Lhoste, V.P., Carrillo-Larco, R.M., Stevens, G.A., Rodriguez-Martinez, A., Bixby, H., Bentham, J., Di Cesare, M., Danaei, G., Rayner, A.W., Barradas-Pires, A., Cowan, M.J., Savin, S., Ezzati, M., 2024. Worldwide trends in underweight and obesity from 1990 to 2022: A pooled analysis of 3663 population-representative studies with 222 million children, adolescents, and adults. *Lancet*, S0140673623027502. https://doi.org/10.1016/S0140-6736(23)02750-2.
Pinot de Moira, A., Haakma, S., Strandberg-Larsen, K., van Enckevort, E., Kooijman, M., Cadman, T., Cardol, M., Corpeleijn, E., Crozier, S., Duijts, L., Elhakeem, A., Eriksson, J.G., Felix, J.F., Fernández-Barrés, S., Foong, R.E., Forhan, A., Grote, V., Guerlich, K., Heude, B., Nybo Andersen, A.-M., 2021. The EU Child Cohort Network's core data: establishing a set of findable, accessible, interoperable and re-usable (FAIR) variables. *Eur. J. Epidemiol.* 36 (5), 565–580. https://doi.org/10.1007/s10654-021-00733-9.
Richiardi, L., Baussano, I., Vizzini, L., Douwes, J., Pearce, N., Merletti, F., 2007. Feasibility of recruiting a birth cohort through the Internet: the experience of the NINFEA cohort. *Eur. J. Epidemiol.* 22 (12), 831–837. https://doi.org/10.1007/s10654-007-9194-2.
Riley, R.D., Dias, S., Donegan, S., Tierney, J.F., Stewart, L.A., Efthimiou, O., Phillippo, D. M., 2023. Using individual participant data to improve network meta-analysis projects. *BMJ Evidence-Based Med.* 28 (3), 197–203. https://doi.org/10.1136/bmjebm-2022-111931.
Robinson, O., Tamayo, I., de Castro, M., Valentin, A., Giorgis-Allemand, L., Krog, N.H., Aasvang, G.M., Ambros, A., Ballester, F., Bird, P., Chatzi, L., Cirach, M., Dėdelė, A., Donaire-Gonzalez, D., Gražuleviciene, R., Iakovidis, M., Ibarluzea, J., Kampouri, M., Lepeule, J., Basagaña, X., 2018. The urban exposome during pregnancy and its socioeconomic determinants. *Environ. Health Perspect.* 126 (7). https://doi.org/10.1289/EHP2862.
Schembari, A., de Hoogh, K., Pedersen, M., Dadvand, P., Martinez, D., Hoek, G., Petherick, E.S., Wright, J., Nieuwenhuijsen, M.J., 2015. Ambient air pollution and newborn size and adiposity at birth: differences by maternal ethnicity (the Born in Bradford Study Cohort). *Environ. Health Perspect.* 123 (11), 1208–1215. https://doi.org/10.1289/ehp.1408675.
Sears, C.G., Mueller-Leonhard, C., Wellenius, G.A., Chen, A., Ryan, P., Lanphear, B.P., Braun, J.M., 2019. Early-life exposure to traffic-related air pollution and child anthropometry. *Environ. Epidemiol.* 3 (5), e061.
Sellier, Y., Galineau, J., Hulin, A., Caini, F., Marquis, N., Navel, V., Bottagisi, S., Giorgis-Allemand, L., Jacquier, C., Slama, R., Lepeule, J., 2014. Health effects of ambient air pollution: Do different methods for estimating exposure lead to different results? *Environ.Int.*
Spencer-Hwang, R., Hwang, J., Sinclair, R., Dennis, S., Montgomery, S., 2023. Adverse health outcomes in early childhood (birth to 5 years) and ambient air pollutant exposures: a systematic review. *Air Qual. Atmos. Health* 16 (5), 913–944. https://doi.org/10.1007/s11869-023-01308-1.
Starling, A.P., Moore, B.F., Thomas, D.S.K., Peel, J.L., Zhang, W., Adgate, J.L., Magzamen, S., Martenies, S.E., Allshouse, W.B., Dabelea, D., 2020. Prenatal exposure to traffic and ambient air pollution and infant weight and adiposity: the Healthy Start study. *Environ. Res.* 182, 109130. https://doi.org/10.1016/j.envres.2020.109130.

Tainio, M., Jovanovic Andersen, Z., Nieuwenhuijsen, M.J., Hu, L., de Nazelle, A., An, R., Garcia, L.M.T., Goenka, S., Zapata-Diomedi, B., Bull, F., de Sá, T.H., 2021. Air pollution, physical activity and health: a mapping review of the evidence. Environ. Int. 147 (February 2021), 105954. https://doi.org/10.1016/j.envint.2020.105954.

Toledo-Corral, C.M., Alderete, T.L., Habre, R., Berhane, K., Lurmann, F.W., Weigensberg, M.J., Goran, M.I., Gilliland, F.D., 2018. Effects of air pollution exposure on glucose metabolism in Los Angeles minority children. *Pediatr. Obes.* 13 (1), 54–62. https://doi.org/10.1111/ijpo.12188.

UNESCO, 2006. International Standard Classification of Education (ISCED 1997).

van Eijsden, M., Vrijkotte, T.G., Gemke, R.J., van der Wal, M.F., 2011. Cohort profile: the Amsterdam born children and their development (ABCD) study. *Int. J. Epidemiol.* 40 (5), 1176–1186. https://doi.org/10.1093/ije/dyq128.

Viechtbauer, W., Cheung, M.-W.-L., 2010. Outlier and influence diagnostics for meta-analysis. *Res. Synth. Methods* 1 (2), 112–125. https://doi.org/10.1002/jrsm.11.

Vrijheid, M., Fossati, S., Maitre, L., Márquez, S., Roumeliotaki, T., Agier, L., Andrusaityte, S., Cadiou, S., Casas, M., de Castro, M., Dedele, A., Donaire-Gonzalez, D., Grazuleviciene, R., Haug, L.S., McEachan, R., Meltzer, H.M., Papadopouplou, E., Robinson, O., Sakhi, A.K., Chatzi, L., 2020. Early-life environmental exposures and childhood obesity: an exposome-wide approach. *Environ. Health Perspect.* 128 (6), 1–14. https://doi.org/10.1289/EHP5975.

Wang, M., Aaron, C.P., Madrigano, J., Hoffman, E.A., Angelini, E., Yang, J., Laine, A., Vetterli, T.M., Kinney, P.L., Sampson, P.D., Sheppard, L.E., Szpiro, A.A., Adar, S.D., Kirwa, K., Smith, B., Lederer, D.J., Diez-Roux, A.V., Vedal, S., Kaufman, J.D., Barr, R. G., 2019. Association between long-term exposure to ambient air pollution and change in quantitatively assessed emphysema and lung function. *J. Am. Med. Assoc.* 322 (6), 546. https://doi.org/10.1001/jama.2019.10255.

Wang, Z., Zhao, L., Huang, Q., Hong, A., Yu, C., Xiao, Q., Zou, B., Ji, S., Zhang, L., Zou, K., Ning, Y., Zhang, J., Jia, P., 2021. Traffic-related environmental factors and childhood obesity: a systematic review and meta-analysis. *Obes. Rev.* 22 (S1). https://doi.org/10.1111/obr.12995.

Warkentin, S., de Bont, J., Abellan, A., Pistillo, A., Saucy, A., Cirach, M., Nieuwenhuijsen, M., Khalid, S., Basagaña, X., Duarte-Salles, T., Vrijheid, M., 2023. Changes in air pollution exposure after residential relocation and body mass index in children and adolescents: a natural experiment study. *Environ. Pollut.* 334, 122217. https://doi.org/10.1016/j.envpol.2023.122217.

Weldy, C.S., Liu, Y., Liggitt, H.D., Chin, M.T., 2014. In utero exposure to diesel exhaust air pollution promotes adverse intrauterine conditions, resulting in weight gain, altered blood pressure, and increased susceptibility to heart failure in adult mice. *PLoS One* 9 (2), e88582. https://doi.org/10.1371/journal.pone.0088582.

West, J., Lawlor, D.A., Fairley, L., Bhopal, R., Cameron, N., McKinney, P.A., Sattar, N., Wright, J., 2013. UK-born Pakistani-origin infants are relatively more adipose than white British infants: Findings from 8704 mother-offspring pairs in the Born-in-Bradford prospective birth cohort. *J. Epidemiol. Community Health* 67 (7), 544–551. https://doi.org/10.1136/jech-2012-201891.

West, J., Santorelli, G., Whincup, P.H., Smith, L., Sattar, N.A., Cameron, N., Farrar, D., Collings, P., Wright, J., Lawlor, D.A., 2018. Association of maternal exposures with adiposity at age 4/5 years in white British and Pakistani children: findings from the Born in Bradford study. *Diabetologia* 61 (1), 242–252. https://doi.org/10.1007/s00125-017-4457-2.

Wilding, S., Ziauddeen, N., Smith, D., Roderick, P., Chase, D., Alwan, N.A., 2020. Are environmental area characteristics at birth associated with overweight and obesity in school-aged children? Findings from the SLOPE (Studying Lifecourse Obesity PrEdictors) population-based cohort in the south of England. *BMC Med.* 18 (1), 43. https://doi.org/10.1186/s12916-020-01513-0.

Woo, J.G., Daniels, S.R., 2019. Assessment of Body Mass Index in Infancy: It Is Time to Revise Our Guidelines. *J. Pediatr.* 204, 10–11. https://doi.org/10.1016/j.jpeds.2018.09.025.

World Health Organization, 2016. Report of the commission on ending childhood obesity. World Health Organization. https://iris.who.int/handle/10665/204176.

World Health Organization, 2021. Obesity and overweight. https://www.who.int/en/news-room/fact-sheets/detail/obesity-and-overweight.

World Health Organization (WHO) Multicentre Growth Reference Study Group, 2006. WHO child growth standards: Length/height-for-age, weight-for-age, weight-for-length, weight-for-height and body mass index-for-age: Methods and development. 312.

Worldbank, 2023. Urban Development. https://www.worldbank.org/en/topic/urbandevelopment.

Wright, J., Small, N., Raynor, P., Tuffnell, D., Bhopal, R., Cameron, N., Fairley, L., Lawlor, D.A., Parslow, R., Petherick, E.S., Pickett, K.E., Waiblinger, D., West, J., 2013. Cohort Profile: The Born in Bradford multi-ethnic family cohort study. *Int. J. Epidemiol.* 42 (4), 978–991. https://doi.org/10.1093/ije/dys112.

Wu, Y., Gong, Q., Zou, Z., Li, H., Zhang, X., 2017. Short sleep duration and obesity among children: a systematic review and meta-analysis of prospective studies. *Obes. Res. Clin. Pract.* 11 (2), 140–150. https://doi.org/10.1016/j.orcp.2016.05.005.

Yuan, M., Yin, C., Sun, Y., Chen, W., 2019. Examining the associations between urban built environment and noise pollution in high-density high-rise urban areas: a case study in Wuhan, China. *Sustain. Cities Soc.* 50, 101678. https://doi.org/10.1016/j.scs.2019.101678.

Zhang, D., Chen, G., Song, Y., Li, S., Yang, Z., Dong, Y., Wang, Z., Ma, J., Guo, Y., 2021. Ambient air pollution and obesity in school-aged children and adolescents: a multicenter study in China. *Sci. Total Environ.* 771, 144583. https://doi.org/10.1016/j.scitotenv.2020.144583.

Zhang, J., Zeng, X., Du, X., Pan, K., Song, L., Song, W., Xie, Y., Zhao, J., 2019. Parental $PM_{2.5}$ exposure-promoted development of metabolic syndrome in offspring is associated with the changes of immune microenvironment. *Toxicol. Sci.* 170 (2), 415–426. https://doi.org/10.1093/toxsci/kfz109.

Zheng, J., Zhang, H., Shi, J., Li, X., Zhang, J., Zhang, K., Gao, Y., He, J., Dai, J., Wang, J., 2024. Association of air pollution exposure with overweight or obesity in children and adolescents: a systematic review and meta-analysis. Sci. Total Environ. 910, 168589. https://doi.org/10.1016/j.scitotenv.2023.168589.