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Urban environment in pregnancy and postpartum depression: An individual participant data meta-analysis of 12 European birth cohorts

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ABSTRACT

Background: Urban environmental exposures associate with adult depression, but it is unclear whether they are associated to postpartum depression (PPD).
Objectives: We investigated associations between urban environment exposures during pregnancy and PPD.
Methods: We included women with singleton deliveries to liveborn children from 12 European birth cohorts (N with minimum one exposure = 30,772, analysis N range 17,686–30,716 depending on exposure; representing 26–46 % of the 66,825 eligible women). We estimated maternal exposure during pregnancy to ambient air pollution with nitrogen dioxide (NO_2) and particulate matter ($PM_{2.5}$ and PM_{10}), road traffic noise (L_{den}), natural spaces (Normalised Difference Vegetation Index; NDVI, proximity to major green or blue spaces) and built environment (population density, facility richness and walkability). Maternal PPD was assessed 3–18 months after birth using self-completed questionnaires. We used adjusted logistic regression models to estimate cohort-specific associations between each exposure and PPD and combined results via *meta*-analysis using DataSHIELD.

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Results: Of the 30,772 women included, 3,078 (10 %) reported having PPD. Exposure to PM_{10} was associated with slightly increased odds of PPD (adjusted odd ratios (OR) of 1.08 [95 % Confidence Intervals (CI): 0.99, 1.17] per inter quartile range increment of PM_{10}) whilst associations for exposure to NO_2 and $PM_{2.5}$ were close to null. Exposure to high levels of road traffic noise (≥65 dB vs. < 65 dB) was associated with an OR of 1.12 [CI: 0.95, 1.32]. Associations between green spaces and PPD were close to null; whilst proximity to major blue spaces was associated with increased risk of PPD (OR 1.12, 95 %CI: 1.00, 1.26). All associations between built environment and PPD were close to null. Multiple exposure models showed similar results.
Discussion: The study findings suggest that exposure to $PM_{10,}$ road traffic noise and blue spaces in pregnancy may increase PPD risk, however future studies should explore this causally.

1. Introduction

Maternal postpartum depression (PPD) is defined within the Diagnostic and Statistical Manual of Mental Disorders (DSM-V) as depressed mood, anxiety and anhedonia within four weeks of giving birth (American Psychiatric Association, 2013). PPD is a major public health challenge estimated to affect 12–25 % of women in high-income countries (Norhayati et al., 2015; Shorey et al., 2018). Risk factors include low socioeconomic position, previous history of depression, intimate partner violence, substance misuse and limited social support (Norhayati et al., 2015; Yang et al., 2022). There is debate over the nosology of PPD, with International Classification of Diseases (ICD)-10 codes not including a specific category for PPD (World Health Organization, 1992), and the DSM-V classifying PPD as a subset of perinatal depression (American Psychiatric Association, 2013). However there is evidence that PPD is characterised by more anxious features than other adult depression, and a higher risk of PPD in subsequent pregnancies (Cooper and Murray, 1995). Furthermore, the postpartum period is important because depression during this time may interfere with the mother's ability to care for the baby and PPD is a risk factor for child mental health problems (Stein et al., 2014).

Currently more than 50 % of the world population lives in cities, a figure projected to rise to 70 % by 2050 (Un, 2015). Notwithstanding the multiple potential benefits of living in urban areas, there is growing evidence that exposure to urban environmental stressors such as ambient air pollution, road traffic noise and lack of access to natural spaces may be detrimental to mental health (Callaghan et al., 2020: Gong et al.,2016; Kotzeva and Brandmüller, 2016; Tzivian et al., 2015). These types of environmental exposures could impact mental health through biological and social mechanisms. For example, exposure to ambient air pollutants has been linked with neurotoxic effects and disturbance of hypothalamus–pituitary–adrenal (HPA) functioning (Amitai et al., 1998; Gruzieva et al., 2017; Thomson, 2019), road traffic noise exposure may cause stress and disrupt sleep (Smith et al., 2022), and lack of natural spaces may reduce opportunities to relax, socialize and exercise (Pirrera et al., 2010; Hartig et al., 2014).

There is a growing epidemiological evidence that urban environmental exposures (such as air pollution) (Fan et al., 2020) may increase the risk of adult depression, however few studies have focused on PPD. Whilst the risk factors are similar (American Psychiatric Association, 2013), the unique context of pregnancy and the transition to motherhood may make women in the perinatal period more susceptible to such exposures. In the few epidemiological studies that have been conducted, pregnancy exposure to higher levels of ambient air pollution with particulate matter (PM) with a diameter smaller than 10 μm (PM_{10}) (Bastain et al., 2021; Duan et al., 2022; Niedzwiecki et al.,2020) and nitrogen dioxide (NO_2) (Bastain et al., 2021; Duan et al., 2022; Shih et al., 2021) have been associated with higher risk of PPD, however evidence for associations between $PM_{2.5}$ exposure and PPD are less consistent (Bastain et al., 2021; Duan et al., 2022; Niedzwiecki et al., 2020; Pourhoseini et al., 2022; Sheffield et al., 2018; Shih et al., 2021; Zhang et al., 2021). Studies have also found that exposure to a range of air pollutants including those mentioned above is associated with increased risk of depression during pregnancy (Lamichhane et al., 2021; Kanner et al., 2021).

Evidence for associations between other urban environmental exposures and PPD is very limited. To our knowledge only two studies have examined associations between exposure to residential road traffic noise and perinatal depression. They suggested that noise exposure was associated with increased risk of antenatal depression (Jigeer et al., 2022), and that a high level of night time noise (>70 dB) was associated with an increased risk of hospitalisation for PPD (He et al., 2019). A recent study investigating the association between surrounding green space and PPD found a small protective effect for some, but not all measures of greenness (Sun et al., 2023). Two studies investigating natural space exposure and antenatal depression reported mixed findings (McEachan et al., 2016; Nichani et al., 2017), and we are unaware of any studies investigating pregnancy exposure to natural spaces and PPD. There is emerging evidence that living in a more walkable neighbourhood may be protective against depression and loneliness (Joshi et al., 2017; Domènech-Abella et al., 2020) including for mothers in the postnatal period (Zhang et al., 2021). Given that social isolation is a risk factor for PPD (Norhayati et al., 2015), we also hypothesise that living in a neighbourhood with greater availability of facilities (such as sport and cultural facilities) could be a protective factor for PPD, however as far as we are aware this has not yet been examined.

In summary, evidence that urban environmental exposures constitute risk factors for PPD is limited and inconsistent. The aim of the present study was to estimate associations between several urban environmental exposures during pregnancy and PPD using harmonised data from multiple European birth cohorts. We hypothesised that (i) residential exposure to ambient air pollution (NO_2, $PM_{2.5}$ and PM_{10}) and road traffic noise (L_{den}) would be associated with higher risk of PPD, and (ii) that residential exposure to natural spaces (Normalised Difference Vegetation Index, NDVI and proximity to major green and blue spaces), increased facility richness and walkability would be associated with lower risk of PPD. As there is very limited previous research, we did not specify a direction of association between population density and PPD.

2. Materials and methods

2.1. Study population

Our study is based on the EU Child Cohort Network (EUCN) (Jaddoe et al., 2020; Pinot de Moira et al., 2021). We obtained data from twelve (out of 22 in the network) European birth cohort studies from eight countries (Table 1), with available data on at least one of the selected environmental exposures and PPD. Together, these twelve cohorts included data on 78,916 participants. We restricted the study population to mothers with (i) singleton pregnancies resulting in a liveborn infant, (ii) no previous history of depression and (iii) complete data on at least one of the selected environmental exposures, PPD, and confounders (selected *a priori* and defined as detailed below). Mothers with a previous history of depression were excluded to identify a sample of women for whom depression began during or after pregnancy (thus corresponding as closely as possible to the DSM-V criteria of depression with perinatal onset). The analysis sample ranged from N = 17,686 (26 % of eligible) in the PM_{10} analyses to N = 30,716 (46 % of eligible women) in

the analyses of green space (Fig. 1).

All participant women gave written informed consent and ethical approval was provided by cohort-specific ethics boards. A summary of the cohort characteristics is provided in Table 1. The analysis plan provided to all cohorts can be viewed at https://osf.io/7cv2d/.

2.2. Exposure assessment

Maternal residential address(es) during pregnancy were used to assign environmental exposures, for further detail see (Robinson et al., 2018; Binter et al., 2022; Maitre et al., 2018; Torres Toda et al., 2022). For all exposures we estimated the average level across pregnancy. The mean concentrations of NO_2, $PM_{2.5,}$ and PM_{10} 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) or the Effects of Low-Level Air Pollution: A Study in Europe (ELAPSE) project (de Hoogh et al., 2018). We used the available air pollution data from the nearest routine air quality monitoring data and ratio-methods to back-extrapolate the estimated time-weighted mean air pollution levels to the exact time period of the individual pregnancies as air sa mpling campaigns were conducted years after the pregnancies of the cohort participants (Pedersen et al., 2013). NO_2 and $PM_{2.5}$ were available for all cohorts and PM_{10} exposure was available for nine cohorts (ABCD, BiB, EDEN-Nancy, EDEN-Poitiers, GEN-R, INMA Sabadell, MoBa, NINFEA & Rhea).

The mean road traffic noise levels over days, evenings and nights (L_{den}) were estimated using existing European road traffic noise maps, which were generated under EC Directive 2002/49/EC (Assessment and Management of Environmental Noise) in the framework of the European Noise Directive (END). Noise exposure for the Rhea cohort was calculated using a newly developed noise map generated in the EXPOsOMICS project. Exposure to road traffic noise was coded as a binary variable (exposed defined as >= 65 dB), as not all cohorts had available continuous data. Data was available for all cohorts except INMA Gipuzkoa.

Three indicators of exposure to natural spaces were selected for the present study. Average exposure to green vegetation (e.g. trees, parks shrubland) was captured using NDVI within a 300 m buffer from the centroid of the residential address derived from the Landsat 4–5 Thematic Mapper I, Landsat 7 Enhanced Thematic Mapper Plus (ETM +), and Landsat 8 Operational Land Imager (OLI)/Thermal Infrared Sensor (TIRS) with 30 m × 30 m resolution (Nieuwenhuijsen et al., 2014). NDVI quantifies vegetation by measuring the difference between near-infrared and red-light reflection based on satellite imagery. Values range from −1 to 1: negative values represent water, low values (0 to 0.1) indicate areas of barren rock, sand or snow; moderate values (0.2 to 0.5) indicate sparse vegetation such as shrubs and grasslands, whilst high values (0.6 to 1) indicate dense vegetation (Weier). Since our focus was on vegetation, negative values were removed leaving a range of 0 to 1. Two additional binary variables were derived indicating residential proximity to green and blue spaces, as indicators of access to these spaces. These were defined as living within a straight-line distance of 300 m of a major green space (parks or countryside) or blue space (sea, lakes, fish ponds, rivers, canals) with an area > 5,000 m^2 (yes or no). These were calculated using the European Environmental Protection Agency Urban Atlas for all cohorts except INMA Gipuzkoa (European Nature Information System data used) and MoBa (local data used) (Urban Atlas). Data was available for all cohorts.

We included three features of the built environment: (i) facility richness (300 m buffer), (ii) walkability (300 m buffer) and (iii) population density (100 m buffer). Facility richness was 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; possible facility types given in Table S1). 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 (Data from: GHS-POP R, 1975) for all cohorts except MoBa for which local data were used (Robinson et al., 2018). Finally, a walkability index was developed consisting of an average of four other indices: (i) Land use Shannon's Evenness Index (an indicator of the evenness of land use (range 0–1) with higher values indicating a more even distribution of land) (Shannon, 1948), (ii) Facility richness, (iii) Population density and (iv) Connectivity index (number of street intersections within 300 m buffer) with a range of 0–1 (1 = most walkable) (Binter et al., 2022; Frank et al., 2006). Information on built environment was available for all cohorts.

2.3. Outcome definition

We categorized maternal PPD as a binary variable (yes/no) based on maternal self-report, with eleven out of twelve cohorts using validated questionnaires. Six cohorts used versions of the Edinburgh Postnatal Depression Scale (ALSPAC, EDEN-Nancy, EDEN-Potiers, GEN-R, MoBa, Rhea) (Cox et al., 1987), three cohorts used the General Health Questionnaire (BiB, INMA Gipuzkoa, INMA Sabadell) (Goldberg et al., 1997), one cohort used the Center for Epidemiologic Studies Depression Scale (ABCD), and one cohort used data from two questionnaires (Symptom Distress Checklist and the General Health Questionnaire; DNBC). Validated cut-offs were used to define the risk of clinical depression. The cohort not applying a validated questionnaire collected data on women-reported doctor diagnoses (NINFEA). Harmonised data on PPD were available at one time point in each cohort, with the time of measurement varying from 2 months (GEN-R) to 18 months after birth (INMA Gipuzkoa, INMA Sabadell & NINFEA). Full details of the measures, cut-offs and measurement periods are described in Table S2 and accompanying text in the Supplemental Materials).

2.4. Confounder definition

We used Directed Acyclic Graphs (DAGs) to depict confounders and determine whether there was any evidence of colliders that we should not adjust for (Figure S1). In our main analysis we included maternal

Table 1
Characteristics of the prospective birth cohorts.

Cohort name	Country	City/area	Subsample[a]	N included	Birth year(s)
ABCD	Netherlands	Amsterdam	Prospective	4,490	2003—2004
ALSPAC	The United Kingdom	Greater Bristol	Greater Bristol	7,141	1991—1992
BiB	The United Kingdom	Bradford	–	1,024	2007—2011
DNBC	Denmark	Nationwide	Greater Copenhagen	4,750	1996—2003
EDEN Nancy	France	Nancy	–	749	2003—2005
EDEN Poitiers	France	Poitiers	–	677	2003—2005
Gen-R	The Netherlands	Rotterdam	–	3046	2002—2006
INMA Gipuzkoa	Spain	Gipuzkoa	–	4,64	1997—2008
INMA Sabadell	Spain	Sabadell	–	4,82	1997—2008
MoBa	Norway	Nationwide	Greater Oslo	6,475	1999—2008
NINFEA	Italy	Nationwide	Florence, Rome & Turin	1,181	2005—2016
Rhea	Greece	Crete	Heraklion	293	2007—2008

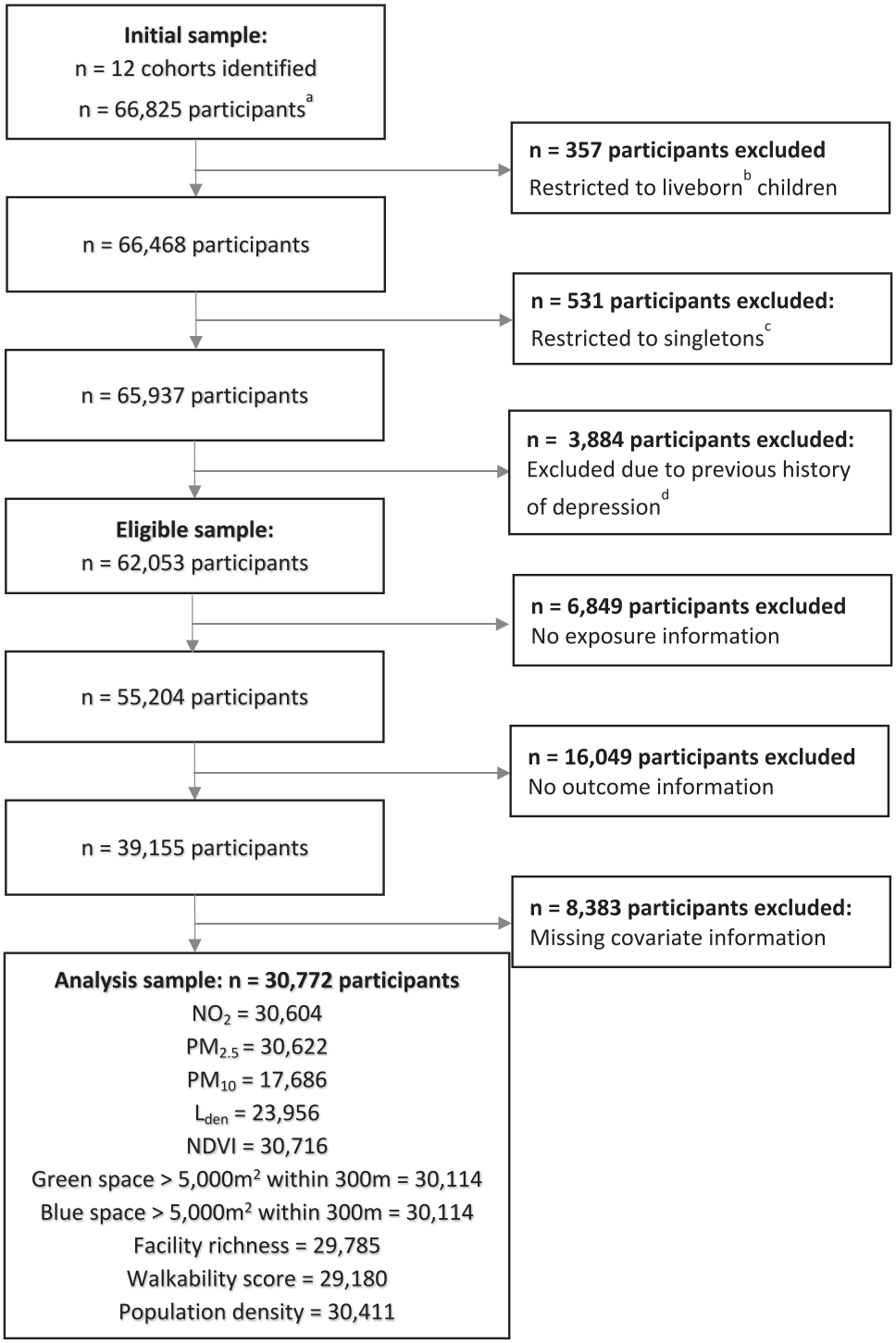

Fig. 1. Flow chart of included participants [a]Refers to participants within each cohort living within areas for which environmental exposures were modelled (Table 1) [b]Data on birth outcome unavailable for MoBa; [c]Data on multiple birth unavailable for GEN-R; [d]Defined as women self-reporting either a history or depression or other psychiatric problems prior to pregnancy.

education in pregnancy (low, medium, high), disposable family income in quintiles (Pizzi et al., 2020), area-specific deprivation for the residential address in pregnancy (low, medium, high; detail of the indices provided in Table S3), parity prior to the index pregnancy (nulliparous vs other), maternal age (years), year and season of birth. Maternal ethnicity (defined as Western vs other) fit our definition of a confounder, but was only available with sufficient variability in four out of the twelve cohorts (ABCD, ALSPAC, BiB, Gen-R, maximum n = 15,607, representing 58 % of the 30,772 participants) and thus was only included in a sensitivity analysis.

2.5. Statistical analysis

We performed federated analysis using DataSHIELD version 6.10 (Gaye et al., 2014) and R version 3.52 (code available at: https://github.com/lifecycle-project/env-pnd). DataSHIELD is a platform for federated data analysis which prevents disclosure of personal identifiable data by preventing researchers from viewing individual participant data and

involves no data transfer. Instead, analysis commands are sent to local servers on which harmonised individual participant data are stored and processed. Only non-disclosive summary statistics are returned.

Our analysis proceeded as follows. First, we described the distribution of the exposures, outcomes and covariates along with (Pearson) correlations between exposures. We then estimated associations between each exposure and PPD (adjusting for confounders) separately for each cohort using logistic regression and combined results via random-effects meta-analysis. We chose random-effects meta-analysis because we hypothesised that due to differences in demographics and urban design of cities in the study centres, the true effect size may vary between study (Borenstein et al., 2010; Robinson et al., 2018). We also hypothesised that some exposures may confound the association between other environmental exposures and PPD (as depicted in the DAG via maternal residential address as an unmeasured common cause); therefore we additionally fit models adjusting air pollutants for road traffic noise, natural spaces and built environment, and road traffic noise for air pollution, natural spaces and built environment (Figure S1). We described heterogeneity between cohorts both qualitatively and using Cochran's Q statistic. Continuous exposure variables were scaled by the interquartile range calculated across all studies whilst binary and categorical exposures were left untransformed.

We conducted the following sensitivity analyses. First, we repeated analyses additionally adjusting for ethnicity in the four cohorts with available data. We also compared findings where PPD was assessed using the EPDS (a specific PPD questionnaire) vs other depression measures.

We handled missing data through complete case analysis, as neither multiple imputation nor inverse probability weighting functionalities were available in DataShield at the time of analysis. To explore missingness of data, we compared the characteristics of participants included in the analysis sample (minimum one environmental exposure, PPD and core confounders, N = 30,772) with those excluded. Some differences were observed: those excluded had on average lower income, lived in more deprived areas and had higher rates of PPD (Table S4).

3. Results

3.1. Study participant characteristics

Approximately a third of participants were from Scandinavian countries and a quarter from the United Kingdom. Participants were mostly highly educated, nulliparous, and cohabiting women of Western European origin (Table 2). A total of 3,078 (10 %) PPD cases were identified, with the percentage of women meeting threshold for PPD ranging from 3 % in NINFEA to 19 % in BiB (Table S5). Women with PPD had on average lower education, lower income and were from more deprived areas compared to those without PPD. There were considerable differences between cohorts in demographics, for example 85 % of women from BiB lived in highly deprived neighbourhoods compared to 1 % in INMA Gipuzkoa, and the proportion of nulliparous women varied from 41 % in BIB to 73 % in NINFEA. The proportion with complete data who contributed to at least one exposure-PPD analyses varied from 25 % (Rhea) to 78 % (INMA Sabadell), with full details for each study provide in Table S6.

The median pregnancy concentrations of NO_2 exposure ranged from 11.6 (Rhea) to 46.6 μg/m^3 (Copenhagen area of DNBC); $PM_{2.5}$ levels ranged from 9.3 (BiB) to 23.3 μg/m^3 (NINFEA), and PM_{10} levels ranged from 13.6 (Oslo area of MoBa) to 40.1 μg/m^3 (NINFEA). The proportion of women exposed to high levels of road traffic noise (>= 65 dB) was greatest in NINFEA (36 %) and lowest in ALSPAC (2 %). NDVI levels ranged from 0.16 (Rhea) to 0.53 (Eden Poitiers). In all cohorts, most women lived within 300 m of a major green space, but only two cohorts had a high percentage of women living close to blue spaces (GEN-R & INMA Gipuzkoa). ABCD had the highest levels of facility richness and walkability whilst EDEN Poitiers had lowest levels. Population density ranged from 1,660 inhabitants/km^2 (EDEN Poitiers) to 12,000 inhabitants/km^2 (INMA Sabadell).

Table 2
Study population characteristics for all and by postpartum depression (PPD).

Variable	Median (IQR) for continuous variables / N (%) for categorical variables		
	All (N = 30,772)	No PPD (N = 27,694)	PPD (N = 3,078)
Birth season			
January – March	8708 (28.3)	7843 (28.3)	863 (28.4)
April – June	6875 (22.3)	6170 (22.3)	694 (22.8)
July - September	7851 (25.5)	7056 (25.5)	783 (25.8)
October - December	7338 (23.8)	6625 (23.9)	701 (23)
Parity			
Nulliparous	13,306 (43.2)	11,855 (42.8)	1451 (47.1)
Not nulliparous	17,466 (56.8)	15,839 (57.2)	1627 (52.9)
Maternal age at birth (years)			
15—–20	498 (1.63)	421 (1.5)	34 (1.5)
21 – 30	14,149 (46.4)	12,873 (46.5)	974 (43.3)
31—–40	15,419 (50.6)	14,031 (50.7)	1203 (53.5)
41 – 50	413 (1.4)	369 (1.3)	38 (1.7
Maternal education at birth			
High	17,275 (56.1)	15,671 (56.6)	1116 (44.1)
Medium	10,086 (32.8)	9174 (33.1)	854 (33.8)
Low	3411 (11.1)	2849 (10.3)	560 (22.1)
Household disposable income			
First quintile	4503 (14.6)	3855 (13.9)	648 (21)
Second quintile	5695 (18.5)	5038 (18.2)	657 (21.4)
Third quintile	7935 (25.8)	7202 (26)	733 (23.8)
Fourth quintile	6861 (22.3)	6300 (22.8)	561 (18.2)
Fifth quintile	5778 (18.8)	5299 (19.1)	479 (15.6)
Maternal ethnicity			
Western origin	14,598 (82.0)	12,469 (82.8)	1034 (62.8)
Non-western origin	3204 (18)	2584 (17.2)	613 (37.2)
NO_2 (μg/m^3)	31.4 (27.4, 35.7)	31.2 (27.2, 35.5)	32.8 (28.9, 37.0)
$PM_{2.5}$ (μg/m^3)	15.3 (14.1, 16.6)	15.3 (14.0, 16.6)	15.5 (14.4, 16.7)
PM_{10} (μg/m^3)	24.5 (22.3, 27.2)	24.4 (22.2, 27.2)	25 (23.3, 27.3)
NDVI (range 0–1; 300 m buffer)	0.4 (0.34, 0.47)	0.4 (0.34, 0.47)	0.38 (0.32, 0.46)
L_{den}			
<55 dB	21,085 (88.0)	19,017 (88.0)	2042 (88.1)
>=65 dB	2,871 (12.0)	2593 (12.0)	276 (11.9)
Major green space > 5,000 m^2 within 300 *m*			
No	8,517 (28.3)	7603 (28.1)	913 (30.6)
Yes	21,597 (71.7)	19,490 (71.9)	2076 (69.4)
Major blue space > 5,000 m^2 within 300 *m*			
No	21,920 (72.8)	19,875 (73.4)	2014 (67.4)
Yes	8,194 (27.2)	7218 (26.6)	974 (32.6)
Facility richness (0–1; 300 m buffer)	0.09 (0.04,0.15)	0.09 (0.04,0.15)	0.1 (0.05,0.17)
Walkability (0–1; 300 m buffer)	0.33 (0.28,0.38)	0.33 (0.28,0.38)	0.34 (0.29,0.4)
Population density (persons km^2 in 100 m)	5900 (4020,9190)	5860 (3990,9150)	6270 (4280,9700)
Area deprivation			
Low	10,493 (34.1)	9531 (34.4)	960 (33.3)
Medium	8344 (27.1)	7592 (27.4)	727 (25.2)
High	11,935 (38.8)	10,571 (38.2)	1196 (41.5)

Figure S2 shows correlations between exposures for each cohort. Correlations between air pollutants were moderate to high (r = 0.25 to 0.96); the correlation between NO_2 and L_{den} ranged from low to moderate (r = 0.01 to 0.26), and air pollutants were negatively correlated with NDVI (r = -0.01 to −0.70). Built environment exposures were highly correlated, especially walkability and facility richness (r = 0.38 to 0.82).

3.2. Associations between urban environment exposures and PPD in single exposure models

Meta-analysed results are shown in Fig. 2 with study-specific results in Figures S3 – S6. Exposure to PM_{10} was associated with increased risk of PPD, although estimates were compatible with no association. Associations between exposure to NO_2, $PM_{2.5}$ and PPD were close to null. Heterogeneity between cohorts was low (NO_2 Q = 13, p = 0.30; $PM_{2.5}$ Q = 13, p = 0.31, PM_{10} Q = 5.3, p = 0.72), and ORs > 1 observed in 6 out of 12 studies for NO_2, 7 out of 12 studies for $PM_{2.5}$ and 6 out of 9 studies for PM_{10} (Figure S3).

Exposure to road traffic noise >=65 dB (vs < 65 dB) was associated with an increased risk of PPD (OR = 1.12, CI 0.95, 1.32) though results were also compatible with no association. Heterogeneity between cohorts was low (Q = 14.1, p = 0.17), with 5 out of 11 cohorts having an OR > 1 (Figure S4).

Associations between NDVI and the presence of major green spaces near the home residence were close to null (Fig. 2), and heterogeneity was low (NDVI Q = 4.2, p = 0.99; access to green space Q = 5.5, p = 0.92). Exposure to blue spaces near the home was associated with an increased risk of PPD (OR = 1.12, CI 1.00, 1.26), with 9 out of 12 cohorts having an OR > 1 (Figure S5).

In terms of the built environment, exposure to greater facility richness and walkability were associated with increased risk of PPD; however results were also consistent with no association (Figure S6). Heterogeneity was low (Facility richness Q = 9.6, p = 0.57; walkability Q = 11.5, p = 0.41), with 7 out of 12 cohorts showing OR > 1 for facility richness and 7 out of 12 cohorts for walkability. Associations between population density were null, with low statistical heterogeneity (Q = 16.8, p = 0.08).

3.3. Associations between urban environment exposures and PPD in multiple exposure models

Additionally adjusting air pollutant models for population density and NDVI (potential confounders) moved estimates slightly away from null (Fig. 2). Additionally adjusting the traffic noise model for population density and NDVI made minimal difference to estimates. Additionally adjusting air pollutant models for traffic noise and vice versa also did not notably change results.

3.4. Sensitivity analyses

We repeated analyses in the subset of 4 cohorts (ABCD, ALSPAC, BiB, GEN-R) with up to 15,607 participants with available data on ethnicity, and found that additionally adjusting for ethnicity changed estimates slightly with the direction varying between exposure (Table S7). We also stratified analysed depending on whether PPD was measured by EPDS or other measures. We found that the strength of association for air pollutants was slightly higher when PPD was measured using the EPDS vs. other measures, whilst for other exposures there was not a consistent pattern of differences. For all exposures 95 % CIs for estimates overlapped comparing estimates where PPD was assessed via the EPDS vs other measures (Table S8).

4. Discussion

In this study of individual level data on up to 30,772 women from twelve European birth cohorts and eight countries, we found suggestive evidence for associations between some of the selected environmental exposures and PPD, but not for all of them. As hypothesised, we found some indication that increased exposure to PM_{10} and high levels of road traffic noise were associated with increased risk of PPD. However, associations for the pooled study population were null for NO_2 and $PM_{2.5}$ and contrary to hypotheses we found that having a residence near to a major blue space was associated an increased risk. For many of the examined associations we observed considerable heterogeneity between cohorts, and except for proximity to a major blue space all pooled effect estimates were compatible with no association.

Exposure	Adjustment	K	No PPD	PPD	I2	OR [95% CI]
Air pollutants						
NO2	Single exposure	12	27556	3048	0.05	0.98 [0.93, 1.03]
	Pop. density & NDVI	11	26792	2990	0	0.97 [0.92, 1.03]
	Pop. density, NDVI & Lden	11	21433	2314	0	0.98 [0.91, 1.04]
PM2.5	Single exposure	12	27573	3049	11.87	1.04 [0.97, 1.11]
	Pop. density & NDVI	11	26809	2991	8.5	1.03 [0.96, 1.11]
	Pop. density, NDVI & Lden	11	21448	2315	13.8	1.05 [0.96, 1.14]
PM10	Single exposure	9	15916	1770	0	1.08 [0.99, 1.17]
	Pop. density & NDVI	9	15776	1756	0	1.06 [0.97, 1.16]
	Pop. density, NDVI & Lden	9	12903	1275	0	1.06 [0.97, 1.17]
Road traffic noise						
Lden >= 65 dB	Single exposure	11	21610	2346	12.95	1.12 [0.95, 1.32]
	Pop. density & NDVI	11	21549	2340	11.09	1.12 [0.95, 1.32]
	Pop. density, NDVI & PM2.5	11	21448	2315	13.64	1.12 [0.94, 1.33]
Natural spaces						
NDVI	Single exposure	12	27645	3071	0	0.99 [0.93, 1.05]
Major green space	Single exposure	12	27093	3021	2.22	0.98 [0.89, 1.07]
Major blue space	Single exposure	12	27093	3021	0	1.12 [1.00, 1.26]
Built environment						
Facility richness	Single exposure	12	26782	3003	11.37	1.04 [0.98, 1.11]
Walkability score	Single exposure	12	26230	2950	17.04	1.05 [0.98, 1.12]
Population density	Single exposure	11	26958	3022	0.05	0.99 [0.95, 1.04]

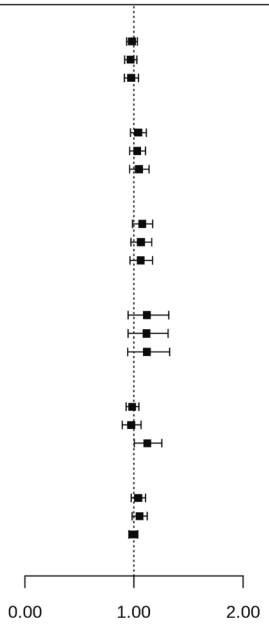

Fig. 2. Associations between urban environmental exposures and postpartum depression in single and multiple exposure models for the full study population using *meta*-analysis with random-effects on cohort. Note: Models adjusted for maternal education, disposable family income, area deprivation, parity, maternal age at birth, birth month and birth year. K = Number of cohorts. "Major green space" = green space > 5000 m^2 within 300 m or residential address; "Major blue space" = blue space > 5000 m^2 within 300 m of residential address. For binary variables (L_{den}, major green space, major blue space) estimates represent odds ratio of exposure category vs reference category, for all other (continuous) variables estimates represent odds ratio por IQR change in exposure.

4.1. Strengths and limitations

One of the major strengths of our study is its large geographical coverage including different regions of Europe, enabling us to cover different urban and cultural settings in our analysis. We included harmonised individual level data on multiple urban environmental exposures, PPD and a range of potential confounders. We were able to include multiple urban environmental exposures and account for potential confounding of one exposure by another. Since we had selected relevant exposures *a priori* for planned analyses and interpreted results based on point estimates and confidence intervals, we did not believe that adjustment for multiple testing was required. We had self-reported data on PPD which captures milder cases than if PPD was based on hospital contact and avoids selection bias into health services.

There were however limitations. First, due to technical limitations with the DataSHIELD infrastructure multiple imputation was not available at the time of analysis. We were therefore limited to complete case analysis, with the percentage of complete cases varying from 25 % (Rhea) to 78 % (EDEN Nancy). Complete case analysis is susceptible to bias where the outcome is related to probability of being a complete case (Hughes et al., 2019), and we observed that on average women included in the analysis sample had slightly lower rates of PPD compared to those excluded (10 % vs. 14 %). Technical limitations with DataSHIELD also meant that normality of variables had to be assessed via inspecting histograms rather than formal tests (e.g. Shaprio-Wilk test). In addition, whilst having participants from different study areas increases the exposure contrast, it also increases the risk of exposure misclassification as the methods and data available for each cohort were not identical. Our natural space indicators provide information about quantity of green and blue spaces within proximity to residential address, however they do not provide information about the access or quality of spaces (e.g. facilities, safety) which may affect their use. All exposures were estimated based on maternal residential address(es) during pregnancy and we did not consider maternal exposure in other microenvironments or other time periods. All our estimates for exposure were based on the location of the home and do not account for the location of the bedroom or apartments within buildings. This could have contributed to non-differential exposure misclassification, diluting the result towards the null. A further limitation was that the same questionnaires to assess PPD were not used in all cohorts and thus the reported rates of PPD are not directly comparable across cohorts. Self-reported assessment of mental health could also be influenced by recall bias and individual characteristics. It is less objective than clinical assessment and we also cannot rule out that self-reported assessment of PPD could have been influenced by unmeasured social and cultural differences that differentiate between and within the study areas such as intimate partner, household and/or residential neighbourhood level of support and crime. Furthermore, there was significant heterogeneity in the time points at which PPD was assessed in each cohort. This may have affected the prevalence and severity of PPD captured within each study. Due to the harmonisation process within the EUCCN, we were limited to a binary variable indicating PPD. Future research could use a continuous measure of PPD which may confer more power to detect associations. In order to focus on women for whom depression begun during or after pregnancy, we excluded women who reported a previous history of depression. Whilst this made our sample more homogeneous (and potentially more in line with the DSM-V criteria of depression with perinatal onset), we acknowledge the risk of introducing collider bias. In addition, as with all observational studies, there is a likelihood that associations are subject to unmeasured and residual confounding. Finally, we focused on long-term exposure to urban environmental exposures and it was not within the scope of this study to evaluate effects or effect modification by short-term exposure.

4.2. Interpretation of findings

Our finding that exposure to PM_{10} was associated with a small increased risk of PPD was consistent with previous studies. For example, a recent study in Shanghai, Hangzhou and Shaoxing, China (n = 10,209) reported that a 10 $\mu g/m^3$ increase in PM_{10} exposure in pregnancy was associated with an approximately 50 % increase in self-reported PPD risk (Duan et al., 2022). However, unlike previous studies we found the association between NO_2 and PPD to be close to null. By contrast, a study including 180 mothers in Los Angeles, USA reported that a standard deviation change in pregnancy NO_2 exposure was associated with a two-fold risk of self-reported PPD (Bastain et al., 2021), whilst a nation-wide study in Taiwan (n = 21,248) also reported a positive association between NO_2 exposure in the first trimester and self-reported PPD (Shih et al., 2021).

Our finding that residential exposure to high levels of road-traffic noise was associated with increased PPD risk was also consistent with our hypothesis and the previous research. Only one previous study (Montreal, Canada) has explored the relationship between traffic noise and hospitalisation for PPD (n = 140, 456) (He et al., 2019). Whilst they reported a close to null association between L_{den} and PPD, they found an increased risk of PPD with greater exposure to night-time noise. Two recent *meta*-analyses have also both reported a 4 % increased risk of adult depression per 10 dB increase in L_{den} (Dzhambov and Lercher, 2019; Hegewald et al. 2020). We lacked information on low noise levels in some cohorts and thus were unable to study effects below 55 dB, although we are aware that noise effects could start at lower levels (World Health Organization, 2018). Further research should replicate our findings using noise as a continuous variable including more fine-grained information on bedroom location relative to the street.

We found little evidence for an association between green space and PPD. A recent study conducted in California also found no association between NDVI and PPD, but did find an association between higher total green space exposure and lower PPD. Two previous studies which explored associations between NDVI and depression risk during pregnancy showed mixed findings. Whilst a previous study using the BiB cohort (n = 7,547) reported a 20 % reduced risk of depression in pregnancy for mothers living in the greenest quintile, a study in New Zealand (n = 6,772) reported close to null associations between the proportion of green space within a census unit and pregnancy depression (Nichani et al., 2017). Contrary to hypotheses, we found that exposure to blue spaces was associated with increased risk of depression. This was consistent with a previous study of 958 adults in Barcelona which similarly reported that adults living within 300 m of a major blue space had increased risk of self-reported depression. As we are not aware of a plausible mechanism by which blue space exposure could increase risk of PPD, it is likely that these findings are due either to residual confounding, or to a major blue space exposure being associated with other aspects of the built environment which themselves are associated with increased risk of PPD, such as ambient air pollution emissions from ships or unmeasured social features that cluster in areas near major blue spaces in our study areas.

In terms of the built environment, we found little evidence that either population density, facility richness or walkability were associated with PPD, with *meta*-analysed estimates close to one. Whilst no previous studies have been conducted using these exposures and PPD, two studies with older adults reported that increased walkability was associated with a lower risk of depression (Joshi et al., 2017, Domènech-Abella et al., 2020). However, again, the context of PPD is different to (older) adult depression and thus the risk factors also may differ.

5. Conclusions

Overall, we found some evidence that greater exposure to PM10, road traffic noise and blue spaces were associated with a higher risk of PPD. Most other associations evaluated were close to null. Many aspects

of the urban environment are highly correlated, so future research could extend on our findings by modelling how exposure to the whole urban exposome is associated with PPD. (Vrijheid,Sep, 2014) Causal inference methods should also be used estimate the causal effects of these exposures.

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CRediT authorship contribution statement

Tim Cadman: Writing – original draft, Visualization, Software, Project administration, Methodology, Funding acquisition, Formal analysis. **Katrine Strandberg-Larsen:** Writing – review & editing, Methodology, Conceptualization. **Lucinda Calas:** Writing – review & editing. **Malina Christiansen:** Writing – review & editing. **Iryna Culpin:** Writing – review & editing. **Payam Dadvand:** Writing – review & editing, Methodology. **Montserrat de Castro:** Writing – review & editing, Methodology. **Maria Foraster:** Writing – review & editing. **Serena Fossati:** Writing – review & editing, Methodology. **Mònica Guxens:** Writing – review & editing, Methodology. **Jennifer R. Harris:** Writing – review & editing. **Manon Hillegers:** Writing – review & editing. **Vincent Jaddoe:** Writing – review & editing. **Yunsung Lee:** Writing – review & editing. **Johanna Lepeule:** Writing – review & editing. **Hanan el Marroun:** Writing – review & editing. **Milena Maule:** Writing – review & editing. **Rosie McEachen:** Writing – review & editing, Methodology. **Chiara Moccia:** Writing – review & editing. **Johanna Nader:** Writing – review & editing. **Mark Nieuwenhuijsen:** Writing – review & editing, Methodology. **Anne-Marie Nybo Andersen:** Writing – review & editing. **Rebecca Pearson:** Writing – review & editing. **Morris Swertz:** Writing – review & editing. **Marina Vafeiadi:** Writing – review & editing. **Martine Vrijheid:** Writing – review & editing. **John Wright:** Writing – review & editing, Methodology. **Deborah A Lawlor:** Writing – review & editing, Methodology, Funding acquisition, Conceptualization. **Marie Pedersen:** Writing – review & editing, Methodology.

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.

Data availability

The data that has been used is confidential.

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For each cohort are provided in Supplementary Materials S2.

Appendix A. Supplementary material

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