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Particulate air pollution exposure during pregnancy and postpartum depression symptoms in women in Mexico City

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Abstract

Background: Postpartum depression (PPD), which affects up to 1 in 5 mothers globally, negatively impacts the health of both mothers and children. Exposure to ambient air pollution has been linked to depressive symptoms in animal models and human studies, but the relationship between air pollution and PPD has not been widely studied.

Methods: In a birth cohort in Mexico City (509 mothers with available data), we examined the association between exposure to particulate matter ≤2.5 μm in diameter ($PM_{2.5}$) with symptoms

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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.

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The authors declare no actual or potential conflicts of interest.

Appendix A. Supplementary material

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envint.2019.105325.

of psychological dysfunction at 1 and 6 months postpartum. Daily $PM_{2.5}$ estimates were derived from a hybrid satellite-based spatio-temporally resolved model and averaged over pregnancy and the first year postpartum. Edinburgh Postnatal Depression Scale (EPDS) scores at 1 and 6 months were used to assess the relationship between $PM_{2.5}$ exposure and probable PPD (EPDS score ≥13) using relative risk regression and symptoms of anhedonia, depression, and anxiety (derived from EPDS subscales) using negative binomial regression.

Results: A 5-$\mu g/m^3$ increase in average $PM_{2.5}$ exposure during pregnancy was associated with an increased risk of PPD at 6 months (RR = 1.59; 95% CI: 1.11 to 2.28) and of late-onset PPD (no PPD at 1 month, PPD at 6 months) (RR = 2.58; 95% CI: 1.40 to 4.73) in covariate-adjusted models. No association was observed between $PM_{2.5}$ exposure in the first year postpartum and PPD. Average $PM_{2.5}$ exposure during pregnancy was also associated with increased 6-month EPDS subscale symptom scores for anhedonia ($p = 0.03$) and depression ($p = 0.04$).

Conclusion: Our results suggest that in women in Mexico City, particulate matter exposure during pregnancy is positively associated with PPD and symptoms of anhedonia and depression at 6 months postpartum. Future studies should examine mechanisms linking air pollution and other environmental exposures during pregnancy with postpartum psychological functioning.

1. Introduction

Postpartum depression (PPD), also called postnatal depression, greatly impacts both the health of the infant and the mother (Gelaye et al., 2016; Nieto et al., 2017; Surkan et al., 2011; Wisner et al., 2006). PPD is a heterogeneous condition with three underlying dimensions—depressed mood, anxiety, and anhedonia—that may have varying times of onset during the postpartum period (Putnam et al., 2017). The prevalence of depressive postpartum symptoms varies worldwide: overall pooled prevalence is estimated to be 12% (Woody et al., 2017), with estimates ranging from 6 to 38% in developed countries and 20–57% in developing countries (Lara et al., 2015; Norhayati et al., 2015). In low- and middle-income countries, PPD is of particular concern given its high prevalence and the limited resources for its diagnosis and management (Lara et al., 2015; Place et al., 2016; Shrestha et al., 2016). Therefore, identifying modifiable risk factors for PPD is an important public health goal.

Evidence from animal and human studies suggests that ambient air pollution is associated with adverse neuropsychological functioning. In adult mice, exposure to ambient air pollutants has been associated with anxiety and depressive-like symptoms (Costa et al., 2014; Mokoena et al., 2015; Zhang et al., 2015). In human studies, most research has been focused on non-pregnant and aging populations. Increased exposure to particulate matter has been linked to depressive and anxiety symptoms in adults in South Korea (Shin et al., 2018), elderly populations in South Korea (Lim et al., 2012) and the United States (Pun et al., 2017), and in women in the Nurses' Health Study in the United States (Kioumourtzoglou et al., 2017; Power et al., 2015). Short-term exposure to $PM_{2.5}$ has also been linked to increased risk of hospitalization for depression in two studies in China (Qiu et al., 2018; Wang et al., 2018).

To our knowledge, only one previous study has examined the association between air pollution in pregnancy and postpartum psychological functioning. Our group reported that $PM_{2.5}$ exposure in mid-pregnancy was associated with increased postpartum anhedonia symptoms, measured using the Edinburgh Postnatal Depression Scale (EPDS), during the first year postpartum among a diverse sample including Black, Hispanic, and white mothers in Boston; further, after stratifying by race/ethnicity, the strongest associations of $PM_{2.5}$ with symptoms of depression and anhedonia were seen in the subpopulation of Black mothers (Sheffield et al., 2018). Herein, we leverage an ongoing birth cohort in Mexico City with data on air pollution and maternal psychological functioning to evaluate the association between exposure to $PM_{2.5}$ in pregnancy and psychological functioning, assessed using the EPDS, at 1 and 6 months postpartum.

2. Methods

2.1. Study population

Women who were pregnant and receiving prenatal care through the Mexican Social Security System (Instituto Mexicano del Seguro Social – IMSS) between July 2007 and February 2011 were recruited in the Programming Research in Obesity, GRowth, Environment and Social Stressors (PROGRESS) study. Women were eligible to participate in the study if they met the following criteria: ≥18 years old, < 20 weeks gestation, planned to stay in Mexico City for the next 3 years, had access to a telephone, had no medical history of heart or kidney disease, did not consume alcohol daily, and did not use any steroid or anti epilepsy medications. Procedures were approved by institutional review boards at the Harvard School of Public Health, Icahn School of Medicine at Mount Sinai, and the Mexican National Institute of Public Health. Women provided written informed consent in Spanish.

2.2. $PM_{2.5}$ levels during pregnancy

Daily exposure to $PM_{2.5}$ was estimated for each cohort participant during pregnancy. Gestational age was based on last menstrual period (LMP) and by a standardized physical examination to determine gestational age at birth (Capurro et al., 1978) because ultrasounds are not part of routine care in Mexico. If the physical examination assessment of gestational age differed by more than 3 weeks from the gestational age based on LMP, the physical exam was used instead of the gestational age determined by LMP. Daily $PM_{2.5}$ exposure was then estimated using a hybrid spatio-temporal model that incorporates Moderate Resolution Imaging Spectroradiometer (MODIS) satellite-derived Aerosol Optical Depth (AOD) measurements at a 1 × 1 km spatial resolution (Just et al., 2015). Remote sensing data were calibrated with municipal ground level monitors of $PM_{2.5}$, land use regression (LUR) variables, and meteorological data to yield estimates of daily residential $PM_{2.5}$ levels for each participant. The model was run using day-specific calibrations of AOD data calibrated against ground $PM_{2.5}$ measurements and LUR and meteorological variables (roadway density, temperature, relative humidity, planetary boundary layer and daily precipitation). Mixed effect models with spatial and temporal predictors and day-specific random effects were used to account for temporal variations in the $PM_{2.5}$–AOD relationship. For days without AOD data, the model was fit with a seasonal smooth function of latitude and longitude and time-varying average incorporating local monitoring. Model performance

was assessed using monitor-level leave one-out cross-validation; the model performed well with a cross-validated R^2 of 0.724. We calculated the average $PM_{2.5}$ over pregnancy and clinically defined trimesters (1st trimester: 1–13 weeks, 2nd trimester: 14–27 weeks, 3rd trimester: 28 weeks-delivery).

2.3. Edinburgh Postnatal Depression Scale

Mothers completed the Spanish version of the EPDS at a visit during the second or third trimester of pregnancy. Following birth, 815 mother-child dyads had at least 1 follow-up visit, of which 679 (83%) and 629 (77%) completed the EPDS at 1 and 6 months, respectively (Cox et al., 1987). The Spanish version of the EPDS is validated in Mexican populations (Oquendo et al., 2008; Ortega et al., 2001). The 10-item EPDS asks about symptoms in the past 7 days, including: “1: I have laughed and been able to see the funny side of things,” “2: I have looked forward with enjoyment to things,” “3: I have blamed myself unnecessarily when things went wrong,” “4: I have been anxious or worried for no good reason,” “5: I have felt scared or panicky for no very good reason,” “6: Things have been getting on top of me,” “7: I have been so unhappy that I have had difficulty sleeping,” “8: I have felt sad or miserable,” “9: I have been so unhappy that I have been crying,” and “10: The thought of harming myself has occurred to me.” Participants rated the severity or frequency of each item based on 4 levels scored from 0 (indicating the most favorable condition) to 3 (indicating the least favorable condition) for each item. Total scores can range from 0 to 30.

The EPDS scale was constructed as a unidimensional tool to screen for postpartum depression; however, multiple studies have demon strated that the EPDS identifies multiple dimensions of postpartum psychological functioning, specifically depression, anxiety and anhedonia (Hartley et al., 2014; Matthey et al., 2013; Phillips et al., 2009). As previously reported, the results from an exploratory factor analysis (EFA) and confirmatory factor analysis (CFA) indicated that a three factor model including depressive, anxiety, and anhedonia symptom subscales was the most optimal fit in our sample (Flom et al., 2018). Item 10 (“thought of self-harming”) was omitted from the subscale analysis given the rare frequency of endorsement (0.3% for quite often, 1.8% for sometimes and 2.1% for not very often). The anhedonia subscale includes items 1 and 2; the anxiety subscale includes items 4, 5 and 6; and the depression subscale includes items 7, 8, and 9.

2.4. Covariates

Mother’s age and educational attainment were ascertained at enrollment. The Crisis in Family Systems-Revised (CRISYS) survey, validated in Spanish (Berry et al., 2006), was administered by a trained psychologist during the second or third trimester of pregnancy. The CRISYS survey assesses life events across 11 domains: financial, legal, career, relationship, home safety, neighborhood safety, medical issues (self and others), home, prejudice and authority. Participants rated life events in the past 6 months as positive, negative or neutral. Domains with one or more negative life event were summed into a negative life event (NLE) domain score, with higher scores indicating greater stress. Only 1 mother reported smoking in pregnancy in our current sample; therefore, prenatal exposure to environmental tobacco smoke was included in the models and was defined as report of

any smoker in the home during the second or third trimester of pregnancy. Birth season was defined according to weather patterns in Mexico City as dry/cold (January-February; November-December), dry/warm (March-April) and rainy (May-October).

2.5. Statistical analysis

Analyses were conducted on the 509 mothers with complete data (EPDS scores during pregnancy and at 1 and 6 months, and all covariates). Probable PPD (hereafter PPD) was defined as a dichotomous measure using EPDS scores at a clinically-relevant cutoff (EPDS score ≥ 13) (Sit and Wisner, 2009). The association between mean $PM_{2.5}$ averaged over the full gestational period and PPD was analyzed using the modified Poisson regression with robust error variance approach of Zou (2004), which provides estimates of relative risk. Due to evidence of overdispersion, negative binomial regression was used to examine the association between $PM_{2.5}$ and EPDS subscale scores. Models were adjusted for maternal age at enrollment, exposure to environmental tobacco smoke (ETS) in pregnancy (yes/no based on the presence of at least one smoker in the household at any point in pregnancy), maternal education, birth season, gestational age and prenatal NLE score. In sensitivity analyses, models were also adjusted for average $PM_{2.5}$ in the first year of life, restricted to mothers with full-term pregnancies (gestational age ≥ 37 weeks), and run using different cutoffs for PPD (EPDS ≥ 10 and EPDS ≥ 15). Analyses were performed in RStudio version 1.1.383.

3. Results

3.1. Sample characteristics

Table 1 shows the distribution of exposure, outcomes, and covariates across our sample. The majority of participants (76%) had a high school education or less, and 36% were exposed to environmental tobacco smoke during pregnancy. The median $PM_{2.5}$ exposure during pregnancy was 22.9 $\mu g/m^3$ (IQR: 3.8 $\mu g/m^3$) and 18% of participants were classified as likely suffering from PPD (EPDS scores ≥ 13) at the 1 and 6 months postpartum visits. At 6 months, EPDS subscale scores were highest for anxiety, followed by anhedonia and depression.

3.2. $PM_{2.5}$ in pregnancy and PPD at 6 months

Table 2 presents the fully-adjusted models for average $PM_{2.5}$ over pregnancy predicting PPD at 1 and 6 months. While $PM_{2.5}$ was not associated with PPD at 1 month (RR [95% CI], 0.85 [0.61, 1.19]), a 5-$\mu g/m^3$ increase in average $PM_{2.5}$ in pregnancy was associated with 1.59 (95% CI: 1.11, 2.28) times the risk of PPD at 6 months. Results at 6 months remained unchanged after inclusion of the average $PM_{2.5}$ levels during the first year of life (RR [95% CI], 1.60 [1.10, 2.33]), and exposure during this period was not independently associated with PPD at 6 months (RR [95% CI] for a 5-$\mu g/m^3$ change in average $PM_{2.5}$ in the 1st year postpartum, 0.98 [0.63, 1.51]).

Based on this result, we additionally examined 6-month PPD subtypes according to the presence of depression at 1 month postpartum. As shown in Table 3, a 5-$\mu g/m^3$ increase in $PM_{2.5}$ exposure during pregnancy was associated with 2.58 (95% CI: 1.40, 4.73) times the

risk of late-onset PPD at 6 months, while no association was observed for "chronic" PPD (PPD at 1 and 6 months: RR [95% CI], 1.06 [0.63, 1.76]). When we used depression during pregnancy (EPDS ≥ 13 in pregnancy) along with 1-month PPD to categorize the 6-month PPD onset groups, the strongest association between $PM_{2.5}$ exposure during pregnancy and PPD at 6 months was observed among women without depression both during pregnancy and at 1 month postpartum (RR [95% CI], 3.12 [1.67, 5.86]) (Supplementary Table 1).

3.3. $PM_{2.5}$ and depression subtypes at 6 months

Fig. 1 shows results from negative binomial regression models examining associations between $PM_{2.5}$ exposure over pregnancy and EPDS subscale scores at 6 months. With an increase in average $PM_{2.5}$ over pregnancy (5-μg/m^3 change), we observed an increase in subscale scores for anhedonia (IRR [95% CI]: 1.28 [1.02, 1.60]) and depression (IRR [95% CI]: 1.29 [1.01, 1.64]), while no association between $PM_{2.5}$ and anxiety scores was observed (IRR [95% CI]: 1.04 [0.89, 1.22]). No associations were observed between $PM_{2.5}$ in pregnancy and EPDS subtypes at 1 month ($p > 0.40$ for anhedonia, anxiety, and depression; data not shown).

3.4. Sensitivity analyses

Results for PPD and EPDS subscales were unchanged after additional adjustment for average $PM_{2.5}$ in the 1st year postpartum and in the subset of mothers with full-term pregnancies (gestational age ≥ 37 weeks) (see Supplementary Materials). Results were also robust to changes in the PPD cutoff to EPDS ≥ 10 or EPDS ≥ 15 (Supplementary Materials).

4. Discussion

We leveraged data from PROGRESS, an ongoing Mexico-based cohort, to examine the association between exposure to fine particulate matter during pregnancy and postpartum psychological functioning. Our findings suggest that at 6 months postpartum, a 5-μg/m^3 increase in average $PM_{2.5}$ exposure during pregnancy (a unit change well within the range of average $PM_{2.5}$ exposures during pregnancy in our study of 16.4 μg/m to –29.3 μg/m^3) is associated with an 83% increase in risk of PPD and a 158% increase in risk of late-onset PPD (PPD at 6 months with no PPD at 1 month). EPDS subscale analyses also revealed that higher $PM_{2.5}$ in pregnancy was associated with increased symptoms of anhedonia and depression at 6 months. $PM_{2.5}$ exposure in the first year postpartum was not independently associated with PPD or subscale scores, and adjustment for postnatal exposure did not mitigate the association with prenatal $PM_{2.5}$ exposure. In sum, our findings suggest that pregnancy is a vulnerable window for the detrimental impacts of $PM_{2.5}$ exposure on postpartum depression and anhedonia in Mexican women, which may not occur until several months following birth.

The current results are in line with other published work in non-pregnant populations. A recent analysis in the Nurses' Health Study found an association between higher $PM_{2.5}$, estimated using a spatio-temporal model, in the past year and an increased hazard of depression diagnosis and antidepressant use (Kioumourtzoglou et al., 2017). In a combined analysis of data from 6 low- and middle-income countries that included Mexico, a 10 μg/m^3

increase in 3-year average $PM_{2.5}$ was associated with increased prevalence of depressive symptoms in the past 12 months (Lin et al., 2017). A recent meta-analysis of observational studies reported a significant increase in the pooled odds for the association between long-term exposure to $PM_{2.5}$, defined as exposure time greater than 1 year, and depression and depressive symptoms (Zeng et al., 2019).

Our findings indicate that particulate matter exposure during pregnancy may not influence PPD until several months postpartum. Historically, PPD has been defined as the onset of major depression within the first 4 weeks after childbirth (Pearlstein et al., 2009), but recent evidence suggests that women are susceptible to PPD far beyond that window, with risks extending well into the first year postpartum (Stewart and Vigod, 2016). Indeed, half of women in our study with PPD at 6 months postpartum did not meet criteria for PPD at 1 month. PPD onset subtypes are characterized by different risk factors, with a study by Wikman et al. (2019) showing that Swedish women with later onset of PPD (at 6 months) were more likely to experience sleep deficits, lack of support from partners, and issues with bonding. Late onset PPD may also be triggered by weaning from breastfeeding and the subsequent recommencement of menstrual cycles, which could result in changes in the levels of oxytocin and other hormones that affect mood (Burke et al., 2019). While beyond the scope of the current analysis, we plan to investigate how the relationship between $PM_{2.5}$ and PPD risk may be modified by maternal factors such as stress, social support, and breastfeeding duration.

While the etiology of PPD is unclear, a proposed mechanism involves the dysregulation of the maternal hypothalamic-pituitary-adrenal (HPA) axis (Jolley et al., 2007). The maternal neuroendocrine system undergoes dramatic changes during pregnancy, largely due to the endocrine effects of the developing placenta (Duthie and Reynolds, 2013; Glynn et al., 2013). Disruption of normal HPA axis activity during pregnancy, possibly through stress-induced elevations in maternal cortisol levels, may extend the postpartum HPA refractory period, thereby increasing risk of depression (Glynn et al., 2013). Given that fine particulate matter has been shown to be associated with alterations in cortisol and other HPA axis hormones in studies in adults (Li et al., 2017; Niu et al., 2018), future studies should investigate the roles of cortisol and HPA axis functioning in the etiology of $PM_{2.5}$-induced PPD.

Mechanistic data suggest that inflammation may also contribute to the impact of fine particulate matter on depression. Mice exposed to $PM_{2.5}$ for 10 months had higher upregulation of pro-inflammatory cytokines in the hippocampus—which are thought to play an important role in the pathophysiology of depression (Raison et al., 2006)—and were more likely to exhibit depressive-like responses when compared to mice who received filtered air (Fonken et al., 2011). Another study in mice also found that 3-month exposure to PM triggered depressive-like response in mice and increased pro-inflammatory cytokine production in the brain (Liu et al., 2018). It is important to note that these studies were conducted using male mice only; thus, findings may not be directly translatable to females, particularly during the antepartum and postpartum periods. Additional studies in female mice and in human cohorts are warranted.

Consistent with our group's previous study in Boston (Sheffield et al., 2018), particulate matter exposure during pregnancy was associated with increased postpartum symptoms of anhedonia. Anhedonia, or the reduction in the ability to experience pleasure (i.e., lack of positive affect) (Winer et al., 2019), may arise, in part, from inflammatory processes. For example, increased inflammation has been shown to induce symptoms of anhedonia in animal models (De La Garza, 2005; Yirmiya, 1996) and randomized controlled trials of inflammatory challenge (Eisenberger et al., 2010), with effects most pronounced among women (Moieni et al., 2019). Future research should explore whether inflammation during pregnancy, particularly through fine particulate matter exposure, may be a contributing factor to the development of anhedonia after birth.

There are two notable differences between our group's previous and current studies on $PM_{2.5}$ exposure and postpartum psychological functioning. First, our previous study in Boston did not find that $PM_{2.5}$ exposure in mid-pregnancy was associated with depression and anhedonia among the subpopulation of Hispanic women (Sheffield et al., 2018). However, the population of Hispanic women in our Boston study was largely born in the United States, with few women born in Mexico or of Mexican origin, while the cohort in the current study was entirely Mexican-born. Second, our previous study did not find that $PM_{2.5}$ averaged over the entire pregnancy was associated with psychological functioning (Sheffield et al., 2018). This discrepancy may be attributable to the differences in $PM_{2.5}$ exposure levels: the median $PM_{2.5}$ exposure during pregnancy was markedly lower in Boston compared to Mexico City (16.5 $\mu g/m^3$ vs. 22.9 $\mu g/m^3$), which may explain the stronger effects observed in the current study. Nonetheless, findings from the two studies together lend support to the link between air pollution exposure during pregnancy and postpartum psychological functioning, which may vary according to the level of $PM_{2.5}$ exposure and across sociodemographic groups.

Our study had several strengths, including a well-characterized, longitudinal cohort with a large sample size; the high spatial resolution of the $PM_{2.5}$ estimates; and the examination of different aspects of postpartum psychological functioning (depression, anxiety, and anhedonia) using EPDS subscale score loadings previously validated in our sample (Flom et al., 2018). We also acknowledge some limitations. The $PM_{2.5}$ exposure estimates from the spatiotemporal model were based on the address of the maternal residence, which do not account for exposures when the women were away from their homes and also do not take into account $PM_{2.5}$ exposures from indoor sources. Notably, adjustment for environmental tobacco smoke, a major contributor to indoor air pollution, did not mitigate the observed effects of ambient $PM_{2.5}$. Further, we do not have information on other air pollutants that may co-occur with $PM_{2.5}$ that could potentially confound our associations. While we adjusted for several other variables that may confound the association between $PM_{2.5}$ and PPD, including maternal age, maternal education, and stress, we cannot dismiss the possibility that there are unknown confounders that may explain our associations. Future studies should be designed to better disentangle the effects of potential confounders, such as proximity to roadways or traffic density, on PPD through indirect pathways involving noise pollution and/or sleep deficits. We do not have data on previous PPD or mental health status, an important predictor of future PPD, in our participants. The original eligibility criteria for inclusion in the study, including access to a telephone, might have led to the exclusion

of populations particularly at risk for depressive outcomes. Finally, while the homogenous makeup of the PROGRESS cohort provides excellent internal validity, our findings may not be generalizable to non-Mexican populations.

In conclusion, we found that among women in Mexico City, increased particulate matter exposure during pregnancy was a risk factor for PPD and symptoms of depression and anhedonia at 6 months postpartum. Our findings add to the growing body of literature implicating air pollution exposure in neuropsychological dysfunction in mothers and in other populations. Future studies should examine mechanisms of inflammation and HPA axis dysregulation that may underlie these associations.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgements

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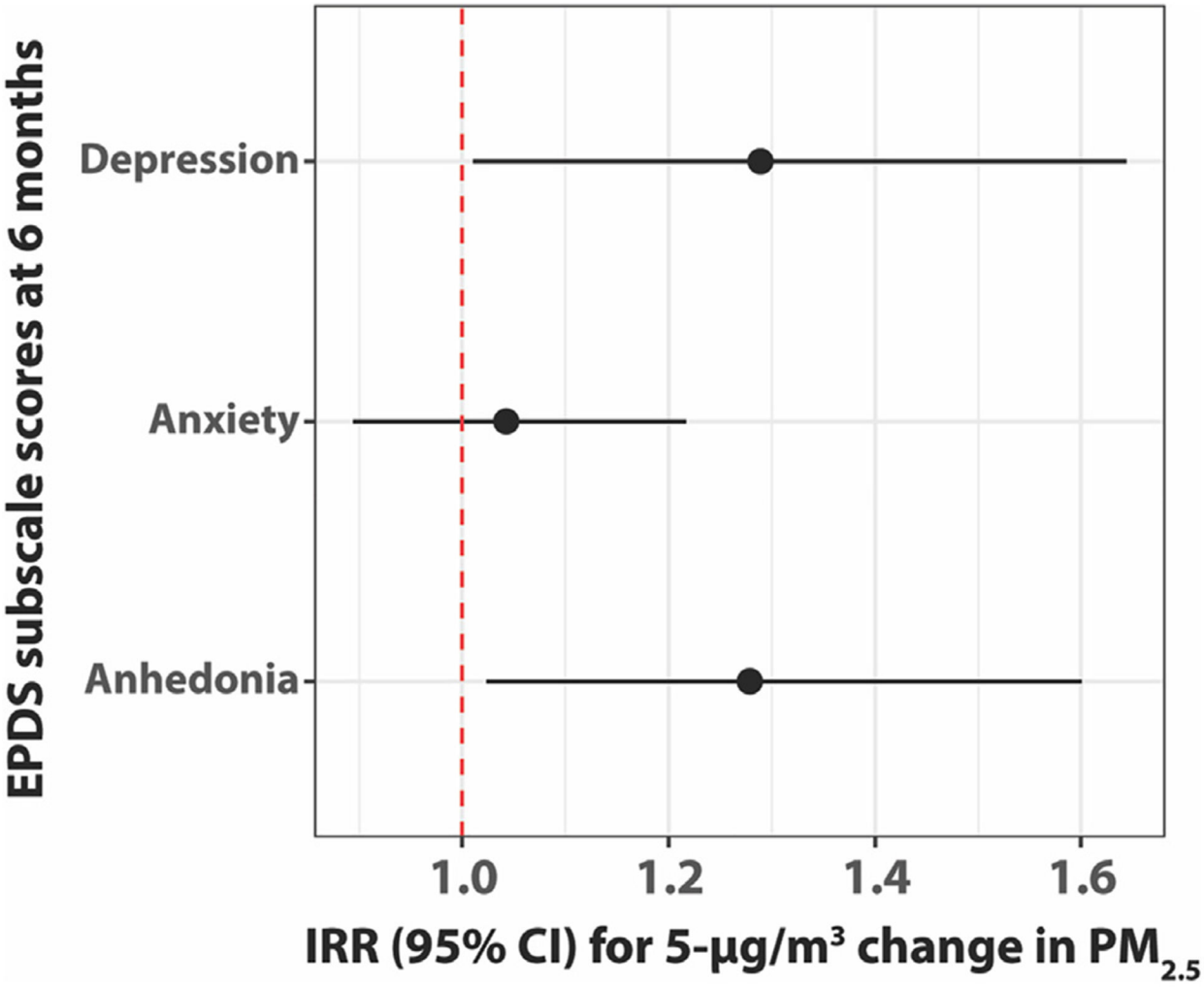

Fig. 1.

Negative binomial regression models for average $PM_{2.5}$ exposure during pregnancy predicting EPDS subscale scores at 6 months postpartum (N = 509). Models adjusted for maternal age at enrollment, tobacco exposure during pregnancy, maternal education, birth season, gestational age, and NLE score during pregnancy.

Table 1

Descriptive characteristics (N = 509).

Characteristics	Level/Unit	Median (IQR) or N (%)
Demographic		
Maternal age at enrollment	Years	27.4 (23.3, 31.7)
ETS exposure in pregnancy[a]	Yes	180 (35%)
Maternal education	< High school	211 (41%)
	High School	178 (35%)
	> High School	120 (24%)
Birth season	Dry/cold	188 (37%)
	Dry/warm	103 (20%)
	Rainy	218 (43%)
Gestational age	Weeks	39 (38, 39)
NLEs during pregnancy[b]	Score	3 (2, 5)
Air pollution		
Average $PM_{2.5}$ in pregnancy	$\mu g/m^3$	22.9 (20.6, 24.4)
Average $PM_{2.5}$ in 1st year postpartum	$\mu g/m^3$	22.8 (20.8, 24.0)
Depression symptoms		
EPDS during pregnancy, total	Score	8 (4, 13)
EPDS at 1 month, total	Score	6(2, 11)
EPDS at 6 months, total	Score	5 (2, 10)
Anhedonia subscale	Score	0 (0, 2)
Anxiety subscale	Score	2 (1, 5)
Depression subscale	Score	1 (0, 4)
Probable depression, pregnancy	EPDS ≥ 13	136 (27%)
Probable PPD, 1 month	EPDS ≥ 13	93 (18%)
Probable PPD, 6 months	EPDS ≥ 13	90 (18%)

[a] Presence of at least one smoker in the household during pregnancy.

[b] Sum of negative life events during pregnancy assessed by the CRISYS survey.

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Table 2

Relative risks[a] of PPD (EPDS ≥ 13) at 1 and 6 months postpartum (N = 509).

Variable	Level/unit	PPD, 1 month RR (95% CI)	P	PPD, 6 months RR (95% CI)	P
$PM_{2.5}$ in pregnancy	5-μg/m^3 change	0.85 (0.61, 1.19)	0.35	1.59 (1.11, 2.28)	0.01
Maternal age at enrollment	1-yr change	0.96 (0.93, 1.00)	0.04	0.99 (0.96, 1.02)	0.51
Tobacco exposure in pregnancy	Yes	0.78 (0.53, 1.15)	0.22	1.03 (0.71, 1.49)	0.88
Maternal education	< High School	*Ref.*	-	*Ref.*	-
	High School	0.95 (0.63, 1.44)	0.82	0.85 (0.56, 1.28)	0.42
	> High School	0.61 (0.36, 1.02)	0.06	0.57 (0.34, 0.93)	0.03
Birth season	Dry/cold	*Ref.*	-	*Ref.*	-
	Dry/warm	1.72 (1.07, 2.76)	0.03	1.17 (0.71, 1.95)	0.54
	Rainy	1.18 (0.76, 1.81)	0.46	1.00 (0.66, 1.53)	0.99
Gestational age	1-week change	0.95 (0.83, 1.08)	0.42	0.87 (0.78, 0.98)	0.02
Pregnancy NLE score	1-event change	1.21 (1.13, 1.30)	9×10^{-8}	1.21 (1.12, 1.31)	1×10^{-6}

[a]Estimated from modified Poisson regression models with robust error variance, adjusted for all variables listed in table.

Table 3

Relative risks[a] of PPD (EPDS ≥ 13) onset subtypes at 6 months postpartum for 5-μg/m^3 change in $PM_{2.5}$ exposure during pregnancy (N = 509).

6-month PPD subtype	No. of cases	RR (95% CI)	P
Late postpartum onset (no PPD at 1 month + PPD at 6 months)	45	2.58 (1.40, 4.73)	0.002
Chronic postpartum (PPD at 1 and 6 months)	45	1.06 (0.63, 1.76)	0.83

[a]Estimated from modified Poisson regression models with robust error variance, adjusted for maternal age at enrollment, tobacco exposure during pregnancy, maternal education, birth season, gestational age, and NLE score during pregnancy.