The association between postpartum depression and air pollution during pregnancy and postpartum period: a national population study in Taiwan

Ping Shih, Chih-Da Wu, Tung-liang Chiang, Pau-Chung Chen, Ta-Chen Su, Tsun-Jen Cheng, Yi-Hua Chen and Yue Leon Guo

1 Department of Environmental and Occupational Medicine, National Taiwan University (NTU) and NTU Hospital, No.1, Changde St., Zhongzheng Dist., Taipei 100229, Taiwan
2 Institute of Environmental and Occupational Health Sciences, College of Public Health, National Taiwan University, No. 17, Xuzhou Rd., Zhongzheng Dist., Taipei 100025, Taiwan
3 Department of Geomatics, National Cheng Kung University, Tainan, Taiwan
4 National Institute of Environmental Health Sciences, National Health Research Institutes, Miaoli, Taiwan
5 Institute of Health Policy and Management, College of Public Health, National Taiwan University, Taipei, Taiwan
6 School of Public Health, College of Public Health, Taipei Medical University, Taipei, Taiwan
7 Division of Cardiology, Department of Internal Medicine, National Taiwan University Hospital, Taipei, Taiwan

* Author to whom any correspondence should be addressed.

E-mail: leonguo@ntu.edu.tw

Keywords: postpartum depression, pregnancy, postpartum exposure, air pollution, NO₂, traffic-related air pollutant

Supplementary material for this article is available online

Abstract

Epidemiological evidence shows the association between air pollutants and several mental health outcomes, including depression, sleep disturbance, dementia, childhood neurodevelopment and suicide. Pregnant women are believed to be more susceptible and vulnerable to environmental pollutants, and postpartum depression (PPD) is a prevalent debilitating mental disorder. However, data on the effects of exposure to air pollution during pregnancy and postpartum period on the risk of PPD remain limited. This study aimed to evaluate the association between exposure to ambient air pollution during pregnancy and postpartum period and the incidence of PPD. The Taiwan Birth Cohort Study recruited representative 12% of all newborn in 2005 and their mothers by two-stage stratified sampling, including 21 248 mother–infant pairs. The occurrence of PPD was assessed by a self-reported questionnaire. Exposure to air pollutants during pregnancy and postpartum period was estimated using hybrid kriging/land-use regression (LUR) and integrated LUR-machine learning model based on data from the air monitoring stations. Logistic regression was then conducted to determine adjusted odds ratios (aORs) of PPD in relation to air pollutants. A total of 21 188 women were included in the final analysis, among whom 3,648 (17.2%) developed PPD within 6 months postpartum. The occurrence of PPD was significantly related to exposure to ambient concentrations of nitrogen dioxide (NO₂) during first trimester after adjustment [aOR: 1.081 per interquartile range (10.67 ppb), 95% confidence interval: 1.003, 1.165], but not to particulate matter ≤2.5 μm in diameter or carbon monoxide. Exposure to ambient NO₂ during early pregnancy was significantly related to the occurrence of PPD among the women investigated in this population-based study.

1. Introduction

Postpartum depression (PPD) is a common and serious mental health problem among women who have given birth. Despite variations in definition among population studies, estimates of prevalence in different countries range from 13% to 19% (O’Hara and McCabe 2013). PPD impairs maternal social and occupational functioning, and distresses the whole family. Furthermore, mother–infant interaction can be affected, resulting in numerous negative consequences on the physical health and
cognitive, linguistic, emotional, social, and behavioral development of the offspring (Boath et al 1998, McMahon et al 2006, Norhayati et al 2015, Slomian et al 2019).

The biological etiology underlying PPD is not fully understood. Most scientists believe that peripartal hormone changes may be involved, including steroid hormone, glucocorticoid, oxytocin, etc (Schiller et al 2015, Brummelte and Galea 2016). A larger number of studies exist investigating neuroendocrine changes, neuroinflammation, neurotransmitter alterations, circuit dysfunction, and also the involvement of genetics and epigenetics in the potential pathophysiological mechanisms contributing to PPD (Payne and Maguire 2019). These diverse mechanisms are also highly interrelated, and make the intricate etiology of PPD challenging.

Ambient air pollution has been reported to increase the risk of central nervous system disease and neuropsychiatric disorders, including depression, in animal and human studies (Block and Calderón-Garciduèñas 2009, Fonken et al 2011, Hahad et al 2020). A meta-analysis reported an increased risk of depression as a result of long-term exposure to particulate matter \( \leq 2.5 \) \( \mu \)m in diameter (PM\(_{2.5}\)) and short-term exposure to particulate matter \( \leq 10 \) \( \mu \)m in diameter (PM\(_{10}\)), nitrogen dioxide (NO\(_2\)), sulfur dioxide (SO\(_2\)), and carbon monoxide (CO) (Zeng et al 2019). Another systematic review included studies published till August 2019 found only short-time NO\(_2\) exposure had significant result, but not PM\(_{2.5}\), PM\(_{10}\) or SO\(_2\) (Fan et al 2020). In addition, a review took publications before September 2020 into consideration and found both long-term of PM\(_{2.5}\) and PM\(_{10}\), and also short-term of PM\(_{2.5}\) exposure increased the risk of depression (Liu et al 2021). Therefore, these may indicate that short-term of PM\(_{2.5}\), NO\(_2\) and CO, as well as long-term of particulate matter air pollution exposure have adverse effects on mental health.

With the dynamic physiologic alterations in their bodies (Soma-Pillay et al 2016), women in pregnancy and in postpartum period are vulnerable to neuropsychiatric disorders, including PPD. However, to our best knowledge, only two studies have examined the relationships between PPD and air pollutants. A U.S. pregnancy cohort study found that higher PM\(_{2.5}\) exposure in the second trimester was associated with increased total Edinburgh Postnatal Depression Scale (EPDS) scores and depressive symptom subscale scores among Black women (Sheffield et al 2018). A study conducted in Mexico revealed that an increase in average PM\(_{2.5}\) exposure during pregnancy was associated with an increased risk of PPD at 6 months (Niedzwiecki et al 2020). In these two studies, only PM\(_{2.5}\) was considered, but not other air pollutants.

Therefore, we assume that exposure to air pollution may contribute to PPD, although there is not much research to draw the conclusion. To exam the hypothesis, we used NO\(_2\) and CO as surrogates for traffic-related air pollutants (TRAP) (Health Effects Institute 2010). The other important category of air pollutants are particulates. PM\(_{2.5}\) is from multiple sources, has been documented to cause oxidative stress, systemic inflammation, and multiple health outcomes (Araujo 2011, Lu et al 2015). This current study examined a representative sample of pregnant mothers who gave birth in 2005, and investigated the relationship between PPD occurrence and maternal exposure to PM\(_{2.5}\), NO\(_2\), and CO during pregnancy and postpartum period, to determine the potential role of air pollutants in maternal PPD.

2. Methods

2.1. Study population and sampling

The Taiwan Birth Cohort Study (TBCS) is a national prospective longitudinal cohort study. A two-stage stratified random sampling method was performed on the Taiwan National Birth Registration data from 2005 to identify representative mother–infant pairs involved in the TBCS. First, 12 strata were defined according to administrative division (four strata: district, city, urban township, and rural township) and total fertility rate (three strata: low, medium, and high). All 369 townships in Taiwan were included, from which 89 were randomly selected as the primary sampling units. Second, newborns were proportionally selected from each primary sampling unit based on the principle of probability proportional to size. A total of 24,200 samples were obtained from the birth registry, which accounted for approximately 12% of all deliveries in Taiwan in 2005. Interviewers visited the selected candidates at their residence, and informed consent was obtained from the parents or main caregivers of the infant.

2.2. Questionnaire and dependent outcome

Structured questionnaires were administered by approximately 80 well-trained interviewers when the children were aged 6 months. Information regarding maternal conditions during pregnancy, infant birth outcomes, infant health conditions, parental socioeconomic status, and other demographics were obtained. Those who were unable to complete the questionnaire were excluded from the cohort. A total of 21,248 mother–infant pairs were included. In the survey, mothers were asked if they had experienced PPD. Answering ‘yes’ to this question was considered a positive response to the dependent variable. Mothers with missing or unsure answers to the question regarding PPD \( n = 43 \) were excluded in the data selection process. In addition, whether or not the mothers have the disability identification of chronic mental health conditions based on Physically and Mentally Disabled Citizens Protection Act in Taiwan, or who met the criteria for a severe illness of chronic mental disorders as defined by the Taiwan's National
Health Insurance (NHI) Administration, were further excluded \((n = 17)\). Therefore, 21,188 participants included in the final analysis (figure 1).

### 2.3. Exposure assessment

In this investigation, PPD was compared to ambient exposure to PM\(_{2.5}\), NO\(_2\), and CO. Participants’ exposed levels to PM\(_{2.5}\) and NO\(_2\) were estimated based on a hybrid kriging/land-use regression (LUR) approach (Wu et al. 2018, Chen et al. 2020). By integrating kriging-interpolated air pollution estimations with LUR models including culture-specific sources as potential predictors, this hybrid approach combines the strengths of both Krigeing and LUR methods and represents a high percentage of explanatory power with \(R^2\) of 0.88 for PM\(_{2.5}\) and 0.90 for NO\(_2\), respectively. In addition, LUR-based extreme gradient boosting algorithm model was conducted to the prediction of spatial-temporal distribution of CO, and the \(R^2\) value was 0.85 (Wong et al. 2021). Each participant’s home address was geocoded to the township level and linked to the spatial mean of township to determine their exposure to air pollution. Exposure periods were defined as (a) first trimester (from conception to 12th week of pregnancy), (b) second trimester (13th to 26th week of pregnancy), (c) third trimester (from 27th week of pregnancy to childbirth), and (d) postpartum 3 months (childbirth to 3 months postpartum). The date of conception was estimated by subtracting the infant’s gestational age from the birth date, and the gestational age was determined based on the mother’s self-response in the first-wave of cohort study. Mothers got the information of gestational age with ultrasound performed by obstetricians during the prenatal health check-ups.

### 2.4. Statistical analysis

The binary outcome variable was the occurrence of PPD, and the major predictor variables were the estimated exposure levels to ambient air pollutants including PM\(_{2.5}\), CO and NO\(_2\). Potential covariates were compared between mothers with and without PPD, including the sex, birth order, gestational age, birth weight of infant, tocolysis during pregnancy, delivery method, delivery season, breastfeeding, infant health condition, maternal age at childbirth, maternal educational level, pre-pregnancy body mass index, perinatal smoking or passive smoking history, perinatal alcohol consumption, marital status, urban living, annual household income, and family support. The urban residence was defined from seven levels of townships, with level 1 as the most urban and level 7 as the least, developed by Academia Sinica, Taiwan (Hou et al. 2008). We categorized the townships in levels 1–2 as urban and those in levels 3–7 as rural. Still, ambient temperature in the same period as the air pollutant was further adjusted, considering the synergic association between temperature and air pollutants (Kalisa et al. 2018) and also the potential health effect of temperature on depression (Chen et al. 2019). Ambient temperature was estimated using an ordinary kriging method, which had been described in detail (Shih et al. 2020). Data on these other potential confounders were collected during face-to-face interviews for the survey, performed when the infant was aged 6 months. The chi-square test was used to compare categorical data and the \(t\) test was used to compare continuous data. Significant covariates were used to adjust further analyses for confounding factors. Logistic regression was employed for each air

---

**Figure 1.** Data selection and linkage process.
pollutant in each period of pregnancy (first, second, or third trimester) and the first 3 months postpartum adjusting for confounders. The findings were reported as the odds ratio (OR) and 95% confidence intervals (95% CIs) of the occurrence of PPD for each 10 µg m\(^{-3}\) increments in PM\(_{2.5}\), 0.1 parts per million (ppm) in CO, 1 part per billion (ppb) in NO\(_2\), as well as per interquartile range increase in pollutant concentration. Further sensitivity analysis was performed using two-pollutant models. Statistical analyses were performed using the statistical software package JMP version 14.0 (SAS Institute Inc., Cary, NC, USA), and statistical significance was set at \(P < 0.05\) based on a two-sided calculation.

3. Results

In the 21,188 mothers included in the final analysis, 3,648 (17.2%) reportedly experienced PPD within 6 months postpartum. The demographic characteristics of the participants are summarized in table 1. Slightly more than half of the infants were boys, and no significant relationship was observed between the sex of infant and PPD. The following factors were significant covariates and were included in the further regression model, namely, first-born infants, tocolysis during pregnancy, cesarean section, premature or low-birthweight infants, delivery season, breastfeeding, poor health status in infant, maternal age, high education level, perinatal smoking or passive smoking exposure, perinatal alcohol consumption, other than married marital status, urban living, annual income of more than US$ 20,000 per year, and poor family support.

Table 2 presents the distribution of exposure to each air pollutant during pregnancy and postpartum period. In 2005, the air quality standards in Taiwan had not regulated the level of PM\(_{2.5}\), but the concentration of PM\(_{2.5}\) in our population was higher than WHO air quality guidelines of 10 µg m\(^{-3}\) annual average (World Health Organization 2006). The NO\(_2\) and CO concentrations were in compliance with Taiwan’s 50 ppm and 9 ppm regulatory standards at that time.

Table 3 details the correlations among these air pollutants by different periods. The level of CO strongly correlated with that of NO\(_2\). Table 4 presents the results of the logistic regression in determining the relationship between PPD and maternal exposure to air pollution according to different periods. Exposure to NO\(_2\) during first trimester was determined to be associated with PPD after adjusting for birth order of infant, tocolysis during pregnancy, delivery method, delivery season, premature and low-birthweight infant, breastfeeding, children general health status, mother’s age at childbirth, mother’s education level, maternal perinatal smoking, passive smoking and alcohol consumption, annual household income, marital relationship, urban living and family supports. Exposure to PM\(_{2.5}\) or CO was not related to PPD. After adjusting for ambient temperature, and also using two-pollutant models with PM\(_{2.5}\), average ambient NO\(_2\) level during first-trimester (aOR: 1.081 per 10.67 ppb, 95% CI: 1.003, 1.165) and within the first 3 months postpartum (aOR: 1.078 per 10.48 ppb, 95% CI: 1.002, 1.160) were significantly related to PPD. However, if we put these two periods together to compete for effects, only first-trimester NO\(_2\) remained statistically significant.

The final regression model including NO\(_2\) exposure during first trimester was showed in table 5. The following factors were associated with an increased risk of PPD, namely, first-born infants, tocolysis during pregnancy, cesarean section, delivery season in spring, breastfeeding, poor health status in infant, maternal age between 25 and 35, high education level, perinatal smoking, passive smoking exposure, perinatal alcohol consumption, other than married marital status, and poor family support. However, there was no significant results from the effect of prematurity or low-birthweight infant, urban living, ambient temperature, nor household income.

4. Discussion

To our knowledge, this is the first study employing a nationwide representative survey that has detected an association between NO\(_2\) exposure during pregnancy and the risk of PPD. Exposure to interquartile range, namely, 10.67 ppb higher in NO\(_2\) in the first trimester was associated with an 8% increase in the likelihood of PPD.

The main source of NO\(_2\) was traffic emission, thus it is viewed as the index of TRAP. These results accord with previous studies. A meta-analysis of seven studies revealed a significant association between short-term NO\(_2\) exposure and depression in general population (OR: 1.02, 95% CI: 1.00, 1.04 per 10 mg m\(^{-3}\) increase, \(I^2: 65.4\)% ) (Zeng et al 2019, Fan et al 2020). Regarding the long-term effect, Spanish and Korean studies reported that depressive disorder was related to NO\(_2\) exposure (Vert et al 2017, Shin et al 2018). However, a cohort study did not detect significant long-term effects of NO\(_2\) exposure in Germany, Norway, and Finland (Zijlma et al 2016). The difference may result from lower exposure to NO\(_2\) in European countries. These abovementioned studies have mainly investigated non-pregnant populations. One study conducted in Shanghai studied pregnant women and showed a significant relationship between NO\(_2\) exposure and maternal stress levels (Lin et al 2017). Therefore, our study is the first one clearly pointing out that TRAP exposure, especially NO\(_2\), during early pregnancy would increase the risk of postpartum mental illness in women.

In this investigation, PM\(_{2.5}\) was found unrelated to PPD occurrence, which was different from the conclusion of a previous study (Niedzwiecki et al 2020),
### Table 1. Demographic characteristics.

| Variables                                      | N = 21 188 | Mother with PPD | Mother without PPD | P-value |
|------------------------------------------------|------------|-----------------|--------------------|---------|
|                                                 |            | 3648 (17.2%)    | 17 540 (82.8%)     |         |
| Sex of infant                                   |            |                 |                    |         |
| Male                                           | 11 114     | 1873 (16.9%)    | 9241 (83.2%)       | 0.1398  |
| Female                                         | 10 074     | 1775 (17.6%)    | 8299 (84.2%)       |         |
| Birth order of infant                           |            |                 |                    | <.0001  |
| First                                          | 10 684     | 2183 (20.4%)    | 8501 (79.6%)       |         |
| Second and others                               | 10 504     | 1465 (14.0%)    | 9039 (86.1%)       |         |
| Tocolysis during pregnancy                      |            |                 |                    | <.0001  |
| Yes                                            | 5051       | 1158 (22.9%)    | 3893 (77.1%)       |         |
| No                                             | 16 137     | 2490 (15.4%)    | 13 647 (84.6%)     |         |
| Delivery method by Caesarean section            |            |                 |                    | 0.0012  |
| Yes                                            | 7278       | 1338 (18.4%)    | 5940 (81.6%)       |         |
| No                                             | 13 909     | 2310 (16.6%)    | 11 599 (83.4%)     |         |
| Premature infant (<37 weeks of gestation)      |            |                 |                    | 0.0321  |
| Yes                                            | 1777       | 339 (19.1%)     | 1438 (80.9%)       |         |
| No                                             | 19 411     | 3309 (17.1%)    | 16 102 (83.0%)     |         |
| Low-birthweight infant (<2500 g)               |            |                 |                    | <.0001  |
| Yes                                            | 1450       | 307 (21.2%)     | 1143 (78.8%)       |         |
| No                                             | 19 738     | 3341 (16.9%)    | 16 397 (83.1%)     |         |
| Delivery season                                 |            |                 |                    |         |
| Spring                                         | 5050       | 925 (18.3%)     | 4125 (81.7%)       | 0.0071  |
| Summer                                         | 5249       | 912 (17.4%)     | 4337 (82.6%)       | 0.1477  |
| Autumn                                         | 5462       | 925 (16.9%)     | 4537 (83.1%)       | 0.3932  |
| Winter                                         | 5427       | 886 (16.3%)     | 4541 (83.7%)       |         |
| Ever breastfeeding                              |            |                 |                    | <.0001  |
| Yes                                            | 17 421     | 3095 (17.8%)    | 14 326 (82.2%)     |         |
| No                                             | 3766       | 553 (14.7%)     | 3213 (85.3%)       |         |
| Children general health status                  |            |                 |                    | <.0001  |
| Fair                                           | 18 103     | 2918 (16.1%)    | 15 185 (83.9%)     |         |
| Poor                                           | 3084       | 730 (23.7%)     | 2354 (76.3%)       |         |
| Maternal age at childbirth                     |            |                 |                    |         |
| <25 years old                                  | 4167       | 646 (15.5%)     | 3521 (84.5%)       | 0.0001  |
| 25–35 years old                                | 14 332     | 2587 (18.1%)    | 11 745 (82.0%)     | 1       |
| >35 years old                                  | 2689       | 415 (15.4%)     | 2274 (84.6%)       | 0.0011  |
| Pre-pregnancy body mass index                  |            |                 |                    |         |
| <18.5 kg m$^{-2}$                               | 4281       | 733 (17.1%)     | 3548 (82.9%)       | 0.9333  |
| 18.5–27 kg m$^{-2}$                            | 15 655     | 2689 (17.2%)    | 12 966 (82.8%)     | 1       |
| ≥27 kg m$^{-2}$                                | 1147       | 217 (18.9%)     | 930 (81.1%)        | 0.1323  |
| Maternal education level                       |            |                 |                    | <.0001  |
| High school education or more                  | 9574       | 1871 (19.5%)    | 7703 (80.5%)       |         |
| Less than a high school education              | 11 579     | 1772 (15.3%)    | 9807 (84.7%)       |         |
| Maternal perinatal smoking                     |            |                 |                    | <.0001  |
| Yes                                            | 1467       | 364 (24.8%)     | 1103 (75.2%)       |         |
| No                                             | 19 708     | 3279 (16.6%)    | 16 429 (83.4%)     |         |
| Maternal perinatal passive smoking             |            |                 |                    | <.0001  |
| Yes                                            | 12 853     | 2341 (18.2%)    | 10 512 (81.8%)     |         |
| No                                             | 8318       | 1302 (15.7%)    | 7016 (84.4%)       |         |
| Maternal perinatal alcohol consumption         |            |                 |                    | <.0001  |
| Yes                                            | 2059       | 441 (21.4%)     | 1618 (78.6%)       |         |
| No                                             | 19 115     | 3201 (16.8%)    | 15 914 (83.3%)     |         |
| Marital relationship                           |            |                 |                    | <.0001  |
| Married                                        | 19 869     | 3351 (16.9%)    | 16 518 (83.1%)     |         |
| Divorce or separated or widowed or single      | 1319       | 297 (22.5%)     | 1022 (77.5%)       |         |
| Residential location                           |            |                 |                    | 0.0003  |
| Urban                                          | 9178       | 1678 (18.3%)    | 7500 (81.7%)       |         |
| Rural                                          | 12 010     | 1970 (16.4%)    | 10 040 (83.6%)     |         |

(Continued.)
Table 1. (Continued.)

| Variables | N = 21 188 | Mother with PPD | Mother without PPD | P-value |
|-----------|------------|-----------------|--------------------|---------|
| Household income | | | | |
| ≤20 000 USD per year | 8824 (41.7%) | 1428 (16.2%) | 7396 (83.8%) | 0.0007 |
| >20 000 USD per year | 12 364 (58.4%) | 2220 (18.0%) | 10 144 (82.0%) | |
| Family supports | | | | |
| Good | 13 276 (63.0%) | 1825 (13.8%) | 11 451 (86.3%) | <.0001 |
| Poor | 7804 (37.0%) | 1799 (23.1%) | 6005 (77.0%) | |

P-values were derived using chi-square test for categorical variables and t test for continuous variables, compared with mothers without PPD. PPD, postpartum depression.

Table 2. Distribution of air pollutants during pregnancy and postpartum period.

| | Mean ± SD | IQR | Minimum | First quartile | Median | Third quartile | Maximum |
|---|---|---|---|---|---|---|---|
| First trimester | | | | | | | |
| PM$_{2.5}$ (μg m$^{-3}$) | 35.33 ± 10.96 | 14.18 | 15.21 | 27.34 | 32.45 | 41.52 | 71.84 |
| CO (ppm) | 0.55 ± 0.19 | 0.26 | 0.15 | 0.41 | 0.53 | 0.67 | 1.12 |
| NO$_2$ (ppb) | 19.67 ± 7.34 | 10.67 | 3.41 | 14.00 | 18.82 | 24.66 | 40.71 |
| Second trimester | | | | | | | |
| PM$_{2.5}$ (μg m$^{-3}$) | 35.25 ± 10.72 | 13.50 | 14.81 | 27.51 | 32.36 | 41.01 | 71.50 |
| CO (ppm) | 0.54 ± 0.19 | 0.26 | 0.14 | 0.40 | 0.52 | 0.66 | 1.11 |
| NO$_2$ (ppb) | 19.29 ± 7.52 | 10.67 | 2.39 | 13.67 | 18.63 | 24.35 | 40.19 |
| Third trimester | | | | | | | |
| PM$_{2.5}$ (μg m$^{-3}$) | 35.54 ± 11.83 | 15.86 | 12.57 | 26.73 | 32.61 | 42.60 | 84.97 |
| CO (ppm) | 0.52 ± 0.20 | 0.27 | 0.13 | 0.38 | 0.50 | 0.64 | 1.12 |
| NO$_2$ (ppb) | 18.57 ± 7.70 | 10.64 | 2.16 | 12.72 | 17.89 | 23.35 | 41.29 |
| Postpartum 3 months | | | | | | | |
| PM$_{2.5}$ (μg m$^{-3}$) | 36.58 ± 12.33 | 15.41 | 13.63 | 27.51 | 34.18 | 42.92 | 83.65 |
| CO (ppm) | 0.52 ± 0.20 | 0.26 | 0.13 | 0.38 | 0.50 | 0.64 | 1.15 |
| NO$_2$ (ppb) | 18.54 ± 7.67 | 10.48 | 2.35 | 12.86 | 17.58 | 23.34 | 40.93 |

IQR, interquartile range; SD, standard deviation; ppb, parts per billion; ppm, parts per million; PM$_{2.5}$, particulate matter ≤ 2.5 μm

First trimester, from conception to 12th week of pregnancy, whereas the date of conception was estimated by subtracting the infant’s gestational age from the birth date; second trimester, 13th–26th week of pregnancy; third trimester, from 27th week of pregnancy to childbirth; postpartum 3 months, from childbirth to 3 months postpartum.

despite the higher concentration of exposure in our study. The hybrid kriging/LUR model used in our study showed high explanatory power with $R^2$ of 0.88 and root mean square error of 7.86 for PM$_{2.5}$ (Wu et al. 2018), suggesting reliable estimation of ambient PM$_{2.5}$. The possible explanation for the non-significant relationship may be caused by different components of PM$_{2.5}$ between countries. Particulate matter is emitted from a variety of source type, such as fuels combustion, industrial processes, road dust and sea salt or soil erosion. The concentration of PM could not present the actual activities in the areas, which would result in the discordant finding. Additionally, we did not consider the long-term effects of particulate matter on depression as in previous studies (Liu et al. 2021). Due to the small number of studies and inconsistent relationship between ambient PM$_{2.5}$ exposure and PPD, further research is warranted.

CO exposure was not statistically significant in the study. Previous systematic review reported a pooled estimate from three related surveys indicated that short-term exposure to CO was borderline associated with an increased likelihood of depression (OR = 1.01; 95% CI: 1.00, 1.01 per 0.1 ppm increase, $I^2$: 70.2%) (Zeng et al. 2019). Korean studies also reported long-term effect of CO exposure on depressive disorder (Shin et al. 2018). Though CO was also viewed as one of the indexes of TRAP, we inferred that the non-significant results in our study may be due to the relatively low ambient CO concentration.

PPD was inquired in 6 month postpartum mothers in TBCS in this investigation. The criteria used by psychiatrists in Taiwan to diagnose PPD in 2005 (year the study was conducted) were based on the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR) (American Psychiatric Association 2000). Major
depressive disorder (MDE) with postpartum onset was recognized in the DSM-IV-TR as PPD. Postpartum onset was defined as being within the 4 weeks following delivery. The essential feature of MDE is a period of at least 2 weeks during which an individual experience either a depressed mood or a loss of interest or pleasure in nearly all activities.

Taiwan's NHI program covers a postpartum checkup 4–6 weeks after childbirth, as well as two well-baby clinic visits before the age of 6 months in Taiwan. Therefore, accessibility to medical care was adequate for the mothers if PPD were to develop during the first 6 months after childbirth.

The prevalence of PPD in 6 month postpartum mothers was observed to be approximately 17.2% in our study. A 10.3% prevalence of PPD 6 weeks postpartum in Taiwan had been reported in 2001–2002 among 203 subjects by psychiatric specialists using the DSM-IV criteria (Teng et al 2005). Another prospective longitudinal study detected that 9.4% (22 out of 234) of mothers displayed postpartum MDEs in the 4th week postpartum (Lin et al 2019). These two investigations were conducted by experienced psychiatric teams. However, they suffered from high rates of refusal and/or low follow-up. It was possible that women with more severe PPD were less able to complete the survey, leading to underestimation of the prevalence. Investigations using self-report structured questionnaires, mainly the Taiwanese version of the EPDS, revealed a prevalence of 20% or higher (Heh 2001, Huang and Mathers 2001), which was slightly higher than the observed PPD in this current study. For the validation of the participating women’s PPD status, reported PPD was compared with their answers to the 36-Item Short Form Survey (SF-36), Taiwan Version (Lu 2003), which was included in TBCS survey at 6 months after childbirth. The four subscales of SF-36, i.e. vitality, social functioning, role-emotional and mental health were compared between mothers with and without PPD (supplementary table 1 (available online at stacks.iop.org/ERL/16/084021/mmedia)). Those mothers with reported PPD had lowered scores in the four subscales. This provided support for validity of the mothers’ reported PPD.

In this study, PPD was found related to primiparas, tocolysis, cesarean section, delivery season, breastfeeding, poor infant health, maternal age at childbirth, high education level, perinatal smoking, passive smoking, alcohol consumption, other than married marital status, and poor family support. Most of these factors represent stressors for mothers (Akincigil et al 2010, Xie et al 2010, Xu et al 2017). In Taiwanese, parents are more concerned about their first baby because of the numerous uncertainties in bearing a child, which causes psychological stress. A Japanese survey also reported higher depressive symptoms for primiparas (Iwata et al 2016). Furthermore, tocolysis reduces oxytocin (Ikeda et al 1984, Vrachnis et al 2011), and a relationship has been observed between low levels of oxytocin during pregnancy and PPD (Skrundz et al 2011, Moura et al 2016). The relationship between delivery season and PPD can be attributed to day length (Goyal et al 2018). The association between breastfeeding and PPD has been studied by several investigators. However, the causal direction and type of this relationship remain unclear (Pope and Mazmanian 2016). In our study, having breastfeeding was associated with an increased risk of PPD. Although breastfeeding might have certain protective effects against stress (Heinrichs et al 2002), the rates of mothers who breastfed or attitudes toward breastfeeding may differ between cultures and countries. Difficulties in breastfeeding may be a factor (Watkins et al 2011). Therefore, investigating the underlying mechanisms of breastfeeding would be valuable. Nicotine uptake from smoking may enhance the hypothalamic–pituitary–adrenal (HPA) axis response to stress, which increases the risk of depression development (Yu et al 2010, Reuter et al 2012). Furthermore, studies had noted maternal age was associated with the risk of PPD, (Aasheim et al 2012, Silverman et al 2017). Our findings differed from a study that reported that lower maternal education levels were related to higher risks of PPD (Goyal et al 2010). Studies in Korean and Chinese participants found that the association of depressive symptoms and educational status was complex, and the results may differ.

### Table 3. Spearman correlation between each pollutant in the same period (multivariate analysis).

| Period                | PM$_{2.5}$ | CO       | NO$_2$    |
|-----------------------|------------|----------|-----------|
| First trimester       | 1          | 0.4601   | 0.5519    |
| Second trimester      | 1          | 0.4410   | 0.5426    |
| Third trimester       | 1          | 0.4223   | 0.5132    |
| Postpartum 3 months   | 1          | 0.4159   | 0.5078    |

By Spearman's rank correlation coefficient, all correlation coefficients are statistically significant at $p < 0.001$.

First trimester, conception to 12th week of pregnancy, whereas the date of conception was estimated by subtracting the infant's gestational age from the birth date; second trimester, 13th–26th week of pregnancy; third trimester, from 27th week of pregnancy to childbirth; postpartum 3 months, from childbirth to 3 months postpartum.
in distinct sociocultural situations (Jo et al 2011, Gan et al 2012). Therefore, various factors affecting the risk of PPD that may be interrelated were closely bound up, and further research is warranted to clarify the intricacies of PPD development.

There are two potential mechanisms of how air pollution leads to PPD. First, literature has demonstrated endogenous nitric oxide (NO) would be associated with alterations in the HPA axis hormones (Mancuso et al 2010). During pregnancy, the endogenous NO metabolite level is increased (Owusu Darkwa et al 2018). Then maternal NO metabolite levels should decline to normal level after giving birth (Okutomi et al 1997). Studies have demonstrated that the pathogenesis of PPD is be related to the change of dysregulation of the maternal HPA axis (Jolley et al 2007, Glynn et al 2013). Therefore, it is possible that exposure to high levels of ambient NO₂ during pregnancy may cause relatively larger-scale disruption of HPA axis activity after childbirth. The effect may cause extended the postpartum HPA refractory period, thus increasing the risk of PPD. The other potential mechanism is that air pollution affects the central nerve system through several cellular and molecular pathways, which can engender diseases caused by neuroinflammation, disruption of the blood–brain barrier, oxidative stress, endothelial dysfunction, and neuronal damage (Calderón-Garcidueñas et al 2008, Block and Calderón-Garcidueñas 2009, Brockmeyer and D’Angiulli 2016). These neuropathophysiological effects would contribute to PPD (Payne and Maguire 2019). Air pollution may induce dopaminergic neurotoxicity, resulting in depression symptoms (Levesque et al 2011, Yolton et al 2019) in both laboratory animal and human studies. Therefore, it was possible that a delay effect or accumulative effect of exposure to air pollutants in early pregnancy may induce PPD occurrence because of neuroinflammation or neurotoxicity.

Our study has some limitations. First, PPD was based on a self-reported answer, and it was not a clinical diagnosis assessed by professionals in this questionnaire-based study. However, among those women with reported PPD, poorer score in the psychometric scores were found by SF-36. Therefore, such an assessment was still credible. Infant’s gestational age was also an important information in

---

Table 4. Relationship between exposure to air pollution in different periods and postpartum depression.

| Period         | Model A               | Model B               | Model C               |
|----------------|-----------------------|-----------------------|-----------------------|
|                | aOR (95%CI) per unit¹ | aOR (95%CI) per unit¹ | aOR (95%CI) per IQR² |
| First trimester|                       |                       |                       |
| PM₂.₅         | 1.026 (0.988–1.066)   | 1.027 (0.988–1.067)   | —                     |
| CO            | 1.020 (0.993–1.048)   | 1.020 (0.993–1.048)   | 1.015 (0.985–1.046)   |
| NO₂           | 1.008 (1.001–1.014)   | 1.008 (1.001–1.014)   | 1.007 (1.000–1.014)   |
| Second trimester|                      |                       |                       |
| PM₂.₅         | 0.998 (0.960–1.038)   | 0.996 (0.958–1.036)   | —                     |
| CO            | 1.004 (0.978–1.032)   | 1.005 (0.978–1.032)   | 1.007 (0.978–1.037)   |
| NO₂           | 1.004 (0.998–1.011)   | 1.004 (0.998–1.010)   | 1.006 (0.999–1.013)   |
| Third trimester|                       |                       |                       |
| PM₂.₅         | 0.995 (0.961–1.031)   | 0.999 (0.963–1.036)   | —                     |
| CO            | 1.000 (0.974–1.028)   | 1.000 (0.974–1.028)   | 1.001 (0.972–1.031)   |
| NO₂           | 1.004 (0.997–1.010)   | 1.004 (0.998–1.010)   | 1.005 (0.998–1.012)   |
| Postpartum 3 months|            |                       |                       |
| PM₂.₅         | 1.006 (0.972–1.041)   | 1.011 (0.977–1.047)   | —                     |
| CO            | 1.010 (0.984–1.037)   | 1.012 (0.986–1.039)   | 1.010 (0.981–1.040)   |
| NO₂           | 1.005 (0.999–1.012)   | 1.006 (1.000–1.013)   | 1.007 (1.000–1.014)   |
| 2.5 µg m⁻³            | 1.081 (1.003–1.165)   | 1.078 (1.002–1.160)   | —                     |

¹ p < 0.05; aOR, adjusted odds ratio; CI, confidence interval; PM₂.₅, particulate matter ≤ 2.5 µm.

Model A: adjusting for birth order of infant, tocolysis during pregnancy, delivery method, delivery season, premature and low-birthweight infant, breastfeeding, children general health status, mother’s ag at childbirth, mother’s education level, maternal perinatal smoking, passive smoking and alcohol consumption, annual household income, marital relationship, urban living, family supports, and one pollutant in a specific period.

Model B: model A plus ambient temperature in the same period; Model C: model B plus PM₂.₅ in the same period.

¹ ORs calculated with unit of 10 µg m⁻³ increments in PM₂.₅, 0.1 parts per million (ppm) in CO, 1 part per billion (ppb) in NO₂.

² ORs calculated with IQR of 14.18 µg m⁻³ increments in PM₂.₅, 0.26 ppm in CO, 10.67 ppb in NO₂ during first trimester; 13.5 µg m⁻³ increments in PM₂.₅, 0.26 ppm in CO, 10.67 ppb in NO₂ during second trimester; 15.86 µg m⁻³ increments in PM₂.₅, 0.27 ppm in CO, 10.64 ppb in NO₂ during third trimester; 15.41 µg m⁻³ increments in PM₂.₅, 0.26 ppm in CO, 10.48 ppb in NO₂ during postpartum 3 months.

First trimester, from conception to 12th week of pregnancy, whereas the date of conception was estimated by subtracting the infant’s gestational age from the birth date; second trimester, 13th–26th week of pregnancy; third trimester, from 27th week of pregnancy to childbirth; postpartum 3 months, from childbirth to 3 months postpartum.
Table 5. Relationship between potential risk factors and postpartum depression.

| Covariates                                                                 | aOR (95%CI) |
|----------------------------------------------------------------------------|-------------|
| Birth order of infant (First vs. Second and others)                        | 1.65 (1.53–1.78)*** |
| Tocolysis during pregnancy (Yes vs. No)                                   | 1.50 (1.38–1.63)*** |
| Delivery method by Caesarean section (Yes vs. No)                         | 1.09 (1.01–1.18)* |
| Premature infant (<37 weeks vs. ≥37 weeks of gestation)                   | 0.98 (0.84–1.14) |
| Low-birthweight infant (<2500 g vs. ≥2500 g)                              | 1.13 (0.96–1.32) |
| Delivery season                                                            |             |
| Spring vs. Winter                                                          | 1.16 (1.05–1.29)** |
| Summer vs. Winter                                                          | 1.03 (0.88–1.20) |
| Autumn vs. Winter                                                          | 0.99 (0.84–1.18) |
| Ever breastfeeding (Yes vs. No)                                            | 1.18 (1.07–1.31)** |
| Children general health status (Fair vs. Poor)                             | 0.65 (0.59–0.72)*** |
| Maternal age at childbirth                                                 |             |
| 25–35 vs. <25 years old                                                   | 1.23 (1.10–1.37)*** |
| >35 vs. <25 years old                                                      | 1.09 (0.94–1.27) |
| Maternal education level (≥ High school education vs. Others)             | 1.38 (1.26–1.50)*** |
| Maternal perinatal smoking (Yes vs. No)                                   | 1.56 (1.35–1.79)*** |
| Maternal perinatal passive smoking (Yes vs. No)                           | 1.10 (1.02–1.19)* |
| Maternal perinatal alcohol consumption (Yes vs. No)                       | 1.16 (1.03–1.30)* |
| Marital relationship (Married vs. Others)                                  | 0.82 (0.71–0.94)** |
| Residential location (Urban vs. Rural)                                    | 0.99 (0.90–1.08) |
| Household income (<20 000 USD per year vs. Others)                        | 0.96 (0.88–1.05) |
| Family supports (Good vs. Poor)                                           | 0.52 (0.48–0.56)*** |
| Ambient temperature during first trimester (Unit of 1 degree Celsius increments) | 1.00 (0.98–1.02) |
| PM2.5 exposure during first trimester (Unit of 14.18 µg m⁻³ increments)  | 1.01 (0.95–1.07) |
| NO₂ exposure during first trimester (Unit of 10.67 ppb increments)        | 1.08 (1.00–1.17)* |

The variables listed were included in the final logistic regression model, equal to Model C in the table 4.

* p < 0.05.
** p < 0.01.
*** p < 0.001; aOR, adjusted odds ratio; CI, confidence interval.

Our study, and it was reported by mothers at 6 months postpartum. In Taiwan, there were 99% of mothers giving birth in hospitals or clinics by obstetricians during recent 15 years. Before delivery, the gestational age would be measured by ultrasound during prenatal health check-ups. We therefore believed the bias would be accepted. Second, the calculated exposure to ambient air pollutants was not personal exposure, because indoor air pollutant levels and time–activity patterns were not considered. Applying personal environmental monitoring in such a large sample was impractical, especially among pregnant subjects. Still, earlier exposure to ambient exposure before pregnancy did not considered in our study, because of lacking previous living address data. Third, only the administrative district of each participant was provided because of ethical privacy considerations. Therefore, exposure was measured at the township level, which inevitably resulted in misclassification of exposure. Such misclassification may theoretically reduce the observed association. However, we observed a significant relationship between air exposure and the occurrence of PPD, which suggests that the actual effect may be stronger. Fourth, approximately 12.2% of mothers did not join the survey and the reason for rejection was not provided. Therefore, we do not know whether the rejection was related to PPD or not. Because the participation rate was relatively high, the influence of bias may be acceptable (Galea and Tracy 2007). Mothers with PPD may have been unable to join the study, which should not bias the exposure–outcome relationship, but could have caused misclassification, thus reducing the observed relationship toward the null hypothesis.

Our study has several strengths. First, the participants were representative of Taiwanese babies born in 2005 from a population-based cohort design, which minimized the uncertainty of random error when collecting detailed data on covariates. Second, the collection of exposure to air pollutants from the Taiwan Environmental Protection Administration was comprehensive and independent of the methods used to obtain the main outcome in our study. Third, the high-accessibility and low-copayment conditions of the NHI program allow people to habitually seek perinatal medical services, which should be associated with higher reliability of the dependent outcome when we approached mothers at approximately 6 months postpartum. Therefore, the assessment of PPD in our study was still reliable. Fourth, two major types of air pollutants, namely, TRAP and particulates were studied during different periods of pregnancy, which allowed for detection of potential effects from various pollutants during critical windows.
In conclusion, this investigation found that the occurrence of PPD was positively associated with maternal NO\textsubscript{2} exposure during early pregnancy in this representative birth cohort from 2005. The present study was the first to identify the effect of TRAP during pregnancy on maternal PPD. Further confirmation of the effects of these air pollutants and investigation of the relevant mechanisms are warranted.

**Data availability statement**

All data that support the findings of this study are included within the article (and any supplementary files).

**Acknowledgments**

This study was based on data from the TBCS Database, provided by the Health Promotion Administration, Ministry of Health and Welfare, Taiwan, R O C. We thank all children and parents who participated in this study, the interviewers who supported data collection, and all of the study groups who participated in the TBCS. We also thank the Department of Medical Research at National Taiwan University Hospital for helpful discussions during manuscript preparation.

**Funding information**

This study was supported by grants from National Taiwan University Hospital, Taiwan (Grant# 110-N4846) and Ministry of Science and Technology (Grant# MOST109-2621-M-002-021). The views expressed herein are the authors’ own.

**Ethical statement**

This study protocol has been reviewed and approved by the Institutional Review Board of National Taiwan University Hospital (#202007068RINC).

**ORCID iDs**

Ping Shih [https://orcid.org/0000-0002-2592-8273](https://orcid.org/0000-0002-2592-8273)
Chih-Da Wu [https://orcid.org/0000-0001-9720-7937](https://orcid.org/0000-0001-9720-7937)
Tung-liang Chiang [https://orcid.org/0000-0002-7876-3943](https://orcid.org/0000-0002-7876-3943)
Pau-Chung Chen [https://orcid.org/0000-0002-6242-5974](https://orcid.org/0000-0002-6242-5974)
Ta-Chen Su [https://orcid.org/0000-0001-7523-7166](https://orcid.org/0000-0001-7523-7166)
Tsun-Jen Cheng [https://orcid.org/0000-0002-2613-8230](https://orcid.org/0000-0002-2613-8230)
Yi-Hua Chen [https://orcid.org/0000-0003-2464-2537](https://orcid.org/0000-0003-2464-2537)

**References**

Aasheim V, Waldenström U, Hjelmstedt A, Rasmussen S, Pettersson H and Schytt E 2012 Associations between advanced maternal age and psychological distress in primiparous women, from early pregnancy to 18 months postpartum BJOG 119 1108–16

Akincigil A, Munch S and Niemczyk K C 2010 Predictors of maternal depression in the first year postpartum: marital status and mediating role of relationship quality Soc. Work Health Care 49 227–44

American Psychiatric Association 2000 Diagnostic and Statistical Manual of Mental Disorders 4th edn text revision (dsm-iv-tr) (Washington, DC: American Psychiatric Association)

Araujo J A 2011 Particulate air pollution, systemic oxidative stress, inflammation, and atherosclerosis Air Qual. Atmos. Health 4 79–93

Block M I and Calderón-Garcidueñas L 2009 Air pollution: mechanisms of neuroinflammation and CNS disease Trends Neurosci. 32 506–16

Boath E H, Pryce A J and Cox J L 1998 Postnatal depression: the impact on the family J. Reprod. Infant Psychol. 16 199–203

Brockmeyer S and D’Angiulli A 2016 How air pollution alters brain development: the role of neuroinflammation Transl. Neurol. 7 24–30

Brummelte S and Galea L A M 2016 Postpartum depression: etiology, treatment and consequences for maternal care Horm. Behav. 77 153–66

Calderón-Garcidueñas L et al 2008 Air pollution, cognitive deficits and brain abnormalities: a pilot study with children and dogs Brain Cogn. 68 117–27

Chen N-T, Lin P-H and Guo Y-L-L 2019 Long-term exposure to high temperature associated with the incidence of major depressive disorder Sci. Total Environ. 659 1016–20

Chen T-H, Hsu Y-C, Zeng Y-T, Candice Lung S-C, Su H-J, Chao H J and Wu C-D 2020 A hybrid kriging/land-use regression model with Asian culture-specific sources to assess NO\textsubscript{2} spatial-temporal variations Environ. Pollut. 259 113875

Fan S-J et al 2020 Ambient air pollution and depression: a systematic review with meta-analysis up to 2019 Sci. Total Environ. 701 134721

Fonken L K et al 2011 Air pollution impairs cognition, provokes depressive-like behaviors and alters hippocampal cytokine expression and morphology Mol. Psychiatry 16 987–95

Galea S and Tracy M 2007 Participation rates in epidemiologic studies Ann. Epidemiol. 17 643–53

Gan Z et al 2012 The impact of educational status on the clinical features of major depressive disorder among Chinese women J. Affect. Disord. 136 988–92

Glynn L M, Davis E P and Sandman C A 2013 New insights into the role of perinatal HPA-axis dysregulation in postpartum depression Neuroptides 47 363–70

Goyal D, Gay C and Lee K A 2018 Shortening day length: a potential risk factor for perinatal depression J. Behav. Med. 41 690–702

Hahad O, Lelieveld J, Birklein F, Lieb K, Daiber A and Munzel T 2020 Ambient air pollution increases the risk of inflammation, and atherosclerosis Int. J. Mol. Sci. 21 4306

HealthEffectsInstitute 2010 Traffic-related air pollution: a critical review of the literature on emissions, exposure, and health effects Health Effects Institute special report 17. (Boston, MA)

Heh S S 2001 Validation of the Chinese version of the Edinburgh postnatal depression scale: detecting postnatal depression in
Taiwanese women Hu Li Yan Jiu = Nursing Res. 9 105–13

Heinrichs M, Neumann I and Ehler U 2002 Lactation and stress: protective effects of breast-feeding in humans Stress 5 195–203

Hou P-C, Tu S-H, Liao P, Hung Y-T and Chang Y-H 2008 The typology of townships in Taiwan: the analysis of sampling stratification of the 2005–2006 “Taiwan Social Change Survey” Survey Res. Method Appl. 23 7–52

Huang Y C and Mathers N 2001 Postnatal depression—biological or cultural? A comparative study of postnatal women in the UK and Taiwan J. Adv. Nursing 33 279–87

Ikeda S, Tamaoki H, Akahane M and Neshi Y 1984 Effects of ritodrine hydrochloride, a beta-2-adrenoceptor stimulant, on uterine motilities in late pregnancy Japan. J. Pharmacol. 35 519–26

Iwata H, Morii E, Sakajo A, Aoki K, Machara K and Tamakoshi K 2016 Prevalence of postpartum depressive symptoms during the first 6 months postpartum: association with maternal age and parity J. Affect. Disord. 203 227–32

Jo S-J, Yim H W, Bang M H, Lee M O, Jun T-Y, Choi J-S, Lee M-S, Lee W-C and Park Y-M 2011 The association between economic status and depressive symptoms: an individual and community level approach Psychiatry Investig. 8 194–200

Jolley S N, Elmore S, Barnard K E and Carr D B 2007 Dysregulation of the hypothalamic-pituitary-adrenal axis in postpartum depression Biol. Res. Nursing 8 210–22

Kalisa E, Fadlallah S, Amani M, Nahayo L and Habiyaremye G 2012 The oxytocin-oxytocin receptor system: review & future perspectives Int. J. Pharmacol. 3 476–80

Lin Y, Zhou L, Xu J, Luo Z, Kan H, Zhang J, Yan C and Zhang J 2017 The impacts of air pollution on maternal stress during pregnancy Sci. Rep. 7 40956

Liu Q, Wang W, Gu X, Deng F, Wang X, Lin H, Guo X and Wu S 2015 Assessment of health-related quality of life in Taiwan J. Public Health 37 194–200

Lu J 2003 Air pollution during pregnancy and postpartum maternal psychological functioning PLoS One 8 e0195267

Mancuso C, Navarra P and Preziosi P 2010 Roles of nitric oxide, carbon monoxide, and hydrogen sulfide in the regulation of the hypothalamic-pituitary-adrenal axis Neuropsychopharmacology 35 198–202

Moura D, Canavarro M C and Figueiredo-Braga M 2016 Oxytocin and depression in the perinatal period—a systematic review Arch. Women's Mental Health 19 561–70

Niedzwiecki M M et al 2020 Particulate air pollution exposure during pregnancy and postpartum depression symptoms in women in Mexico City Environ. Int. 134 105325

Norhayati M N, Nik Hazlina N H, Arseeen A R and Wan Emlin W M A 2015 Magnitude and risk factors for postpartum symptoms: a literature review J. Affect. Disord. 175 34–52

O’Hara M W and McCabe J E 2013 Postpartum depression: current status and future directions Annu. Rev. Clin. Psychol. 9 379–407

Oktomti T, Nomoto K, Nakamura K and Goto F 1997 Nitric oxide metabolite in pregnant women before and after delivery Acta Obstet. Gynecol. Scand. 76 222–6

Owusu Darkwa E, Djablevely R, Sottie D, Owou C, Vanderpuye N M, Essuman R and Areye G 2018 Serum nitric oxide levels in healthy pregnant women: a case-control study in a tertiary facility in Ghana Maternal Health Neonatol. Perinatal. 4 3

Payne J L and Maguire J 2019 Pathophysiological mechanisms implicated in postpartum depression Front. Neuroendocriniol. 52 165–80

Pope C J and Mazmanian D 2016 Breastfeeding and postpartum depression: an overview and methodological recommendations for future research Depress Res. Treat 2016 4765310

Reuter M, Markett S, Melchers M and Montag C 2012 Interaction of the cholinergic system and the hypothalamic-pituitary-adrenal axis as a risk factor for depression: evidence from a genetic association study Neuroreport 23 717–20

Schiller C E, Meltzer-Brody S and Rubinow D R 2015 The role of reproductive hormones in postpartum depression CNS Spectr. 20 48–59

Sheffield P E et al 2018 Association between particulate air pollution exposure during pregnancy and postpartum maternal psychological functioning PLoS One 13 e0195607

Silverman M E, Reichenberg A, Savitz D A, Cnattingius S, Lichtenstein P, Hultman C M, Larsson H and Sandin S 2017 The risk factors for postpartum depression: a population-based study Depress Anxiety 34 178–87

Skurzda M, Bolten M, Naft I, Hellhammer D H and Meinlischmidt G 2011 Plasma oxytocin concentration during pregnancy is associated with development of postpartum depression Neuropeptides 46 213–5

Somma-Pillay P, Nelson-Piercy C, Tolppanen H and Mebazaa A 2016 Physiological changes in pregnancy Cardiovasc. J. Afr. 27 89–94

Teng H-W, Hsu C-S, Shih S-M, Lu M-L, Pan-J-J and Shen W W 2005 Screening postpartum depression with the taiwanese version of the 20-item version of the Edinburgh Postnatal Depression Scale Compr. Psychiatry 46 261–5

Vert C et al 2017 Effect of long-term exposure to air pollution on anxiety and depression in adults: a cross-sectional study Int. J. Hyg. Environ. Health 220 1074–84

Vert C et al 2017 Effect of long-term exposure to air pollution on anxiety and depression in adults: a cross-sectional study Int. J. Hyg. Environ. Health 220 1074–84

Vrachnis N, Malamas F M, Sifakis S, Deligorouglou E and Ilidromiti Z 2011 The oxytocin-oxytocin receptor system and its antagonists as tocolytic agents Int. J. Endocrinol. 2011 350546

Watkins S, Meltzer-Brody S, Zolnoun D and Stuebe A 2011 Early breastfeeding experiences and postpartum depression Obstet. Gynecol. 118 214–21

Whitaker R, West R T, Jaffery R, Lewis S, Kociolek A, Santolik P and Cingolani A 2018 Tibialis anterior strength and functional performance in adult Cerebral palsy: an exploratory study Phys. Ther. 99 131–9

World Health Organization Regional Office for Europe 2006 Air Quality Guidelines. Global Update 2005. Particulate Matter, World Health Organization Regional Office for Europe 2006 Air Quality Guidelines.
Ozone, Nitrogen Dioxide and Sulfur Dioxide (Copenhagen: World Health Organization Regional Office for Europe)

Wu C-D, Zeng Y-T and Lung S-C-C 2018 A hybrid kriging/land-use regression model to assess pm2.5 spatial-temporal variability Sci. Total Environ. 645 1456–64

Xie R H, Yang J, Liao S, Xie H, Walker M and Wen S W 2010 Prenatal family support, postnatal family support and postpartum depression Aust. N. Z. J. Obstet. Gynaecol. 50 340–5

Xu H, Ding Y, Ma Y, Xin X and Zhang D 2017 Cesarean section and risk of postpartum depression: a meta-analysis J. Psychosom. Res. 97 118–26

Yolton K, Khoury J C, Burkle J, LeMasters G, Cecil K and Ryan P 2019 Lifetime exposure to traffic-related air pollution and symptoms of depression and anxiety at age 12 years Environ. Res. 173 199–206

Yu G, Chen H, Wu X, Matta S G and Sharp B M 2010 Nicotine self-administration differentially modulates glutamate and gaba transmission in hypothalamic paraventricular nucleus to enhance the hypothalamic-pituitary-adrenal response to stress J. Neurochem. 113 919–29

Zeng Y, Lin R, Liu L, Liu Y and Li Y 2019 Ambient air pollution exposure and risk of depression: a systematic review and meta-analysis of observational studies Psychiatry Res. 276 69–78

Zijlema W L et al 2016 The association of air pollution and depressed mood in 70,928 individuals from four European cohorts Int. J. Hyg. Environ. Health 219 212–9