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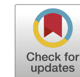
Review article



Environmental risk factors, protective factors, and biomarkers for postpartum depressive symptoms: an umbrella review

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ABSTRACT

We performed an umbrella review on environmental risk/protective factors and biomarkers for postpartum depressive symptoms to establish a hierarchy of evidence. We systematically searched PubMed, Embase, and the Cochrane Database of Systematic Reviews from inception until 12 January 2021. We included systematic reviews providing meta-analyses related to our research objectives. Methodological quality was assessed by AMSTAR 2, and the certainty of evidence was evaluated by GRADE. This review was registered in PROSPERO (CRD42021230784). We identified 30 articles, which included 45 environmental risk/protective factors (154,594 cases, 7,302,273 population) and 9 biomarkers (2018 cases, 16,757 population). The credibility of evidence was convincing (class I) for antenatal anxiety (OR 2.49, 1.91–3.25) and psychological violence (OR 1.93, 1.54–2.42); and highly suggestive (class II) for intimate partner violence experience (OR 2.86, 2.12–3.87), intimate partner violence during pregnancy (RR 2.81, 2.11–3.74), smoking during pregnancy (OR 2.39, 1.78–3.2), history of premenstrual syndrome (OR 2.2, 1.81–2.68), any type of violence experience (OR 2.04, 1.72–2.41), primiparity compared to multiparity (RR 1.76, 1.59–1.96), and unintended pregnancy (OR 1.53, 1.35–1.75).

1. Introduction

Postpartum depression is defined as a major depressive episode occurring within four weeks after delivery, which is encompassed by the "with peripartum onset" specifier in the DSM-5. In the eleventh revision of the ICD, postpartum depression is included in "mental or behavioral disorders associated with pregnancy, childbirth or the puerperium." In the clinical and research settings, however, postpartum depression is typically defined as the presence of depressive symptoms occurring up to 12 months after birth rather than the DSM or ICD definition (Stewart and Vigod, 2016). As one of the most common complications of pregnancy, the prevalence of postpartum depression is estimated to be approximately 9.2–19.2 % (Banti et al., 2011; Gavin et al., 2005), with variability arising from different diagnostic criteria and population-specific factors (O'Hara and McCabe, 2013). The disorder has a profound impact on the quality and function of the mother's life (Field, 2010; Salmela-Aro et al., 2001), affecting her children's behavior, cognitive development, and physical health (Goodman et al., 2011; Gump et al., 2009) and can lead to potentially fatal consequences for both the mother and her children (Gressier et al., 2017; Pearson et al., 2013).

Because of this high personal, clinical, and societal burden of postpartum depression, preventive approaches have been investigated. Understanding risk and protective factors associated with postpartum depression is a prerequisite to advancing preventive care (Jones et al., 2021). Accordingly, numerous primary studies have explored genetic and environmental factors, as well as biomarkers that might reflect their effects, showing that postpartum depression is caused by a complex interaction of genetic predispositions and environmental factors (Mahon et al., 2009; Payne and Maguire, 2019; Robertson et al., 2004; Segman et al., 2010). Although these studies have been summarized by meta-analyses, these are typically restricted to a single factor and do not carefully examine important biases including publication bias or reporting bias (Ioannidis, 2005, 2008). Therefore, the consistency and magnitude of environmental factors or biomarkers associated with postpartum depression are undetermined. Meanwhile, given that most previous studies used questionnaires such as the Edinburgh Postnatal Depression Scale (EPDS) rather than the DSM or ICD diagnosis, it would be more accurate to note that they investigated postpartum depressive 'symptoms' rather than 'disorder.' Moreover, some previous meta-analyses included less objective diagnostic methods such as self-reports or set too liberal cutoffs for determining postpartum depressive symptoms, which may have resulted in potential false positives and exaggerated effects. In this regard, this umbrella review aimed to provide a bird's eye view on environmental risk factors, protective factors, and biomarkers for postpartum depressive symptoms by applying the state-of-the-art hierarchical system and presenting detailed underlying mechanisms.

2. Methods

2.1. Protocol, registration, and study design

We performed an umbrella review of systematic reviews and meta-analyses in compliance with the updated PRISMA guidelines (Appendix pp 5–7) (Page et al., 2021). This review is registered with PROSPERO, number CRD42021230784, which is available online. The screening process, data extraction, and methodological appraisal of eligible articles were conducted independently by two investigators (JHK and SL), and any disagreement was resolved through discussion among four authors (JHK, JYK, SL, and JIS).

2.2. Search strategy and eligibility criteria

We systematically searched PubMed, Embase, and the Cochrane Database of Systematic Reviews from database inception to Jan 12, 2021, without any language restrictions. We used predetermined search terms including "postpartum", "depress* ", and "meta-analysis", and full search strategies for each database are presented in appendix p 8. To find eligible articles among the searched articles, each investigator screened titles, abstracts, and full texts in order. We also manually searched the references of relevant articles (Fig. 1).

We included systematic reviews providing meta-analyses that examined associations between postpartum depressive symptoms and environmental risk factors, protective factors, or biomarkers. The definitions of environmental risk factor, protective factor, and biomarker are presented in appendix p 9. Since most meta-analyses used questionnaires such as the EPDS rather than DSM or ICD criteria, we investigated 'postpartum depressive symptoms' that occurred within 12 months after childbirth. We included studies that used the validated diagnostic methods for determining postpartum depressive symptoms including not only DSM (any edition), ICD (any edition), and medical records but also EPDS, the Center for Epidemiologic Studies Depression scale (CES-D), Beck Depression Inventory (BDI), etc.

We excluded articles that did not study environmental risk factors, protective factors, or biomarkers for postpartum depressive symptoms; articles that did not provide meta-analyses; articles that did not provide sufficient data for the re-analysis of a meta-analysis (i.e., individual study estimates or the data to calculate them). We also excluded non-human studies, purely genetic studies, primary studies, and conference abstracts. If more than one meta-analysis covered the same topic, we prioritized the one with the largest number of individual studies, then the most recent one, and lastly, the one with the largest number of cases with postpartum depressive symptoms. The list of articles excluded at the full-text screening stage is presented in appendix pp 13–18.

2.3. Data extraction

From each eligible meta-analysis, we extracted the following data: the names of the authors; publication year; environmental risk factors, protective factors, or biomarkers; operationalization of depressive symptoms and applied cutoff for each individual study if available; number of cases with postpartum depressive symptoms and total study population; maximally adjusted individual study estimates and corresponding 95 % confidence intervals (95% CIs); metrics used in the original analyses (e.g. odds ratio [OR], relative risk [RR], Hedge's g); and study designs of individual studies (e.g. cohort, case-control, cross-sectional).

2.4. Data analysis

2.4.1. Main data analysis

We conducted a series of statistical tests to examine the robustness and consistency of data in accordance with previous umbrella reviews (Belbasis et al., 2015; Bellou et al., 2017; Kim et al., 2020, 2019) and recent guidance for umbrella review (Fusar-Poli and Radua, 2018). We re-analyzed each eligible meta-analysis based on extracted individual study estimates, using metrics used in the original meta-analysis. We calculated the summary effect estimate, corresponding 95 % CI, and p values under both random and fixed effects models. We further assessed whether p values < 0.001 or 0.000001 (Ioannidis et al., 2011; Sterne and Davey Smith, 2001). To evaluate heterogeneity, we performed Cochran's Q test and calculated the I^2 statistic ($I^2 > 50$ % indicates high heterogeneity) (Cochran, 1954). We assessed the existence of small study effects (i.e., larger studies have significantly more conservative results than smaller studies) with the regression asymmetry test proposed by Egger and colleagues (Egger et al., 1997), and small study effects were noted at Egger p value < 0.1. We estimated the 95 % prediction interval, the range in which we expect the effect of association would lie for 95 % of future studies (Higgins et al., 2009). We performed p-curve analysis and assessed the distribution of statistically significant p values to detect publication bias or p-hacking among the individual studies (Simonsohn et al., 2014a, 2014b), and we denoted a set of individual studies to have evidential value when the possibility of selective reporting was ruled out (p value for the right-skewness test for the half curve < 0.05 or p value for the right-skewness test < 0.1 for both the half and full curve) (Simonsohn et al., 2014b). We also performed random-effects meta-analyses under 5 %, 10 %, 15 %, and 20 % credibility ceilings to account for the potential methodological limitations of observational studies that might result in spurious significance (Papatheodorou et al., 2015; Salanti and Ioannidis, 2009).

The methodological quality of each eligible article was assessed using A Measurement Tool to Assess Systematic Reviews 2 (AMSTAR 2) by two independent investigators (JHK and SL) and any disagreements were resolved by consensus (Shea et al., 2017). The overall certainty of the estimate was evaluated based on the Grading of Recommendations, Assessment, Development, and Evaluations (GRADE) method by two authors (JHK and JYK), and any disagreements were resolved by consensus (Balshem et al., 2011). Because all included individual studies were observational studies, the decision for the certainty of evidence started at 'low' and downgraded to 'very low' when at least one reason to downgrade was identified, while upgraded to 'moderate' when some reason was found to upgrade such as large effect size.

2.4.2. Sensitivity analyses

We performed sensitivity analyses of the validated cutoff scores for determining postpartum depressive symptoms by excluding individual studies that used lower cutoffs than the validated ones, which may lead to false positive and exaggerated effects. The validated cutoffs we used for each included operationalization of depressive symptoms are presented in appendix p 10. We also conducted sensitivity analyses of cohort studies (retrospective or prospective), prospective cohort studies,

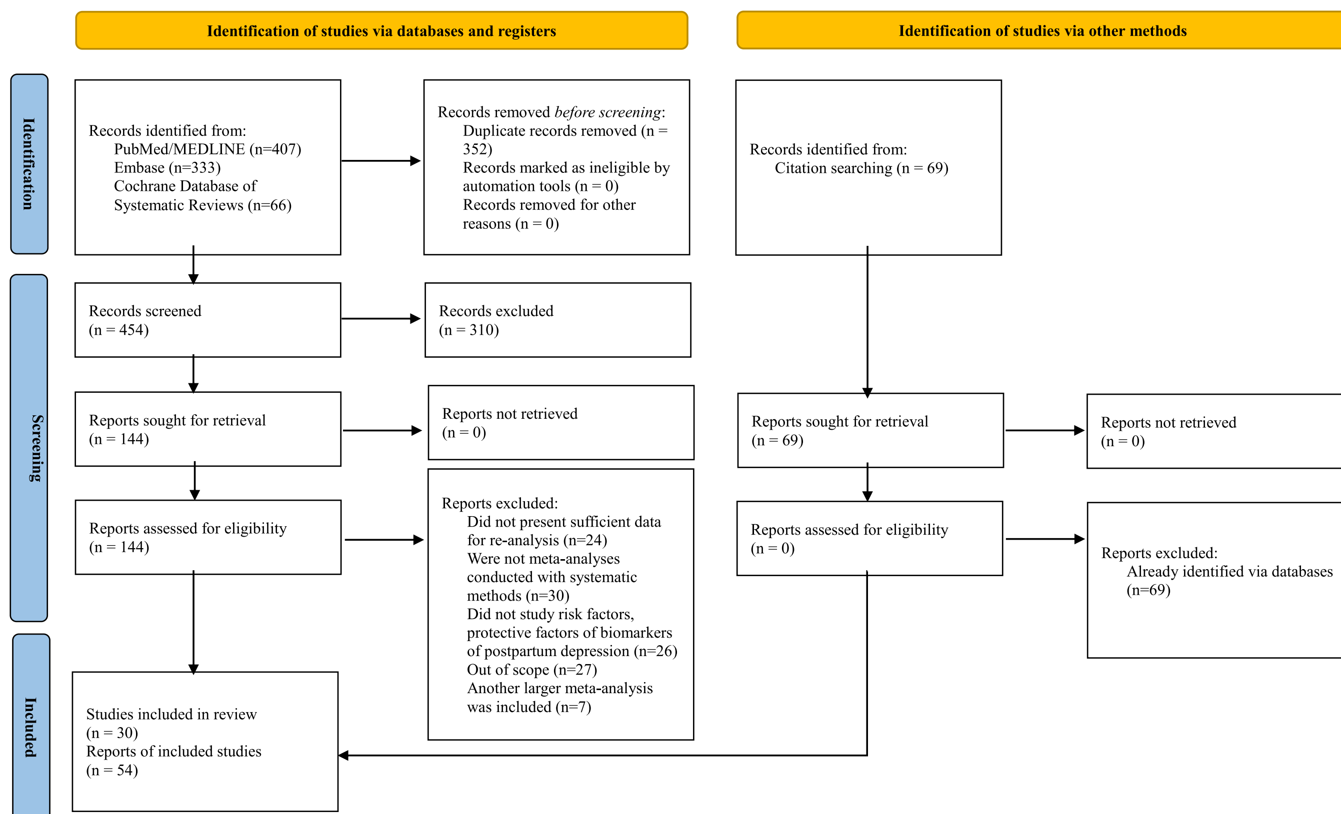

Fig. 1. PRISMA flow chart of study selection.

and study estimates adjusted for at least one confounder to further assess the robustness of the evidence. All sensitivity analyses were performed for associations graded as providing convincing or highly suggestive evidence. All statistical tests were two-sided and statistical significance was set at $p < 0.05$. All statistical analyses were performed by R version 4.0.4 and its packages.

2.5. Determining the credibility of evidence

Referring to the classification system of recent umbrella reviews (Belbasis et al., 2015; Bellou et al., 2017; Kim et al., 2020, 2019), we classified the identified associations into five classes by their level of credibility, based on the results of our statistical analyses – convincing (class I), highly suggestive (class II), suggestive (class III), weak (class IV), and not significant (NS) (Table 1). Criteria for classifying the level of evidence included p value under a random-effects model, number of cases with postpartum depressive symptoms, the p value of the largest study, the I^2 statistic, small study effects, results of the p-curve analysis, the 95 % prediction interval, and a random-effects p value under a 10 % credibility ceiling.

3. Results

3.1. Search results

From database inception to Jan 12, 2021, we identified 454 articles of which only 30 met the inclusion criteria (Fig. 1). Among the 30 articles, 54 unique meta-analyses were identified (45 environmental risk/protective factors and nine biomarkers; Table 2, Appendix p 19, 22–24, 28–122) (Azami et al., 2019a, 2019b; Bacchus et al., 2018; Beydoun et al., 2012; Cao et al., 2020; Chen et al., 2019; Dachew et al., 2021; Dadi et al., 2020; de Paula Eduardo et al., 2019; Desta et al., 2021; Falah-Hassani et al., 2015; Grigoriadis et al., 2019; Howard et al., 2013; Kang et al., 2020; Kountanis et al., 2020; Lin et al., 2017; Minaldi et al., 2020; Moameri et al., 2019; Molyneaux et al., 2014; Necho et al., 2020; Qiu et al., 2020; Tan et al., 2021; Tokumitsu et al., 2020; Tolossa et al., 2020; Wang et al., 2018; Yang et al., 2020; Yargawa and Leonardi-Bee, 2015; Ye et al., 2020; Zhang et al., 2019; Zhu et al., 2019).

3.2. Environmental risk factors and protective factors

The 45 meta-analyses of environmental risk/protective factors were based on 154,594 cases with postpartum depressive symptoms (median 1031 per meta-analysis, interquartile range [IQR] 551–5835, range 89–17,954) and included 7,302,273 total population (median 11,758 per meta-analysis, IQR 4437– 77,838, range 875– 2,302,311). Among them, 34 meta-analyses were based on cohorts, of which, 23 also included case-control or cross-sectional studies. The median number of study estimates was eight (IQR 5–12, range 2–39). Effect metrics were either OR or RR. Among 45 associations, 43 (96 %) associations were statistically significant with $p < 0.05$, 35 of 45 (78 %) with $p < 0.001$, and 13 of 45 (29 %) with $p < 0.000001$. Among 43 statistically significant associations, 25 (58 %) included more than 1000 cases with postpartum depressive symptoms. Only 14 of 45 (31 %) associations showed no heterogeneity (I^2<50 %). Among 45 associations, three (7 %) were not appropriate for Egger's test since they included less than three individual studies. Subsequently, 30 of 42 (71 %) associations presented no small study effect. Further, 39 of 45 (87 %) associations suggested no problems in the p-curve analysis, 33 of 45 (73%) retained statistical significance with a 10 % credibility ceiling, and the 95% prediction interval excluded the null value in 7 of 45 (16 %).

Only two environmental risk factors were graded as convincing evidence (class I; Table 2, Fig. 2): antenatal anxiety (OR 2.49, 95% CI 1.91–3.25) and psychological violence (OR 1.93, 95 % CI 1.54–2.42). Seven were graded as highly suggestive evidence (class II; Table 2, Fig. 2): intimate partner violence experience (OR 2.86, 95 % CI 2.12–3.87), intimate partner violence during pregnancy (RR 2.81, 95 % CI 2.11–3.74), smoking during pregnancy (OR 2.39, 95 % CI 1.78–3.2), history of premenstrual syndrome (OR 2.2, 95 % CI 1.81–2.68), any type of violence experience (OR 2.04, 95 % CI 1.72–2.41), primiparity compared to multiparity (RR 1.76, 95 % CI 1.59–1.96), and unintended pregnancy (OR 1.53, 95 % CI 1.35–1.75). Remarkably, 4 of 9 (44 %) factors with high level of evidence were related to violence against the mother. Other factors included preterm birth, pre-pregnancy obesity, cesarean section (class III), low income, poor social support, and poor marital relationship (class IV). Meanwhile, active husband participation in maternal healthcare/services during pregnancy and postpartum showed protective effects against postpartum depressive symptoms with statistical significance (class IV).

Table 1
Level of evidence for grading levels.

Main analysis					
Evidence level / Statistical analysis	Convincing (class I)	Highly suggestive (class II)	Suggestive (class III)	Weak (class IV)	Not significant (NS)
Random effects p value	$< 10^{-6}$	$< 10^{-6}$	$< 10^{-3}$	< 0.05	> 0.05
Number of cases with postpartum depressive symptoms	> 1000	> 1000	> 1000	x	x
P value of the largest study	< 0.05	< 0.05	x	x	x
Heterogeneity: I^2	< 50%	x	x	x	x
Small study effects	Not detected	x	x	x	x
P curve analysis	Evidential value found	x	x	x	x
95% prediction interval	Excludes the null	x	x	x	x
P value under 10% credibility ceiling	< 0.05	x	x	x	x

Sensitivity analyses
Subgroup analysis after excluding individual studies using low cut-off symptom score
Subgroup analysis of adjusted study estimates
Subgroup analysis of cohort studies
Subgroup analysis of prospective cohort studies

Table 2
Environmental risk/protective factors of postpartum depressive symptoms.

Exposure	Author, year	Number of cases / total population	Number of study estimates	Study design	Effect metrics	Random effects summary estimate (95 % CI)	Random effects p-value	I^2	95 % prediction interval	Large heterogeneity, small study effect, loss of significance under 10 % credibility ceiling, or evidential value not found under p-curve analysis	AMSTAR 2	GRADE
Convincing (class I)												
Antenatal anxiety	Grigoriadis 2019	1023 / 11,758	7	Cohort	OR	2.49 (1.91–3.25)	<0.000001	12 %	1.54–4.04	None	Critically low	Moderate
Psychological violence	Zhang 2019	6734 / 59,132	8	Cohort	OR	1.93 (1.54–2.42)	<0.000001	48 %	1.1–3.4	None	Critically low	Low
Highly suggestive (class II)												
Intimate partner violence experience	Howard 2013	1076 / 7497	12	Cohort, cross-sectional	OR	2.86 (2.12–3.87)	<0.000001	58 %	1.15–7.1	Large heterogeneity; small study effect	High	Very low
Intimate partner violence during pregnancy	Beydoun 2012	6106 / 21,339	17	Cross-sectional	RR	2.81 (2.11–3.74)	<0.000001	87 %	0.86–9.21	Large heterogeneity; small study effect	Critically low	Very low
Smoking during pregnancy	Chen 2019	2466 / 1,424,800	11	Cohort, case-control, cross-sectional	OR	2.39 (1.78–3.2)	<0.000001	80 %	0.88–6.45	Large heterogeneity	Critically low	Very low
History of premenstrual syndrome	Cao 2020	1400 / 8990	19	Cohort, case-control, cross-sectional	OR	2.2 (1.81–2.68)	<0.000001	42 %	1.21–4.01	Small study effect	High	Very low
Any type of violence experience	Zhang 2019	16,953 / 177,148	32	Cohort	OR	2.04 (1.72–2.41)	<0.000001	94 %	0.88–4.73	Large heterogeneity; small study effect	Critically low	Very low
Primiparity compared to multiparity	Tokumitsu 2020	14,048 / 102,006	39	Cohort, case-control, cross-sectional	RR	1.76 (1.59–1.96)	<0.000001	52 %	1.2–2.58	Large heterogeneity; small study effect	Critically low	Very low
Unintended pregnancy	Qiu 2020	5563 / 62,778	30	Cohort, case-control	OR	1.53 (1.35–1.75)	<0.000001	77 %	0.88–2.68	Large heterogeneity; small study effect	Low	Very low
Suggestive (class III)												
History of mental disorders	Dadi 2020	1106 / 14,991	5	Cohort, cross-sectional	OR	2.78 (1.82–4.27)	0.000003	85 %	0.61–12.69	Large heterogeneity; loss of significance under 10 % credibility ceiling	Moderate	Very low
Intimate partner violence in the past year	Bacchus 2018	> 1000 / 9175	7	Cohort	OR	2.19 (1.39–3.45)	0.00069	80 %	0.51–9.4	Large heterogeneity; loss of significance under 10 % credibility ceiling	Moderate	Very low
Preterm birth	de Paula Eduardo 2019	1042 / 8357	12	Cohort, case-control, cross-sectional	OR	2.14 (1.39–3.3)	0.00052	66 %	0.54–8.45	Large heterogeneity	Low	Low
Perinatal anemia	Kang 2020	2741 / 77,838	6	Cohort, case-control	RR	2.13 (1.54–2.95)	0.000005	44 %	0.92–4.91	None	Critically low	Low
Domestic violence	Zhang 2019	2123 / 23,996	16	Cohort	OR	2.05 (1.5–2.8)	0.000006	85 %	0.6–7.03	Large heterogeneity	Critically low	Very low
Physical violence	Zhang 2019	6489 / 57,783	8	Cohort	OR	1.9 (1.36–2.67)	0.00018	59 %	0.76–4.78	Large heterogeneity	Critically low	Very low
Immigration	Falah-Hassani 2015	3857 / 32,227	5	Cohort, cross-sectional	OR	1.84 (1.32–2.57)	0.0003	71 %	0.65–5.21	Large heterogeneity; small study effect	Low	Very low
Pre-pregnancy underweight	Dachew 2021	> 1000 / 617,985	5	Cohort	OR	1.71 (1.27–2.31)	0.00042	45 %	0.74–3.98	None	High	Low

(*continued on next page*)

Table 2 (*continued*)

Exposure	Author, year	Number of cases / total population	Number of study estimates	Study design	Effect metrics	Random effects summary estimate (95 % CI)	Random effects p-value	I^2	95 % prediction interval	Large heterogeneity, small study effect, loss of significance under 10 % credibility ceiling, or evidential value not found under p-curve analysis	AMSTAR 2	GRADE
Sexual violence	Zhang 2019	6196 / 56,117	6	Cohort	OR	1.56 (1.28–1.9)	0.000011	17 %	1.04–2.33	None	Critically low	Low
Cesarean section	Moameri 2019	8870 / 614,789	38	Cohort, case-control	OR	1.36 (1.2–1.55)	0.000001	54 %	0.82–2.26	Large heterogeneity	Critically low	Very low
Pre-pregnancy obesity	Molyneaux 2014	9085 / 90,777	14	Cohort, cross-sectional	OR	1.34 (1.19–1.51)	0.000003	48 %	1–1.8	None	Moderate	Very low
Elective cesarean section	Moameri 2019	8589 / 609,598	28	Cohort, case-control	OR	1.29 (1.12–1.49)	0.00036	48 %	0.8–2.1	Loss of significance under 10 % credibility ceiling	Critically low	Low
Weak (class IV)												
Poor social support	Tolossa 2020	832 / 5104	5	Cross-sectional	OR	6.6 (2.59–16.77)	0.000075	96 %	0.17–249.06	Large heterogeneity; small study effect	Critically low	Low
History of depression	Tolossa 2020	698 / 2876	6	Cross-sectional	OR	4.52 (2.69–7.59)	<0.000001	79 %	0.79–25.99	Large heterogeneity	Critically low	Very low
History of postpartum depression	Desta 2021	306 / 1361	3	Cross-sectional	OR	4.51 (2.4–8.45)	0.000003	65 %	0–5009.21	Large heterogeneity	Low	Low
Poor sleep quality	Yang 2020	89 / 7131	4	Cross-sectional	OR	4.06 (1.82–9.08)	0.00064	87 %	0.1–171.18	Large heterogeneity	Low	Very low
History of substance abuse	Desta 2021	306 / 1261	3	Cross-sectional	OR	3.78 (1.81–7.88)	0.0004	82 %	0–26468.56	Large heterogeneity	Low	Very low
History of infant death	Tolossa 2020	483 / 1909	5	Cross-sectional	OR	3.75 (1.85–7.61)	0.00025	83 %	0.29–49.21	Large heterogeneity	Critically low	Very low
Poor marital relationship	Necho 2020	948 / 5505	6	Cross-sectional	OR	3.38 (2.39–4.79)	<0.000001	100 %	0.92–12.41	Large heterogeneity	Critically low	Very low
History of stressful life event	Necho 2020	529 / 3658	2	Cross-sectional	OR	3.15 (1.71–5.79)	0.00023	77 %	NA	Large heterogeneity; loss of significance under 10 % credibility ceiling; p curve analysis unavailable due to less than three significant studies	Critically low	Very low
Exposure to different types of intimate partner violence	Dadi 2020	446 / 4473	10	Cohort, cross-sectional	OR	2.91 (2.37–3.59)	<0.000001	17 %	1.96–4.34	None	Moderate	Moderate
Low income	Necho 2020	699 / 4437	3	Cross-sectional	OR	2.52 (1.74–3.63)	<0.000001	4 %	0.21–30.86	None	Critically low	Moderate
Adverse birth and infant health conditions	Dadi 2020	554 / 13560	5	Cohort, cross-sectional	OR	2.38 (1.56–3.64)	0.000063	75 %	0.56–10.14	Large heterogeneity	Moderate	Very low
Postpartum anemia	Azami 2019	1031 / 3084	10	Cohort, cross-sectional	RR	1.89 (1.25–2.84)	0.0023	75 %	0.5–7.17	Large heterogeneity; small study effect; loss of significance under 10 % credibility ceiling	Critically low	Very low
Poor obstetric conditions	Dadi 2020	939 / 17,095	8	Cohort, cross-sectional	OR	1.72 (1.36–2.17)	0.000005	71 %	0.86–3.44	Large heterogeneity; small study effect	Moderate	Very low
Gestational diabetes	Azami 2019	17,954 / 2,302,311	14	Cohort, case-control, cross-sectional	RR	1.66 (1.21–2.27)	0.0015	89 %	0.52–5.3	Large heterogeneity	Critically low	Very low
Emergency cesarean section	Moameri 2019	4815 / 79,442	10	Cohort, case-control	OR	1.63 (1.21–2.21)	0.0014	68 %	0.66–4.04	Large heterogeneity; small study effect	Critically low	Very low
Childhood abuse	Zhang 2019	800 / 5027	5	Cohort	OR	1.62 (1.28–2.07)	0.000085	44 %	0.81–3.27	None	Critically low	Low

(*continued on next page*)

Table 2 (*continued*)

Exposure	Author, year	Number of cases / total population	Number of study estimates	Study design	Effect metrics	Random effects summary estimate (95 % CI)	Random effects p-value	I^2	95 % prediction interval	Large heterogeneity, small study effect, loss of significance under 10 % credibility ceiling, or evidential value not found under p-curve analysis	AMSTAR 2	GRADE
HIV infection	Zhu 2019	548 / 3780	10	Cohort, case-control, cross-sectional	OR	1.58 (1.08–2.32)	0.019	65 %	0.48–5.17	Large heterogeneity; loss of significance under 10 % credibility ceiling	Low	Very low
Anemia during pregnancy	Azami 2019	261 / 2785	8	Cohort	RR	1.24 (1–1.54)	0.048	39 %	0.73–2.12	Loss of significance under 10 % credibility ceiling; p curve analysis unavailable due to less than three significant studies	Critically low	Low
Female infant compared to male infant	Ye 2020	14,358 / 119,281	29	Cohort, case-control	OR	1.15 (1.01–1.31)	0.035	75 %	0.66–2	Large heterogeneity; small study effect; loss of significance under 10 % credibility ceiling	Critically low	Very low
Pre-pregnancy overweight	Dachew 2021	983 / 619,568	6	Cohort	OR	1.14 (1–1.3)	0.043	27 %	0.85–1.53	Loss of significance under 10 % credibility ceiling; p curve analysis unavailable due to less than three significant studies	High	Low
Active husband participation in maternal healthcare/ services during pregnancy	Yargawa 2015	156 / 875	2	Cohort, cross-sectional	OR	0.36 (0.2–0.68)	0.0014	48 %	NA	Loss of significance under 10 % credibility ceiling; p curve analysis unavailable due to less than three significant studies	Low	Low
Active husband participation in maternal healthcare/ services postpartum	Yargawa 2015	484 / 2149	5	Cohort, case-control, cross-sectional	OR	0.34 (0.19–0.62)	0.00038	57 %	0.06–2	Large heterogeneity	Low	Low
Not significant (NS)												
Family history of mental illness	Necho 2020	299 / 1198	2	Cross-sectional	OR	1.93 (0.66–5.62)	0.23	75 %	NA	Large heterogeneity	Critically low	Very low
Labor epidural analgesia	Kountanis 2020	609 / 5322	10	Cohort, case-control	OR	1.03 (0.71–1.52)	0.86	79 %	0.3–3.55	Large heterogeneity	Critically low	Very low

All statistical tests are two-sided.
Abbreviations: AMSTAR 2, A Measurement Tool to Assess Systematic Reviews 2; CI, confidence interval; GRADE, Grading of Recommendations, Assessment, Development and Evaluations; HIV, human immunodeficiency virus; NA, not available; OR, odds ratio; RR, relative risk

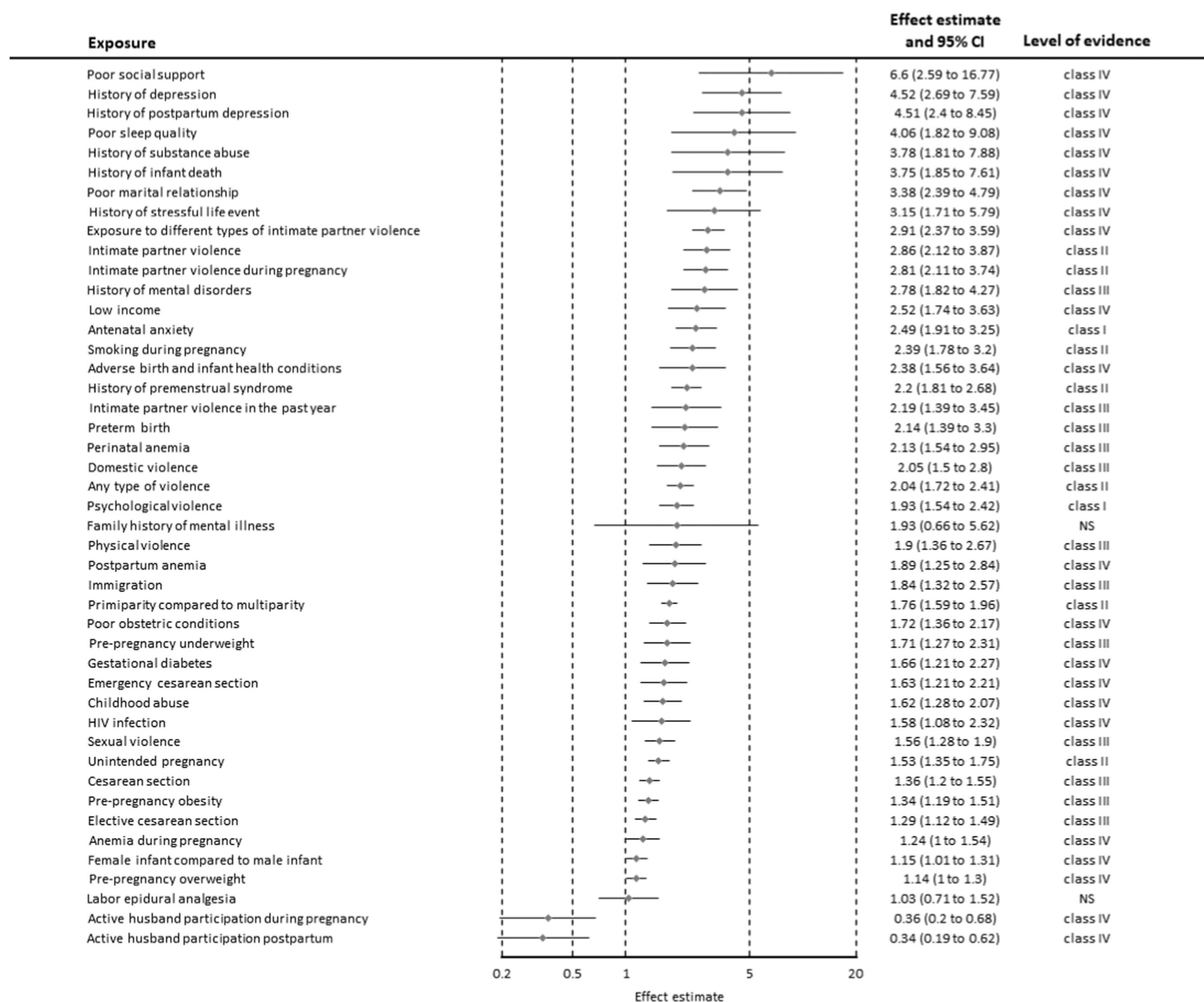

Fig. 2. Summary estimates of environmental risk and protective factors for postpartum depressive symptoms.

3.3. Biomarkers

The nine biomarker meta-analyses covered 2018 cases with postpartum depressive symptoms (median 201 per meta-analysis, IQR 200–215, range 168–404) and 16,757 total population (median 1793 per meta-analysis, IQR 1741–1793, range 1432–2375). All nine meta-analyses were based on cohorts, of which, four also included case-control or cross-sectional studies. The median number of study estimates was five (IQR 5–6, range 3–7). Effect metrics were either OR, RR, or Hedge's g. Among nine associations, only three (33 %) were statistically significant with $p < 0.05$, while there was no association with $p < 0.0001$. No association included more than 1000 cases with postpartum depressive symptoms, and only 3 of 9 (33 %) associations showed no heterogeneity. All associations were available for Egger's test and 7 of 9 (78 %) showed no small study effect. However, all but one suggested a problem in the p-curve analysis. No association retained statistical significance with a 10 % credibility ceiling and excluded the null value in the 95 % prediction interval. Accordingly, no association was graded as convincing or highly suggestive evidence (Appendix p 19).

3.4. AMSTAR 2 quality assessment

AMSTAR 2 quality assessment was available for all associations. Among 30 articles, 26 reported environmental risk/protective factors and four biomarkers. Of 26 meta-analysis articles on environmental risk/protective factors, only three (11 %) were graded as high quality, two (8 %) moderate, seven (27 %) low, and 14 (54 %) critically low. Of four meta-analysis articles on biomarkers, one (25 %) was graded as low, and three (75 %) were critically low. Among factors with a high level of evidence, only two (intimate partner violence experience and history of premenstrual syndrome) were graded as high quality.

3.5. Certainty of evidence using the GRADE method

Certainty of evidence was assessed for each estimate based on the GRADE method (Table 1, Appendix p 19). Out of 45 meta-analyses of environmental risk/protective factors, three (7 %) were rated as moderate, 13 (29 %) were low, and 29 (64 %) were very low. Out of nine meta-analyses of biomarkers, one (11 %) was rated as low and eight (89 %) were very low. Among the factors with a high level of evidence, only one (antenatal anxiety) was graded as moderate. Detailed information on the decision of certainty of evidence for each estimate is presented in appendix pp 25–27.

3.6. Sensitivity analyses

Sensitivity analyses of the validated cutoff scores for meta-analyses with a high level of evidence (class I or II) were conducted. After excluding individual studies that used a lower cutoff than the validated one, 7 of 9 (78 %) factors retained their level of evidence: antenatal anxiety (class I), intimate partner violence experience, intimate partner violence during pregnancy, smoking during pregnancy, history of premenstrual syndrome, any type of violence experience, and unintended pregnancy (class II), whereas the rest were downgraded to class III or IV. Sensitivity analyses of (1) cohort (retrospective and prospective), (2) prospective cohort, and (3) adjusted study estimates for meta-analyses with a high level of evidence (class I or II) were also performed. In the cohort sensitivity analyses, five factors retained their level of evidence: antenatal anxiety, psychologic violence (class I), any type of violence experience, primiparity compared to multiparity, and unintended pregnancy (class II), whereas the rest were downgraded to class III or IV, or inappropriate for subgroup analysis since they included fewer than two cohort studies. In the prospective cohort subgroup analysis, the same factors retained the level of evidence except for antenatal anxiety (class I to III). In the sensitivity analyses of adjusted study estimates, which was unavailable for one (intimate partner violence experience), 5 of 8 (63 %) factors graded as class II: psychologic violence, intimate partner violence during pregnancy, any type of violence experience, primiparity compared to multiparity, and unintended pregnancy, whereas the rest were downgraded to class III or IV. All statistical details of the sensitivity analyses are presented in Appendix pp 20–21.

4. Discussion

4.1. Summary of important results

To the best of our knowledge, this study is the first umbrella review based on the state-of-the-art evidence grading strategy, which systematically and quantitatively collected and assessed the hierarchy of evidence for environmental risk factors, protective factors, and biomarkers for postpartum depressive symptoms. Only nine associations of environmental risk factors showed evidence of high credibility (antenatal anxiety, psychological violence [class I], intimate partner violence experience, intimate partner violence during pregnancy, smoking during pregnancy, history of premenstrual syndrome, any type of violence experience, primiparity compared to multiparity, and unintended pregnancy [class II]).

4.1.1. Strength of the present study

Indeed, there are three previous studies attempted to summarize the evidence on environmental risk factors of postpartum depressive symptoms (Gastaldon et al., 2022; Hutchens and Kearney, 2020; Zhao and Zhang, 2020). However, two reviews (Hutchens and Kearney, 2020; Zhao and Zhang, 2020) did not apply a hierarchical system that can account for several types of biases (Fusar-Poli and Radua, 2018). Meanwhile, Gastaldon et al. (Gastaldon et al., 2022) established a hierarchy of the evidence but reported 12 potential risk factors which is fewer than 45 risk factors identified in our review. We also found two risk factors with convincing evidence (Class I) (antenatal anxiety and psychological violence), whereas Gastaldon et al. found none. It should also be noted that the criteria for convincing evidence (class I) is stricter in our review than the review by Gastaldon et al., given that we used 10 % credibility ceilings test, which was introduced in previous umbrella reviews (Kim et al., 2020, 2019), and we also used a novel p-curve analysis to detect p hacking. Lastly, we endeavored to address the underlying biological and/or behavioral mechanisms in detail for each risk factors with high level of evidence (class I and II).

4.2. Psychological violence, intimate partner violence experience, intimate partner violence during pregnancy, and any type of violence experience

Various types of violence against the mother (psychological violence (Zhang et al., 2019) [class I]; intimate partner violence experience (Howard et al., 2013), intimate partner violence during pregnancy (Beydoun et al., 2012), and any type of violence experience (Zhang et al., 2019) [class II]) were associated with a higher risk of postpartum depressive symptoms. Of note, psychological violence was downgraded to class III in the sensitivity analysis of the validated cutoff scores, while others were not. Though the underlying mechanism is unclear, given that violence against the mother is a type of stress, stress-related neuroendocrine dysfunction and gene-stress interaction seem to be the most plausible explanations. The former suggests that the unbalanced secretion of glucocorticoids, the final product of the hypothalamic-pituitary-adrenal (HPA) axis, which is activated by a stress response, may affect psychological function, leading to depression (Brummelte and Galea, 2010; Meltzer-Brody, 2011). The latter proposes that reduced activity of brain-derived neurotrophic factors resulting from stressful events may lead to the diminished function of brain regions, including those involved in emotional processing and cognition, and eventually, subsequent changes in mood and depression (Begni et al., 2017; Brunoni et al., 2008; Molendijk et al., 2014). Notably, the majority of factors related to violence against the mother —including class I, II, and also others —had effect sizes larger than two. In this regard, the violence experience of the mother may be a robust predictor of postpartum depressive symptoms despite its somewhat large heterogeneity. These findings emphasize the necessity of screening for domestic and intimate partner violence and promoting maternal mental health.

4.3. Antenatal anxiety

Antenatal anxiety (Grigoriadis et al., 2019) provided convincing evidence for increasing the risk of postpartum depressive symptoms with an effect size larger than two (OR 2.64, 95 % CI 2.02–3.46), retaining convincing evidence in sensitivity analysis of the validated cutoff scores. Notably, antenatal anxiety showed moderate certainty of evidence according to the GRADE method even though its analysis only contained observational studies. It should be mentioned that the factor is simply anxiety, which represents symptoms rather than the disorder. Indeed, individual studies in the meta-analysis included not only those that used the diagnostic criteria of anxiety disorder but also those that used anxiety questionnaire scores (e.g., state-trait anxiety inventory-trait score itself) or an additional cut-off system (e.g., state-trait anxiety inventory-trait score $>$ 45). Of note, the latter distinguished excessively anxious mothers from those experiencing anxiety of a normal range by setting certain cutoff scores such as one standard deviation above the mean or the top 25th percentile. In terms of anxiety disorders, antenatal social phobia (Coelho et al., 2011), generalized anxiety disorder (Coelho et al., 2011), and panic disorder (Rambelli et al., 2010) are also suggested to be independent risk factors for postpartum depressive symptoms respectively. Although robust biological mechanisms have yet to be identified, it is important to point out that (1) anxiety symptoms are frequently reported in pregnancy and often even considered a typical experience of pregnancy, and (2) problematic anxiety symptoms in pregnancy were not well distinguished from normal anxiety, and thereby the anxiety symptoms of mothers should not simply be considered to be a normal adaptive part of pregnancy.

4.4. Smoking during pregnancy

Smoking during pregnancy (Chen et al., 2019) was associated with an increased risk of postpartum depressive symptoms with highly suggestive evidence, retaining the level of evidence in sensitivity analysis of the validated cutoff scores while downgraded to weak in other

sensitivity analyses. Regarding its biological mechanisms, it has been proposed that smoking may have anti-estrogenic effects by disrupting endogenous estrogen biosynthesis and bioavailability (Baron, 1984; Ruan and Mueck, 2015), given that women are prone to mood fluctuation during the period when hormone levels (especially sex steroid hormones such as estrogen and progesterone) change rapidly (Schiller et al., 2015). HPA axis activation due to immune system alteration (Lee et al., 2012; McEvoy et al., 2015; Pace and Miller, 2009), increased oxidative stress (Black et al., 2015; Yanbaeva et al., 2007), and nicotine acetylcholine receptors (Philip et al., 2010) induced by smoking are other potential mechanisms. Meanwhile, numerous investigations have been conducted regarding the various smoking cessation patterns and corresponding risk of postpartum depressive symptoms. Salimi et al. (Salimi et al., 2015) reported the odds of postpartum depressive symptoms in women who quit smoking during the final 3 months of pregnancy but resumed after parturition (OR 1.28, 1.06–1.53) and who did not quit at all (OR 1.48, 1.26–1.73) compared to those who quit during the final 3 months of pregnancy and remained non-smokers after parturition. Although using a less rigorous definition of postpartum depression, this finding demonstrates that smoking cessation is important not only before or during pregnancy but also in the postpartum period to prevent postpartum depressive symptoms. In addition, passive smoking should also be avoided (Song et al., 2019). Potential confounders of the association should be accounted for, such as prenatal stressful events which may be associated with both smoking and postpartum depressive symptoms (Kassel et al., 2003; Necho et al., 2020).

4.5. History of premenstrual syndrome

History of premenstrual syndrome (Cao et al., 2020) was associated with an increased risk of postpartum depressive symptoms with highly suggestive evidence, retaining the level of evidence in sensitivity analysis of the validated cutoff scores while downgraded to weak in other sensitivity analyses. This association is noteworthy because premenstrual syndrome has a high prevalence of around 70 % (Ranjbaran et al., 2017). Regarding its underlying mechanisms, increased sensitivity to hormonal fluctuation has been suggested to be the most plausible one (Schiller et al., 2016; Yonkers et al., 2008). Two reproductive steroid hormones, estrogen and progesterone, may play a major role (Schiller et al., 2016; Stoner et al., 2017). The levels of both hormones increase before the luteal phase and during pregnancy but rapidly decrease in the luteal phase and after parturition, and this kind of fluctuation contributes to the development of the premenstrual syndrome and postpartum depressive symptoms respectively, in those vulnerable to it (Bloch et al., 2000; Franz, 1988). It should be emphasized that hormonal fluctuation itself in patients with premenstrual syndrome or postpartum depressive symptoms is not the issue as these patients have been found to have a normal hormone level, rather, the problem is patients' vulnerability to hormonal fluctuation (Rubinow and Schmidt, 2006). Although this may not appliable to late-onset postpartum depressive symptoms since the hormones level recovers to a steady state, this explanation seems to be most persuasive given that depression is more prevalent in women from puberty to menopause than in men of the same age, but this is reversed in childhood or after menopause (Bebbington et al., 2003; Birmaher et al., 1996; Jung et al., 2015). Meanwhile, other mechanisms have also been proposed such as inadequate vitamin D status (Jarosz and El-Sohemy, 2019; Wang et al., 2018) and cytokine effects (Stoner et al., 2017).

4.6. Primiparity compared to multiparity

Primiparity (Tokumitsu et al., 2020) is associated with a higher risk of postpartum depressive symptoms compared to multiparity with highly suggestive evidence, which was confirmed in all subgroup analyses except for the validated cutoff score analysis. Indeed, several reasons have been suggested as to why postpartum depressive symptoms are more prevalent in primiparity than multiparity. First, multiparity may be more experienced in adapting to stress or other adversities accompanied by pregnancy and parturition. Second, given that history of postpartum depression may be another risk factor for postpartum depressive symptoms despite its low level of evidence (class IV) (Desta et al., 2021), those who have experienced postpartum depression may endeavor not to endure it again by receiving psychological education, taking preventive measures against depression, or being reluctant to conceive again. Third, primiparous women are at an increased risk of having anxiety and sexual problems, which may eventually lead to postpartum depressive symptoms (Martínez-Galiano et al., 2019). Although the aforementioned factors may not fully account for the association and other unidentified factors may exist, this association might have major implications for healthcare professionals or national health care planners by alerting them to the necessity of paying more attention to mothers who become pregnant for the first time.

4.7. Unintended pregnancy

Unintended pregnancy (Qiu et al., 2020) provided highly suggestive evidence for higher risk of postpartum depressive symptoms, which was confirmed in all sensitivity analyses. In the regard that women who conceive unintentionally seem to experience psychosocial stress due to concerns after pregnancy such as interruptions in their education, career, or other life aspirations (Faisal-Cury et al., 2017; Steinberg and Rubin, 2014), stress-related neuroendocrine dysfunction and gene-stress interaction seems to be the two most plausible biological mechanisms that underlie the association between unintended pregnancy and postpartum depressive symptoms. A detailed explanation of these suggested mechanisms has already been mentioned above. Further, other behavioral mechanisms have also been suggested. First, mothers conceive without intention tend to start late and seldom complete prenatal care, which can be detrimental to maternal mental health (Karaçam et al., 2011). Second, a pregnancy that is unexpected and thus unplanned may result in adjustment stress in the mother, leading to concerns about maternal and fetal health and even conflicts regarding maintaining versus terminating the pregnancy (Faisal-Cury et al., 2017). Third, mothers with unintended pregnancies tend to smoke more and take fewer vitamins than those who have planned pregnancies, which plausibly explains their higher risk of postpartum depressive symptoms given that smoking (Chen et al., 2019) and lack of vitamin D supplementation (Sheikh et al., 2017) were significantly associated with postpartum depressive symptoms.

4.8. Limitations

The present study has some limitations. First, since all meta-analyses were based on observational studies, reported associations do not necessarily imply causality and we could not completely exclude potential confounders, which requires a caution in interpreting the findings. Second, most of the identified associations showed large heterogeneity. This may be due to the unstandardized way in which variables have been operationalized as well as various cutoff points for determining postpartum depression. Meanwhile, the operationalization of environmental factors may be also inconsistent across studies. Third, a large portion of meta-analyses showed "low" or "critically low" methodological quality. Majority of them did not report a protocol before conducting a review and did not provide the list of excluded articles and exclusion reason. Fourth, we could only address the associations which were synthesized by meta-analyses; that is, we may have inevitably missed some important factors. Besides, although the most current concept of "perinatal depression" includes both prenatal and postnatal maternal depression, which does not allow the discrimination between intrauterine and postnatal effects, we focused on the sole postpartum depressive symptoms. We may miss some factors related to both maternal/newborn outcomes and interventions that may directly

affect and modulate the magnitude of the effects of the candidate environmental factors and biomarkers appraised herein. However, this is an intrinsic limitation since our study was based on previous meta-analyses that only focused on postpartum depression.

5. Conclusions

Our umbrella review identified convincing evidence indicating that antenatal anxiety and psychological violence are robustly associated with postpartum depressive symptoms, while no associated protective factors or biomarkers showed robust evidence. Since these associations cannot imply causality, further well-designed primary studies with the ICD/DSM-established operationalization of postpartum depression are needed to confirm these findings.

Competing interests

All authors have completed the ICMJE uniform disclosure form at www.icmje.org/coi_disclosure.pdf and declare: GSP is supported by Alicia Koplowitz Foundation and Janssen Cilaq. BS is supported by a National Institute for Health Research (NIHR) Advanced Fellowship (301206, 2021–20216). BS is a lead/co-investigator the following active grants 1) NIHR program grant: Supporting Physical and Activity through Co-production in people with Severe Mental Illness (SPACES,2021–2027); 2) TB multimorbidity with the Medical Research Council (GCRF call (2020–2022); 3) Determinants of MLTCs among young adults with mental disorders: a data-linkage study, Guy's & St Thomas' Charity (2020–2022); 4) Mechanisms underlying the role of gut-microbiota in exercise-induced changes in cognitive function in middle-age, Reta Lila Weston Trust For Medical Research (2021–2024); 5) Improving Outcomes in Mental and Physical Multi-morbidity and Developing Research Capacity (IMPACT) in South Asia, NIHR Global Research program grant (2017–2022). BS also works at King's College London; disseminating and publishing evidence is integral to his employment (although his salary is wholly covered by the above fellowship). BS also works clinically with people that use mental health services in the National Health Service (NHS). BS has published a book on exercise and mental illness and is on the Editorial board of Ageing Research Reviews, Mental Health and Physical Activity, The Journal of Evidence Based Medicine and The Brazilian Journal of Psychiatry. BS has received honorarium for advisory work from ASICS Europe BV & Parachute BH for work unrelated to this project. The views expressed are those of the author(s) and not necessarily those of mentioned above, the NHS, the NIHR, the Department of Health and Social Care, the MRC or GSTT.

Data availability

Data that has been used are available in online without restriction. All data used in this study were from publicly available articles.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.neubiorev.2022.104761.

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