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Biopsychosocial predictors of perinatal depressive symptoms: Moving toward an integrative approach



Perinatal depression is defined as the occurrence of a depressive episode in pregnancy or within six weeks post partum (DSM-5; American Psychiatric Association, 2013). It is a major global health concern, affecting the health and well-being of more than 10% of pregnant women, new mothers and their families each year (Gavin et al., 2005). Accordingly, a growing body of strong evidence addresses the prevalence, risks and consequences of this disorder.

Specifically, a large number of studies have investigated biopsychosocial risk factors for perinatal depression. In our 2015 systematic review of biological and psychosocial predictors of postpartum depressive symptoms (Yim, Tanner Stapleton, Guardino, Hahn-Holbrook, & Dunkel Schetter, 2015), we identified 199 relevant studies. One of our major findings was that the separate literatures investigating biological and psychosocial contributors to postpartum depression risk had evolved independently and remained distinct. That is, of the 199 studies, a mere eleven considered at least one biological and one psychological predictor together in the same study; of those eleven, only six included the biological and psychological variables in the same statistical model. Thus, we concluded our previous review paper with a “Call for Integration.”

Since the publication of our systematic review, the number of studies testing biopsychosocial factors in postpartum depressive symptoms has increased somewhat. In an effort to quantify this increase, we again systematically searched the literature in the same manner as in our initial systematic review conducted five years ago (Yim et al., 2015). Publication dates were limited to January of 2014 to December of 2018 for the present search. Briefly, combinations of the key words “depression,” “postpartum” and “postnatal” were combined with relevant broad and specific biological terms (e.g., biological, hormone, cortisol) and psychosocial terms (e.g., psychological, stress, demands). Similar to our previous search, we included only English language publications and peer-reviewed human studies within the first year following birth that reported a statistical association between the relevant variables. However, because we were only interested in studies that included both a biological *and* psychosocial predictor, we required this when searching – in deviation from our 2015 search. In other words, search findings had to include the depression-related keyword, a biological term, and a psychosocial term to be included here.

We identified 69 records in PubMed and 42 in PsycINFO, resulting in 98 records after removal of 13 duplicates (see flow diagram in Fig. 1). Four additional manuscripts were identified through a reference section review. Of these 102 records, 61 were deemed clearly irrelevant after abstract review. The remaining 41 full text articles were assessed for eligibility, and 32 studies were excluded on the basis of the criteria stated. The remaining nine papers met inclusion criteria and are

summarized in detail in Table 1.

What stands out is that the rate of publications of integrative studies has indeed increased. We now identified nine integrative papers published across roughly three years, whereas in our previous review we identified six papers published over the course of 13 years. We interpret this to reflect an important shift among researchers toward recognition of the importance of integrative approaches. Moreover, we note that the new studies identified are less likely to rely on solely cortisol measures and instead include other important biological markers such as oxytocin and markers of inflammation treated as both cross-sectional correlates and longitudinal predictors of postpartum depressive symptoms.

To summarize briefly the publications over the last three years, studies of oxytocin confirm our previous conclusion that evidence is largely in favor of an inverse association with postpartum depressive symptoms (e.g., Eapen et al., 2014), with some important qualifications. For example, one study found this association only among individuals who also scored high on a measure of psychosocial stress (Zelkowitz et al., 2014), and another found no association between endogenous oxytocin and postpartum depressive symptoms overall, but yielded a positive association with synthetic oxytocin administered at birth, which held in a model covarying relationship status (Gu et al., 2016). One last study found that childhood abuse and postpartum depression interacted to modulate oxytocin receptor DNA methylation (Kimmel et al., 2016).

In studies assessing inflammatory markers, one suggests that inflammatory markers (IL-6, CRP) were associated with depressive symptoms within two days of hospital admission for delivery, and controlling for stressful life events and low partner support (Liu, Zhang, Gao, & Zhang, 2016). Another investigation found that TNF- α and IL-10 were associated with EPDS scores measured at 1 week post partum, but this association did not hold in a multivariate model controlling for the effects of perceived stress (Dunn, Paul, Ware, & Corwin, 2015). Similarly, in a path model revealing significant direct effects of perceived stress on EPDS symptoms, neither a latent inflammation variable nor diurnal salivary cortisol emerged as significant (Ruyak, Lowe, Corwin, Neu, & Boursaw, 2016). Also concerning HPA axis function, one study showed that the association between an HPA axis-related polymorphism and depressive symptoms at six weeks post partum was mediated by neuroticism (Iliadis et al., 2017). And, a single study reported no evidence of an association of postpartum depressive symptoms and self-reported social support or adiponectin, a hormone involved in glucose and lipid metabolism (Rebelo, Farias, Struchiner, & Kac, 2016).

This brief summary of the literature published between our previous review and the publication of this special issue provides further

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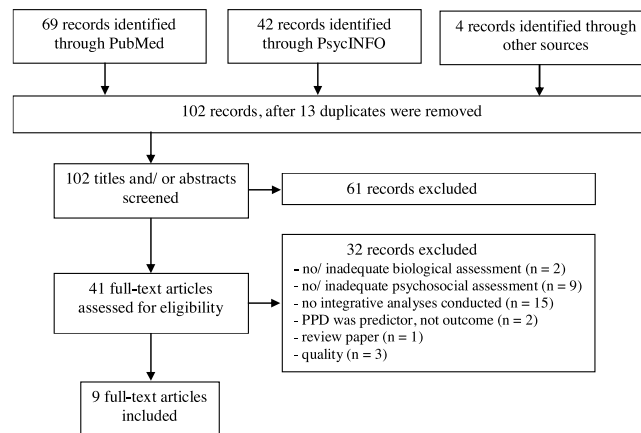


Fig. 1. Flow Chart Following Guidelines in the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) statement [Moher].

evidence that meaningful significant associations exist between a range of biological markers and postpartum depressive symptoms, and, more importantly, that these associations can be moderated or mediated by the psychosocial context in which they occur. However, it is clear that much more research is needed to understand how the emergence of postpartum depressive symptoms is influenced by the intricacies of the biopsychosocial context in which they occur.

For this special issue, we invited an international group of experts to contribute papers on their research findings on biopsychosocial processes in perinatal depression. We opened the topic to perinatal from postpartum so as to include prenatal depressive symptoms. This decision was made in part because the new guidelines in the DSM-5 use a perinatal specifier instead of a postpartum specifier to diagnose pregnancy-related depression, and also because postpartum depressive symptoms often can be traced to prenatal origins (Beck, 2001). Thus, focusing on the continuum rather than merely postpartum depression seemed appropriate.

For this special issue of *Biological Psychology*, we included seven manuscripts. The volume starts with a systematic literature review of the association between premenstrual syndrome and postpartum depression. Amiel Castro, Pataky, and Ehlert (2018) compile and critically discuss empirical evidence suggesting that the – widely assumed – positive association between premenstrual syndrome and postpartum depression finds support in the currently available literature, and that this association may be affected by confounders, including vulnerable personality and socioeconomic factors.

Following this introductory piece, we present three manuscripts addressing associations between cortisol and perinatal depressive symptoms. In the first of these, Luecken, Crnic, Gonzales, Winstone, and Somers (2018) demonstrated in a sample of 322 low-income Mexican-American mother-infant dyads the existence of a pathway linking prenatal stress to postpartum depressive symptoms up to six months post partum through dysregulated mother-infant interactions and the infant's cortisol stress response, an effect that emerged above and beyond the significant effects of prenatal on postnatal depressive symptoms that were also observed. Also in a low-income, and ethnically diverse sample, Urizar et al. (2018) showed that women with high depression risk showed blunted cortisol responses to a laboratory stressor, controlling for gestational age, parity and education. Ethnicity, however, was an important moderator in this relationship. African American women, in particular those who were at high risk for depression, showed a blunted cortisol response, compared to non-African American women. In the third study, Kofman et al. (2019) report that, controlling for gestational age, lack of support from baby's father, cortisol reactivity, and maternal neuroticism all contributed to depressive symptoms during pregnancy, with improved prediction when variables were

simultaneously entered into the statistical model. The interaction between maternal neuroticism and perceived support from baby's father emerged as a significant predictor for depressive symptoms when cortisol reactivity was conceptualized as the cortisol awakening response, suggesting that women low in neuroticism might benefit more from partner social support such that this support can act as a buffer in the link between stress and health.

Two papers addressed the association between oxytocin and perinatal depressive symptoms. Bhatti, Delaney, Poulin, and Hahn-Holbrook (2019) report in a study of 220 new mothers that variations on the oxytocin receptor gene interacted with father social support to predict postpartum depressive symptoms. GG homozygote, and to a lesser degree AG heterozygote women showed more postpartum depressive symptoms when father support was low, an association that did not emerge in homozygous AA women. Saxbe, Khaled, Horton, and Mendez (2019) highlight a different way in which findings in this literature need to be studied more integratively. In their study, they report differential findings depending on whether oxytocin levels were measured with or without extraction before the bioassay. These findings highlight that it is not only important to take into account the complexity of individuals' biopsychosocial experiences in the prediction of perinatal depressive symptoms, but to also carefully consider the way in which these experiences are measured.

A final paper by Mulligan, Flynn, and Hajcak (2018) is the first to address a combination of neural risk factors and self-report measures to predict pregnancy depressive symptoms. Findings indicate that blunted reward positivity, an event-related potential elicited in response to feedback indicating a monetary reward, a greater psychosocial pregnancy risk score, and lower annual income were associated with increased antenatal depressive symptoms.

In sum, the studies in this special issue further contribute to a growing body of empirical findings that indicate an understanding of the complexity of perinatal depressive symptoms is best achieved by an integrative approach that takes into consideration multiple biobehavioral and psychosocial measures of risk in the same study. Underlying this are complex biopsychosocial pathways and processes that are valuable to model and test over time. This argues further for multidisciplinary teams bringing strong psychological and biological expertise to collaborations. It is our hope that the studies in this special issue will stimulate further work of this type on this topic.

We close by thanking the authors of the manuscripts published here for their submissions, the reviewers of the articles for their helpful advice and guidance, the editor-in-chief and administrative staff at *Biological Psychology* and Elsevier for their support and patience, and last – but surely not least – the many pregnant women and new mothers who volunteered their time to participate in research studies.

Table 1
Studies of biological and psychosocial predictors of postpartum depressive symptoms published between 2014 and 2018.

Authors (Date)	Participants	Predictor(s)/Timing	Measure of PPD (Timing)	Control Variables/ Other Predictors in Final Model (asterisk indicates significance)	Did Measure(s) Predict PPD
Eapen et al. (2014)	127 Australian women; 52% Caucasian, 23% Asian, 10% Indian, 8% Arabic background Incl.: ≥ 18 yrs at delivery, < 38 wks' GA, singleton preg.	Biolog.: OXT in plasma Psychosoc.: Attachment style 28 (n = 97), 32 (n = 22) or 32-39 (n = 8) wks' GA; 3 mos pp	EPDS sx During preg., 3 mos pp	Maternal separation anxiety*	Simple comparisons: More pp sx w/ lower preg and pp OXT. Corr. not reported for attachment; Multivar.: Maternal separation anxiety mediates pos. association btw. anxious attachment and depressive sx; depressive sx mediate neg. assoc. btw. separation anxiety and OXT
Zelkowitz et al. (2014)	287 Canadian women, mostly married or living w/ partner, 93% breastfeeding at 7–9 wks pp Incl.: ≥ 18 yrs old, 38 wks' GA, singleton preg.; Excl.: birth ≤ 36 wks' GA, infant in NICU	Biolog.: OXT in plasma 12-14 and 32-34 wks' GA; 7-9 wks pp > 30 min following breastfeeding Psychosoc.: Psychosocial stress (ANRQ) sx and cat.: ≥ 23 (composite score of emotional support in childhood, mental health, SLEs, social support, marital relationship, abuse) 12-14 wks' GA	EPDS sx 12-14 and 32-34 wks' GA; 7-9 wks pp	Age, marital status, yrs of education* (sig. at 12-14 wks' GA), depressive symptoms at 12-14 wks' GA* (sig. at 32-34 wks' GA and 7-9 wks pp)	Simple comparisons: More psychosocial stress (sx, cat) w/ higher depressive sx at all time points; OXT n.s. High stress group: higher OXT w/ fewer depressive sx at 12-14 wks' GA and 7-9 wks pp. Low stress group: high depressive sx at 12-14 wks w/ high OXT at 33-35 wks' GA Multivar.: At 12-14 wks' GA and 7-9 wks pp, mothers w/ high stress (cat.) had fewer depressive symptoms. Moderation effect: Low OXT and high stress w/ higher EPDS. Simple comparisons: At 1 wk pp, TNF-α and IL-10 w/ EPDS scores among women w/ second degree or higher laceration Multivar.: At 1 mos, severe perineal laceration and perc. stress w/ higher EPDS in final model
Dunn et al. (2015)	155 U.S. women, 80.6% married, 76.8% Caucasian Incl.: 18-40 yrs old, < 36 wks' GA, non-smokers, w/ vaginal singleton birth, healthy preg. w/o restrictions, chronic illnesses or medications; birth w/o hemorrhage or transfusion, mother and infant left hospital within 72 hours; no sx of infection at visits	Biolog.: Proinflammatory (IL-6, IL-1β, TNF-α, INF-γ) and anti-inflammatory cytokines (IL-10) in plasma Psychosoc.: Perc. Stress (14-item PSS) sx 32-36 wks' GA, 1 wk, 2 wks, and 1, 2, 3, and 6 mos pp	EPDS sx 32-36 wks' GA, 1 wk, 2 wks, and 1, 2, 3, and 6 mos pp	Laceration (cat.: < 2nd degree vs. ≥ second degree)*, age, length of labor, personal or family hx of depression, proinflammatory to anti-inflammatory ratios	Simple comparisons: Higher dose of synthetic OXT at birth and more post-traumatic stress symptoms w/ higher EPDS sx Multivar.: Relationship status (partnered) and higher intrapartum synthetic OXT w/ more EPDS sx Childhood abuse and PPD interacted to modulate DNA methylation levels. Posthoc: Increased OXT receptor DNA methylation only among women without PPD Simple comparisons: Higher IL-6 and hs-CRP w/ higher EPDS sx and when ≥ 12. Analyses not reported for psychosocial variables. Multivar.: Higher IL-6, hs-CRP, more SLEs, and less partner support w/ EPDS ≥ 12
Gu et al. (2016)	386 Canadian women from two cohorts, 95.1% married Incl.: synthetic OXT information available. Excl.: < 18 yrs, birth before 35 wks' GA	Biolog.: Endogenous OXT in plasma, synthetic OXT administered at birth from medical records Psychosoc.: Relationship status, birth-related post-traumatic stress 2 mos pp, > 30 min after breastfeeding (OXT)	EPDS sx 2 mos pp	Years of education*	Simple comparisons: Higher IL-6 and hs-CRP w/ higher EPDS sx and when ≥ 12. Analyses not reported for psychosocial variables. Multivar.: Higher IL-6, hs-CRP, more SLEs, and less partner support w/ EPDS ≥ 12
Kimmel et al. (2016)	51 U.S. women w/ previous diagnosis of mood disorder, 70.6% African American, 29.4% Caucasian	Biolog.: OXT receptor DNA methylation Psychosoc.: Childhood abuse (single question) During preg.	DSM-IV criteria Each trimester, 1 wk, 1 mos pp	Antenatal depression*	Simple comparisons: Higher IL-6 and hs-CRP w/ higher EPDS sx and when ≥ 12. Analyses not reported for psychosocial variables. Multivar.: Higher IL-6, hs-CRP, more SLEs, and less partner support w/ EPDS ≥ 12
Liu et al. (2016)	296 Chinese pp women, 79.7% married, 60.1% high school or less; 92.9% Han Chinese Incl.: Singleton, full-term (≥ 37 wks' GA), live-born infant; Excl.: infant in NICU, depression before or during preg.	Biolog.: IL-6, hs-CRP in serum (fasting) 7 a.m. – 8 a.m. within 2 days of hospital admission for delivery Psychosoc.: Social network, social support, SLEs by interview	EPDS sx and ≥ 12	Age*, maternal or neonatal hospital readmission*, BMI*, unplanned preg., assisted delivery, income, education, breastfeeding	Simple comparisons: Higher IL-6 and hs-CRP w/ higher EPDS sx and when ≥ 12. Analyses not reported for psychosocial variables. Multivar.: Higher IL-6, hs-CRP, more SLEs, and less partner support w/ EPDS ≥ 12

(continued on next page)

Table 1 (continued)

Authors (Date)	Participants	Predictor(s)/Timing	Measure of PPD (Timing)	Control Variables/ Other Predictors in Final Model (asterisk indicates significance)	Did Measure(s) Predict PPD
Rebello et al. (2016)	235 women from Brazil, most living w/ partner (79.2%) and < 11 yrs education. Subsample (n = 41) in clinical trial to test efficacy of 3 rd trimester omega-3 supplementation. Incl: 20-40 yrs old, free of disease (except obesity), singleton preg.; Excl.: antidepressant drugs.	Biolog.: Adiponectin in plasma 6:50 a.m. - 7:50 a.m. and fasting during preg.; 10 a.m. - 11a.m., fasting not required, pp 5-13, 22-26, 30-36 wks' GA, 30-45 days pp Psychosoc.: Social support (Medical Outcome Study social support scale), marital status 5-13 wks' GA, social support also at 30-45 days pp	EPDS sx 5-13, 22-26, 30-36 wks' GA, 30-45 days pp	Preg. BMI, education, desire to become pregnant*, physical activity*, sleep duration, time elapsed after conception*, gestational age, omega-3 supplementation*	Marital status (not living w/ partner), w/ higher EPDS scores in univar. and multivar. models. Social support, and adiponectin n.s.
Ruyak et al. (2016)	111 U.S. women, mostly married (84.7%); 82.9% White, 5.4% Black, 3.6% Asian or Pacific Islander, 0.9% American Indian or Alaska Native, 7.2% Other Incl.: 18-40 yrs, < 36 wks' GA, no chronic illness or preg. complications, vaginal birth, discharge of mother and newborn within 72 hrs of birth.	Biolog.: Cortisol in saliva, IL-6 , TNF-α , leptin in plasma 32-36 wks' GA, 4 wks pp Saliva: +0, +30 min after waking, 11am, 4 pm, 8 pm; blood on following day btw. 3:30pm - 4:30pm Psychosoc.: Perc. stress (PSS) , marital status 32-36 wks' GA, perc. stress also at 4 wks pp Biolog.: HPA axis-related polymorphism (rs12565406) Psychosoc.: SLE in past 12 mos (Rosengren scale), partner support (single question: yes - no) 6 wks pp	EPDS sx 32-36 wks' GA, 4 wks pp	Prepreg. BMI	Path analysis shows direct effect of perceived stress w/ higher EPDS pp. Perceived stress predicted by prepreg. BMI, but no indirect effect emerged. Cortisol AUC and latent inflammation variable (IL-6, TNF-α, leptin) n.s.
Iliadis et al. (2017)	769 Swedish women	Biolog.: HPA axis-related polymorphism (rs12565406) Psychosoc.: SLE in past 12 mos (Rosengren scale), partner support (single question: yes - no) 6 wks pp	EPDS sx and ≥ 12 6 wks pp	Neuroticism*, depression history*, breastfeeding, poor sleep*, smoking	Simple comparisons: SLE and neuroticism higher in women w/ EPDS ≥ 12, no assoc. w/ genotype. Multivar.: Trend for genotype (p = .050) and SLEs (p = .058). Path analysis: neuroticism mediates relationship btw. genotype and depressive sx

ANRQ = Antenatal Risk Questionnaire, biolog. = biological, BMI = body mass index, btw. = between, cat. = categorical, EPDS = Edinburgh Postnatal Depression Scale, excl. = exclusion, GA = gestational age, HPA = hypothalamus pituitary adrenal, hr(s) = hour(s), hs = high sensitivity, IL = interleukin, incl. = inclusion, mo(s) = month(s), multivar. = multivariate, NICU = Neonatal Intensive Care Unit, n.s. = not significant, OXT = oxytocin, perc. = perceived, pp = post partum, PSS = Perceived Stress Scale, preg. = pregnancy, psychosoc. = psychosocial, SLE = stressful life events, sx = symptoms, univar. = univariate, w/ = with, wk(s) = week(s), w/o = without, yr(s) = year(s).

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