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Is Postpartum Depression a Distinct Disorder?

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Abstract The nosology of postpartum depression (PPD) is controversial. We review the evidence and arguments for and against the recognition of PPD as a distinct disorder and discuss the etiopathogenic and diagnostic validity of PPD as a distinct disorder, including its utility and indications for further research. Although multiple epidemiological and clinical studies have found that depression is more common following childbirth than at other times in a woman's life, there is conflicting evidence for the validity of PPD as a distinct disorder. PPD is likely to be a complex phenotype, encompassing several disorders with different disease pathways. It is plausible that for a sub-group of vulnerable women, childbirth triggers episodes of depression. However, even within this group, the mechanisms underpinning the mood disturbances are likely complex and heterogeneous. The distinction between depression occurring in the perinatal period and depression at other times is important for both research and clinical practice. Research should differentiate between episodes that begin during pregnancy and postpartum, as the pathogenetic factors involved may differ and require specialized treatment.

Keywords Postpartum depression · Pregnancy · Nosology · Validity · Timing · Phenotype

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Introduction

The nosology of postpartum depression (PPD) is controversial. The current diagnostic systems employed by the American Psychiatric Association and the World Health Organization (WHO) do not consider PPD as a separate disorder but rather a subtype of major depression. For example, in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), PPD is coded as a major depressive episode and the relationship with childbirth is recorded by applying the specifier "with peripartum onset," defined as onset of symptoms during pregnancy and/or within the first 4 weeks postpartum [1]. This is a change from the previous DSM-IV, which only included postpartum onset within the first 4 weeks of childbirth [2]. The 10th revision of the International Statistical Classification of Diseases and Related Health Problems (ICD-10) limits the separate classification of any postpartum psychiatric disorder to episodes occurring within 6 weeks postpartum that cannot be classified elsewhere (code F53, "mental and behavioral disorders associated with the puerperium, not elsewhere classified") [3]. In contrast, the WHO recommends using the F53 code only when "unavoidable." According to the WHO, the F53 diagnostic category does not reflect differences in depressive episodes occurring in the postpartum period versus episodes occurring at other times but is a mechanism to address logistical issues associated with collecting information about puerperal illness in many developing countries [3].

The problematic considerations surrounding the nosology of PPD reflect the uncertainty about the definition and classification of psychiatric disorders in general [4, 5]. Our knowledge of psychiatric disorders cannot meet the high epistemic standards of scientific realism *tout court* [6], leading to criticism of current diagnostic systems. These concerns have led the US National Institute of Mental Health (NIMH) to develop

the Research Domain Criteria (RDoC) framework that supports a dimensional rather than categorical approach to psychiatric research [7]. Cuthbert and Insel [8], exploring the implications of the RDoC for women's mental health, suggested that research should focus on the response to hormonal changes of neuronal networks rather than the clinical picture.

Here, we will review the evidence and the arguments for and against the recognition of postpartum depression as a distinct disorder in the light of the general limitations of psychiatry nosology. The discussion is organized in three sections: first, we will discuss the etiopathogenic and diagnostic validity of PPD as a distinct disorder, then its utility,[1] and conclude with some indications for further research.

Validity of Postpartum Depression as a Distinct Disorder

Although the evidence is not as robust as for bipolar disorder, multiple epidemiological and clinical studies have found that depression is more common following childbirth than at other times in a woman's life suggesting an etiological link. Population-based studies conducted in Scandinavia, using register-based hospital admission data or questionnaires and taking into account possible confounders, have found that the risk of depression in women is significantly increased in the postpartum period compared to other time periods [10, 11]. A retrospective clinical study examining the life-time course of mood disorders also found that depressive episodes were overrepresented in the postpartum period in parous women with history of major depression [12•].

Recent studies, however, have questioned the paradigm of a firm temporal relationship between childbirth and depression. Wisner et al screened 10,000 women after childbirth for PPD and found that only 40 % of those who screened positive had a postpartum onset [36•]. Similarly, the Postpartum Depression: Action Towards Causes and Treatment (PACT) Consortium recently conducted a latent class analysis study on over 6000 women in Europe and the USA and estimated that the onset of severe PPD (mean Edinburgh Postnatal Depression Scale score 20.1) occurred before childbirth in 67 % of cases [13•]. According to these studies, in many cases of PPD, the onset of symptoms began in pregnancy and continued into the postpartum period. Therefore, the timing of onset becomes an important area to examine and raises interesting questions. How are women that develop symptoms of depression during pregnancy different from those that experience postpartum onset? What are the underlying biological mechanisms that characterize the subset of women in whom childbirth triggers an episode of major depression with onset in the immediate postpartum? Moreover, social and psychological factors must also be considered.

Timing of Onset as a Clue Into the Pathogenesis of the Childbirth Trigger

The association between depressive episodes that occur during pregnancy versus the postpartum period is particularly interesting and challenges many assumptions about the etiology PPD. In the PACT study, the cases of PPD with onset of the symptoms during pregnancy were the most severe and more likely to be associated with delivery complications and suicidal ideation, while episodes beginning within 4 weeks postpartum had more complications during pregnancy [13•]. Clinical studies have also provided evidence of the importance of timing: compared to onset of depression during pregnancy, postpartum onset is more likely to be associated with bipolarity [14] and recurrent episodes of PPD [15].

In summary, timing of onset remains an area of controversy and requires further investigation into the biological and psycho-social determinants underlying perinatal depression.

Biological Validators

Dramatic and unique biological changes occur after delivery. The supra-physiologic levels of circulating hormones (i.e., gonadal steroids, cortisol) observed during pregnancy decline abruptly in the postpartum period [16]. Surprisingly, very few studies have robustly investigated the biological underpinnings of childbirth triggering a depressive episode. For example, Bloch et al. studied a group of euthymic women with histories of PPD and matched controls, who underwent laboratory manipulation of the hormonal changes that characterize pregnancy and postpartum. First, the women were given a GnRH agonist to suppress endogenous production of gonadal hormones; second, supra-physiologic levels of gonadal steroids were administered to mimic those observed during pregnancy (add-back phase); and third, the gonadal steroids were abruptly withdrawn as observed immediately postpartum. While controls were not affected by the hormonal challenge, over 60 % of women with history of PPD developed significant mood symptoms during the withdrawal phase, suggesting that for a sub-group of women who are more sensitive to hormonal changes, childbirth represents a specific biological trigger [17]. Interestingly, although the severity of the symptomatology in women with history of PPD peaked during the withdrawal phase simulating the hormonal conditions of the immediate postpartum, significant symptoms of depression

[1] The term "validity" is broad and not univocally defined, especially in psychiatry. The distinction between validity and utility represents an extension of the model presented in Kendell and Jablensky's seminal paper. Here, it is used with the sole intent of organizing the arguments. An examination of the philosophical grounds of psychiatric nosology goes beyond the scope of this paper. The authors refer the readers to [9] for an in-depth discussion.

also developed at the end of the add-back phase, when progesterone and estradiol were administrated to simulate the sustained elevation of gonadal steroids of pregnancy. These results corroborate the evidence that in many cases, the onset of a full episode of depression after childbirth may start during pregnancy and that the relationship between hormonal changes and PPD is more complex than previously thought.

There is also a dearth of information on the genetic vulnerability to a postpartum trigger of depression. The lack of consensus on the definition of the phenotype, especially the length of the acute postpartum onset, has probably hindered research in this area. For example, according to a family study, the contribution of familial factors on the risk of postpartum depression maximizes for episodes occurring within 6–8 weeks after delivery [18], implicating that the current DSM-5 criterion for postpartum onset is too narrow. Although the contribution of genetic factors for PPD is similar to that of general major depression (13–52 % [19, 20•]), three different studies, using different methodologies and independent samples, have suggested differences in the genetic contributions to PPD versus depression occurring at other times. First, a twin registry study conducted in Australia found a stronger genetic association between postpartum depressive symptoms (PPDS) and neuroticism (r=0.33) than between PPDS and depression at other times in a woman's life (r=0.17) [19]. Second, a recent Swedish study conducted on 3427 female twins who completed a self-rated scale for screening PPD and a population-based cohort of 580,006 sisters with clinical diagnosis of PPD found that 14 % of the variance (one third of the total hereditability estimate) of PPD was explained by genetic factors not shared with non-puerperal major depression [20•]. Third, by applying polygenic risk scores of bipolar disorder and major depression from the Psychiatric Genomics Consortium datasets, Byrne et al. found a stronger genetic overlap between bipolar disorder and self-reported PPD, than between bipolar disorder and major depression outside the postpartum period [21]. However, the relatively small sample size of this study (1420 cases and 9473 controls) and consequent large standard errors limited the interpretation of the findings.

Psychological and Social Determinants

Transition into motherhood is a major life event, with profound psycho-social implications. The arrival of an infant brings social and financial strains, especially for low-income families with little socio-economic support. The birth of a child provides significant financial stress to low-income families. However, financial and social problems are risk factors for a broad range of illnesses [22], and the link between depression and poor socio-economic status is not specific for depressive episodes occurring after childbirth. The research to date in this area is sparse, and studies comparing the effect of poor socio-economic status between women with PPD and women with depression occurring at other times of life are needed in order to establish the specificity and magnitude of social determinants in depression.

Psychological theories of PPD have been widely explored, and evidence seems to converge on an association with psychological traits such as neuroticism [23], insecure attachment styles [24, 25], and negative cognitive schemata [26]. It is however not clear whether childbirth specifically triggers these traits [24] or acts as a generalized highly stressful life event [25–27].

In summary, PPD is a complex nosological entity; encompassing syndromes with likely different disease pathways. For some women, childbirth may act as a specific trigger, but for many women with depression after delivery, the onset of symptoms may have started in pregnancy and the pathogenesis may be more complex and may include factors not specifically associated with childbirth.

Utility of PPD as a Distinct Disorder

Despite the controversies surround nosology and etiology of PPD, there are multiple reasons to classify it as a separate nosological entity based on clinical, social, and research considerations.

Clinical Guidelines

Because of the complex biological, social, and psychological context associated with pregnancy and childbirth, the identification and treatment of women with PPD requires specific considerations that usually do not apply to recurrent major depression outside of the perinatal period. Separate ad hoc guidelines for the detection and treatment of perinatal mental disorders, including PPD, have been created [28, 29]. Over half of the cases of PPD go unrecognized, and many women with a correct diagnosis do not receive adequate treatment [30]. Underdiagnosis and lack of proper treatment have adverse consequences on the woman, on the child, and on the entire society [30]. Because of the high incidence and detrimental consequences, universal screening of puerperal depression has been recommended in several countries [28, 31]. The PPD diagnostic label helps by acknowledging that common neurovegetative symptoms for general major depression, such as somatic symptoms of changes in sleep and weight, may be difficult to interpret in the postpartum period [32]. For example, the Edinburgh Postnatal Depression Scale [33], one of the most widely used screening tools for PPD, does not assess for insomnia and weight changes, as these symptoms are experienced by most women in the perinatal period and do not necessarily reflect an underlying psychiatric disorder.

The therapeutic needs of women with PPD also differ from those of women with depression at other times and often require a multidisciplinary approach [28, 34]. For example, physicians need to consider the implications of psychotropic medications during breastfeeding and to acknowledge and treat the mother in the context of the dyadic relationship with the baby.

Prognosis and Outcomes

The postpartum onset also carries information about prognosis and treatment outcomes. Evidences from both epidemiological and clinical studies have suggested an association between a bipolar diathesis and PPD. A population-based study with a 15-year follow-up calculated that the risk of diagnostic conversion to bipolar disorder is over fourfold higher for women admitted to the hospital in the postpartum than for those admitted at other times [35]. In a clinical sample of 60 women with resistant PPD, over half of them had a diagnosis of bipolar disorder, initially missed by clinicians [14]. In a sample of 10,000 women screened at 4–6 weeks postpartum for PPD, 22.6 % of those who screened positive had bipolar disorder [36•]. Rates of conversion from unipolar to bipolar disorders are also increased in the postpartum period. In a longitudinal clinical study, over 6 % of 90 women with recurrent major depression had a hypomanic episode following childbirth, a much higher risk than that observed outside the postpartum period [37]. All together, the evidence of a link between childbirth and bipolar disorder invites clinicians to be particularly careful in the evaluation, treatment, and follow-up of women with PPD, as they have an increased risk of suffering from a bipolar diathesis.

In this context, it is important to make some distinctions between PPD and postpartum psychosis. Postpartum psychosis is a psychiatric emergency characterized by severe manic or psychotic episodes occurring in the first weeks after childbirth. It has been recommended to consider psychotic depression after childbirth as a manifestation of bipolar disorder, even when manic symptoms or history of mania are absent, and to avoid treatment with an antidepressant [38]. Although the majority of studies focus either on unipolar perinatal depression or on postpartum psychosis, non-psychotic PPD is common in women with bipolar disorder affecting over one in four deliveries [12•]. The treatment of these women requires special consideration, especially because of the risk of switch to mania if antidepressants were used [14, 39]. In women with bipolar disorder, episodes of mania and psychosis are more prevalent after childbirth than at other times in life; however, in marked contrast to unipolar depression, episodes of bipolar depression are not more common postpartum than at other times [12•, 40].

Administrative Validity

PPD has also "administrative validity" within the healthcare and research funding systems [41]. The label of PPD legitimates the need for research. Many funding bodies, in fact, are often required to defend the validity of the disorder addressed by the proposed study [41]. Similarly, the recognition of the link with childbirth allows for better organization of healthcare systems to support the women and their families and has led to the development of ad hoc, specialized services. The recognition of perinatal disorders as separate entities has, for example, allowed not only research into the causes of PPD but also an estimation of their costs and impact on the society. The United Kingdom has pioneered this area, by including suicide as a cause of maternal death in the Confidential Enquiries into Maternal Deaths [42] and by producing the first estimates of the costs of perinatal mental health [30], subsequently increasing awareness of PPD and providing guidance for service implementation. For example, this work has demonstrated that maternal suicide is one of the great causes of postpartum mortality [42] and that the cost to the public sector for untreated perinatal mental illness is more than four times the expenditure needed to improve the services to an adequate level suggested by national guidelines [30]. Importantly, "administrative validity" is critical in countries with an insurance-based healthcare system, where reimbursement is driven by diagnostic coding.

Research Into the Causes and Treatment

The identification of the temporal relationship between onsets of depression with childbirth can benefit research into the causes and treatment of PPD. In this context, the definition of the period that should be considered as "postpartum" is a matter of debate. While clinical practice focused on screening and treatment may warrant a broader definition of up to 6 months to 1 year postpartum, this becomes problematic for biological and genetic research focused on an acute postpartum trigger (i.e., onset limited to the first 6–8 weeks following childbirth) [32]. A later cut-off would increase the heterogeneity and potentially increase the number of cases not etiologically related to childbirth.

Indication for Further Research

The study of women who suffer from depression during pregnancy or postpartum presents a unique opportunity to investigate the etiopathogenesis of the categorical diagnosis of PPD by exploring and clarifying the issues

related to timing of onset and duration of the acute postpartum period. Moreover, in order to test the hypothesis that PPD is a distinct disease entity, it becomes mandatory to include samples of parous women with major depression outside the postpartum period. This would allow for an evaluation of whether the abnormalities observed in PPD are specific or not to the perinatal period or if they extend to all cases of major depression, regardless of their relationship to delivery.

Applying the RDoC approach, a promising strategy would be to investigate the vulnerability to affective switching triggered by reproductive events. It has been hypothesized that neurosteroids, in particular allopregnanolone, a metabolite of progesterone, have an important role in mediating the affective dysregulation observed in some women with PPD [43]. The identification of factors that contribute to the individual susceptibility to hormonal changes remains to be established and may lead to the stratification of women at risk and to better strategies to prevent and treat mood disorders associated with reproductive events. Important insights on individual susceptibility can be provided by molecular genetic studies, especially genome-wide single nucleotide polymorphisms and expression analyses. Although the use of omics approaches has focused mainly on genetics, advances in mass spectrometry technology now enable global profiling of the metabolites involved in disorders and can advance our knowledge by detecting the alterations in the biochemical pathways in women with PPD.

Conclusion

It is common opinion among experts that psychiatric nosology should be "grounded in the matrix of the empiric world" [6] and based on evidence and clinical utility [44].

Currently, there is insufficient evidence to classify PPD as a separate disorder. PPD is likely to be a complex phenotype, encompassing several disorders with different disease pathways. It is plausible that for a sub-group of women, childbirth triggers episodes of depression. However, even within this group, the mechanisms underpinning the mood disturbances are likely complex and heterogeneous.

Nevertheless, the distinction between depression occurring in the peripartum period and depression occurring at other times is important for both research and clinical practice. Research should differentiate between episodes occurring at different stages of pregnancy and after delivery, as the pathogenetic factors involved are likely to differ and may require specialized treatment.

Compliance with Ethics Guidelines

Conflict of Interest Arianna Di Florio declares that she has no conflict of interest.

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Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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