Journal of Affective Disorders 224 (2017) 2–9

Contents lists available at ScienceDirect

Journal of Affective Disorders

journal homepage: www.elsevier.com/locate/jad

Perinatal nutrition interventions and post-partum depressive symptoms

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ARTICLE INFO

Keywords:
Depression
Nutrition
Postpartum
Diet
Intervention

ABSTRACT

Background: Postpartum depression (PPD) is the most prevalent mood disorder associated with childbirth. No single cause of PPD has been identified, however the increased risk of nutritional deficiencies incurred through the high nutritional requirements of pregnancy may play a role in the pathology of depressive symptoms. Three nutritional interventions have drawn particular interest as possible non-invasive and cost-effective prevention and/or treatment strategies for PPD; omega-3 (n-3) long chain polyunsaturated fatty acids (LCPUFA), vitamin D and overall diet.

Methods: We searched for meta-analyses of randomised controlled trials (RCT's) of nutritional interventions during the perinatal period with PPD as an outcome, and checked for any trials published subsequently to the meta-analyses.

Results: Fish oil: Eleven RCT's of prenatal fish oil supplementation RCT's show null and positive effects on PPD symptoms. Vitamin D: no relevant RCT's were identified, however seven observational studies of maternal vitamin D levels with PPD outcomes showed inconsistent associations. Diet: Two Australian RCT's with dietary advice interventions in pregnancy had a positive and null result on PPD.

Limitations: With the exception of fish oil, few RCT's with nutritional interventions during pregnancy assess PPD.

Conclusions: Further research is needed to determine whether nutritional intervention strategies during pregnancy can protect against symptoms of PPD. Given the prevalence of PPD and ease of administering PPD measures, we recommend future prenatal nutritional RCT's include PPD as an outcome.

1. Introduction

Postpartum depression (PPD)[1] is the most prevalent mood disorder associated with childbirth. Clinically it is not different to depression at other times and symptoms include mood disturbances (such as sadness, loss of pleasure, guilt or worthlessness), sleep disturbances (not related to the baby), appetite disturbances or weight loss, and suicidal ideation. Systematic reviews report that 19.2% of women experience depression within 12 weeks of birth (Gaynes et al., 2005) and 10–20% of women experience PPD within the first year, regardless of race, parity, age, education and socioeconomic status (Gavin et al., 2005), with symptoms persisting beyond the first year in 8% of effected mothers (Dennis et al., 2012).

Minimising PPD is particularly important due to the implications for the mother and child. Depression impairs social and psychological functioning and there is a body of evidence to suggest a deleterious effect of PPD on child development outcomes (Conroy et al., 2012; Field, 2010; Goodman et al., 2011; Zhu et al., 2014). A number of caregiving activities are considered to be adversely impacted by PPD, including infant feeding practices, sleep routines and routine infant and child health visits (Field, 2010), as well as disturbances to the mother-infant interaction such as less sensitivity or responsiveness to infants (Field, 2010). A meta-analysis of 193 studies found that maternal depression at any time is associated with increased offspring internalising and externalising behaviours and psychopathology (Goodman et al., 2011). International experts acknowledge that maternal depression has long-term adverse effects on child development (England and Sim, 2009). Public health preventive strategies to lower the prevalence of PPD would have wide ranging benefits for mothers, children and families.

PPD is thought to be multifactorial, rather than due to a single causative factor (Beck, 2001; Bobo and Yawn, 2014), making preven-

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[1] 125(OH)D: vitamin D, DHA: docosahexaenoic acid, EPDS: Edinburgh Postpartum Depression Scale, LCPUFA: long-chain polyunsaturated fatty acid, n-3: omega-3, PPD: postpartum depression, RCT: randomised controlled trial.

http://dx.doi.org/10.1016/j.jad.2016.12.014
Received 1 July 2016; Accepted 17 December 2016
Available online 18 December 2016
0165-0327/

tion problematic. However, nutrition is considered one of the likely contributing factors and is modifiable. Pregnancy and lactation are a time of particular stress on a woman's nutrient reserves, and nutrient deficits incurred may increase the likelihood of depression in the postpartum period. Nutritional interventions during the perinatal period may offer a simple and cost effective strategy to prevent nutritional deficiencies and hence reduce the prevalence of PPD. Three nutritional strategies that have drawn particular interest for depressive symptoms are n-3 LCPUFA, vitamin D and overall diet.

This review evaluates possible nutritional interventions that may be implemented as public health strategies for effective prevention of PPD symptoms by summarising meta-analyses of randomised controlled trials (RCT's), as the highest level of evidence, of omega-3 (n-3) long-chain polyunsaturated fatty acids (LCPUFA), vitamin D and overall diet interventions during pregnancy and/or lactation with PPD outcomes. We also searched for any relevant RCT's with PPD that have been published following the included reviews.

1.1. Omega-3 LCPUFA

There has been considerable interest in the role n−3 LCPUFA in mental health in the general population, however little is known about their effects on maternal mental health. Pregnancy and the postpartum period is a time when n-3 LCPUFA supply is particularly significant. The metabolic demand for n-3 LCPUFA, in particular docosahexaenoic acid (DHA) is increased as maternal tissue stores are used for the developing fetus (Makrides and Gibson, 2000). Metabolic and post-mortem studies indicate that the fetus accumulates an average of 67 mg of docosahexaenoic acid (DHA) per day during the last trimester of pregnancy (Innis, 2003). Modern western diets are low in n-3 LCPUFA and this level of DHA exceeds the intake of many pregnant women highlighting a potential dietary insufficiency. During the postpartum period there is further risk of DHA deficiency as depletion of maternal serum DHA declines following delivery (Otto et al., 2001).

Alterations in fatty acid metabolism and the composition of phospholipids in serum and membranes have been implicated in the pathophysiology of depression in the general population. Observational studies in the general population have shown that levels of DHA in serum and cell membranes are lower in people who suffer from depression compared to healthy controls (Edwards et al., 1998). Studies during the perinatal period also suggest an association between decreased maternal n-3 LCPUFA intake during pregnancy (Gow and Hibbeln, 2014) (or low or DHA status following delivery) and the occurrence of postpartum depression (De Vriese et al., 2003; Golding et al., 2009). Epidemiological studies observing the association of higher intakes of n-3 LPCUFA during pregnancy have appeared promising and suggest a reduction of depressive symptoms in the postnatal period (Hibbeln, 1998; Oken and Belfort, 2010), however these studies are unable to establish causality because of the difficulty in adjusting for complex confounding factors (Lawlor et al., 2004).

Emerging evidence from randomised trials indicates that DHA interventions in patients with major depression improve depressive symptoms compared with control (Marangell et al., 2003; Stoll et al., 1999; Su et al., 2003). A recent systematic review and meta-analysis including 35 randomised controlled trials (RCTs) concluded that 'the evidence available provides some support of a benefit of n-3 PUFAs in individuals with depressive illness but no evidence of any benefit in individuals without a diagnosis of depressive illness' (Appleton et al., 2010).

Supplementation of pregnant women with n-3 LCPUFA has been proposed as a potential strategy to prevent and/or treat PPD. The effect of perinatal n-3 LCPUFA supplementation on PPD has been studied in 11 RCTs (Table 1). Supplementation of women has occurred either 'during pregnancy' (antenatally), 'postpartum' or a combination of both time periods. Participant inclusion criteria varies greatly with trials including women with 'major depressive disorder' (Freeman et al., 2008; Rees et al., 2008; Su et al., 2003), 'current PPD'(Freeman et al., 2006), 'at risk of PPD'(Kaviani et al., 2014; Mozurkewich and Klemens, 2012) or apparently healthy women (Doornbos et al., 2009; Krauss-Etschmann et al., 2007; Llorente et al., 2003; Makrides and Gibson, 2000; Mattes et al., 2009). Nine of these trials did not find a significant benefit of n-3 LCPUFA supplementation administered either antenatally, postpartum or a combination of both periods. Two small trials including depressed women reported a significant reduction in depression symptoms in the n-3 LCPUFA group (Kaviani et al., 2014; Su et al., 2003). The majority of these trials conducted have been of low-to-moderate quality, mainly due to small sample size (< 100) and failure to adhere to Consolidated Standards of Reporting Trials guidelines (Jans et al., 2010). Perhaps the strongest evidence available comes from the largest trial to date to investigate n-3 LCPUFA as a preventative strategy. This double blind RCT included 2399 women supplemented with DHA-rich fish oil or placebo from 20 weeks gestation until delivery (Makrides and Gibson, 2000)Results demonstrated that there was no statistically significant benefit of DHA supplementation during pregnancy in preventing depressive symptoms in the first six months postpartum (Makrides and Gibson, 2000).

As systematic reviews are generally considered to provide the best evidence to answer a research question we searched the databases and identified nine reviews of the literature examining the effect of n-3 LCPUFA supplementation in the perinatal period on PPD. Six reviews were narrative and three reviews combined studies with heterogeneous inclusion criteria, timing and duration of intervention in meta-analyses (Table 2). One review including meta-analysis of 620 depressed or non-depressed women with at least 4 weeks of n-3 LCPUFA supplementation (during pregnancy, the postpartum period or both) showed no significant effects of n-3 LCPUFA compared to placebo. A recent Cochrane review examining the effect of dietary supplements for the prevention of postpartum depression (Miller et al., 2013) included one trial that compared n-3 LCPUFA's DHA and eicosapentaenoic acid to placebo in women at high risk of PPD. This trial including 126 women found no effect of either DHA or eicosapentaenoic acid on the prevention of PPD in at risk women. The only other review with meta-analysis included pregnant women as a subgroup of their report on the effects of n-3 fatty acids on depressive disorders (Grosso et al., 2014). The authors reported inconclusive results from RCTs including women with a major depressive disorder (Freeman et al., 2006; Rees et al., 2008; Su et al., 2003) and RCTs including apparently healthy women (Doornbos et al., 2009; Llorente et al., 2003; Mozurkewich et al., 2013) (primary prevention). Not all reviews were systematic or comprehensive of the relevant studies (as illustrated in Table 2) and the inclusion of small scale studies with high risk of bias combined with the heterogeneity of available RCTs of perinatal n-3 LCPUFA supplementation prevents consistent conclusions.

There is considerable biological plausibility to support a role of n-3 LCPUFA in PPD and some evidence for observational studies however the current evidence from RCTs is inconclusive. RCTs reporting the effects of perinatal n-3 LCPUFAs on PPD has increased in recent years though remains difficult to summarise because of small sample size, low-to-moderate quality, and considerable heterogeneity between studies. Overall, the evidence shows that supplementation with marine oil or n-3 LCPUFA is safe in the perinatal period and is generally well tolerated. However, more work is needed to understand the optimal maternal n-3 LCPUFA status for the prevention and/or treatment of PPD. Questions remain in regard to optimal dose (amount and type of n-3 LCPUFA i.e. DHA vs eicosapentaenoic acid) and timing of supplementation as well as which sub-populations may benefit most. Currently, there is not enough evidence to support the routine use of marine oil, or other prostaglandin precursor, supplements in the perinatal period to reduce the risk of PPD.