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The effects of paternal depression on child and adolescent outcomes

A systematic review

Citation for published version:
Sweeney, S & Macbeth, A 2016, 'The effects of paternal depression on child and adolescent outcomes: A systematic review', *Journal of Affective Disorders (JAD)*. https://doi.org/doi:10.1016/j.jad.2016.05.073

Digital Object Identifier (DOI):
doi:10.1016/j.jad.2016.05.073

Link:
Link to publication record in Edinburgh Research Explorer

Document Version:
Peer reviewed version

Published In:
Journal of Affective Disorders (JAD)

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Download date: 12. Oct. 2024

Title: The effects of paternal depression on child and adolescent outcomes: A systematic review

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Running head: Paternal depression and offspring outcomes

Conflicts of Interest

Dr MacBeth has previously received funding from an NHS Research Scotland Career Researchers Fellowship to develop a programme of research into the mental health in the children of parents with complex mental health difficulties.

Disclosure

The authors have no current disclosures pertaining to this manuscript.

Contributors

Both authors designed the review and wrote the protocol. SS managed the literature searches and review. SS wrote the first draft of the manuscript. Both authors contributed to revisions and have approved the final manuscript.

Role of Funding Source

There was no external funding involved in this review.

Acknowledgements

None.

Abstract

Background: Paternal depression has been associated with suboptimal developmental outcomes in offspring. We sought to systematically review the research evidence from prospective studies for an association between paternal depression and child adolescent emotional and behavioural outcomes. We also reviewed potential mediators of this association and sources of methodological bias.

Methods: A systematic review was conducted using the following databases: Medline, EMBASE, PsycINFO and Google Scholar. Reference lists of the included papers were also searched.

Results: Twenty-one studies were included in the review. Findings suggested that paternal depression does negatively impact upon offspring development. This impact is observable when paternal depression is present in the antenatal and postnatal stages and during offspring adolescence. The strength of this association is strongly reliant upon a number of contextual mediators, namely; paternal negative expressiveness, hostility and involvement and marital conflict. A quality assessment rating showed the studies were relatively strong methodologically.

Limitations: Heterogeneity regarding method of assessment and the magnitude and timing of exposure hinder attempts to make strong conclusions regarding the trajectory of paternal depression and its effects on child and adolescent outcomes.

Conclusions: Paternal mental health screening during pregnancy is necessary in order to identify and prevent depression negatively impacting offspring functioning. Including both parents in this process should encourage the alleviation of the environmental mediators which dominate the negative association outlined within the review. Research examining gene-environment interaction is necessary to uncover more accurate details regarding paternal depression and subsequent offspring vulnerability.

Key words: Paternal, depression, child, adolescent, development

Highlights

- Paternal depression is associated with negative offspring outcomes.
- This association is subject to mediating factors to transmit risk.
- Potential mediators were marital conflict and paternal parenting behaviors.
- Internalizing behaviours were a more common outcome than externalizing behaviours.

Introduction

Depression is a significant mental health issue, commonly associated with functional impairments across the lifespan. Evidence suggests that depression in childhood and adolescence is associated with a heightened risk of psychiatric hospitalization, recurrent disorder and elevated risk of attempted suicide in adulthood (Harrington et al. 1990, Harrington et al. 1994). Depression is also highly comorbid and commonly associated with increased risk of alcoholism and anxiety disorders (Boden & Ferguson, 2011; Moffitt et al. 2007). Offspring of depressed parents are frequently exposed to an elevated risk of externalizing and internalizing problems, lower neurobiological development, social and academic difficulties and subsequent development of adult psychopathology (Goodman & Gotlib, 1999; Natsuaki et al. 2014; Weissman et al. 2006). Indeed, children of depressed parents are almost three times as likely to experience a lifetime episode of depression as offspring of non-depressed parents (Lieb et al. 2002; Weissman et al. 1997). These negative associations between perinatal maternal depression and adverse child developmental outcomes have been widely studied (Beck, 1998; Goodman et al. 2011). However, the literature has tended to focus on the impact of maternal depression on offspring outcomes. There has been a recent increase in empirical research investigating the effects fathers' depression can have on offspring outcomes, with some evidence for an effect. However, the magnitude, mediators and timing is poorly understood (Kane & Garber, 2004; Kvalevaag et al. 2013), particularly with regard to potential critical periods.

Timing of Paternal Depression and Offspring Outcomes

Paternal depressive symptoms during pregnancy have been shown to increase likelihood of excessive infant crying (van den Berg et al. 2009). From a developmental perspective, excessive infant crying has been associated with higher levels of negative reactivity and lower emotional regulation, as well as hearing difficulties as development continues (Hestbaek et al. 2014; Stifter & Spinrad, 2002). This association has also been linked with higher scores on the emotional symptoms scale when compared with children of fathers without depression at that stage (Ramchandani et al. 2008) and a higher risk of internalizing and externalizing problems (Kane & Garber, 2009). Research on postnatal depression and child

development is more abundant and results indicate a strong association, with increased risk of emotional and behavioural problems in school-aged children (Weitzman et al. 2011). These heightened risks have also been linked with poorer academic performance (Doctoroff et al. 2006; Metsapelto et al. 2015), prosocial behaviour and peer problems (Davé et al. 2008), as well as internalizing symptoms (Ramchandani et al. 2005) which are highly comorbid (Cummings et al. 2014). Moreover, fewer paternal depressive symptoms can act as a protective factor or "buffer" for offspring when maternal depression is present in the family environment (Melrose, 2010). Similar results exist regarding the association of paternal depression and adolescent functioning with increased likelihood of anxiety and depressive symptoms and Major Depressive Disorder (Klein et al. 2005; Reeb et al. 2010; Reeb et al. 2015). Functional impairments like these make adolescents 6 times as likely to receive a diagnosis of a disorder in adulthood when compared with their typically developing peers (Hofstra et al. 2002).

Mechanisms of Risk

Although genetic risks have been identified in the transmission of depression (Merikangas et al. 2002), gene environment interactions may constitute important moderators of risk for developing children (Caspi et al. 2003). Cultural and societal changes may also be relevant as fathers spend more direct time with their children than historically (Cabrera et al. 2000) with a corresponding impact on child development (Ramchandani & Psychogiou, 2009). Being raised by a depressed father may thus constitute an 'environmental' risk for offspring development (Natsuaki et al. 2014). Epidemiological research suggests that approximately 10% of fathers are susceptible to depression in the prenatal and postpartum stage with the highest risk existing in the 3-6 month postpartum period (Paulson et al. 2010).

There is also evidence that suggests depressed mothers are more likely than non-depressed mothers to practice poor parenting behaviours and have negative interactions with their children (Hops, 1995). Depressed mothers are also less likely to promote safety behaviours that may prevent injury and harm among their children and are more likely to use corporal punishment (Chung et al. 2004; Mclennan & Kotelchuck, 2000). In light of this, it has been proposed that the impact of depression on parenting behaviours (Middleton et al. 2009; Ramchandani & Psychogiou, 2009)

could be a mediating mechanism for transmission of risk to offspring. Indeed, an impaired caregiver is considered to be a major childhood risk in the Adverse Childhood Experience (ACE) literature. ACE literature has found that parental mental illness and subsequent parenting behaviours contribute to an increased risk of offspring developing a range of medical and psychological disorders (Anda et al. 2002; Chartier et al. 2010). In addition, the ACE literature indicates that exposure to stress has a greater detrimental effect of developmental outcomes on a dose-equivalent basis, and if exposure to stress occurs in the first 2 -3 years of life (e.g. Shonkoff et al., 2012; Kelly-Irving et al., 2013). This corresponds with evidence that the parenting a child or adolescent receives can significantly impact on emotional and behavioural development (Bayer et al. 2008; Stormshak et al. 2000). As parenting behaviours change due to depressive symptoms, hostility and marital conflict may increase, whilst the quality and frequency of father-child interaction decreases, which consequently impacts on offspring functioning (Davé et al. 2008; Franck & Buehler 2007; Gutierrez-Galve et al. 2015; Middleton et al. 2009; Wilson & Durbin, 2010). Parents who exhibit harsh parenting techniques or display low levels of warmth towards their children have been found to increase the risk of developing depressed mood and conduct problems for their offspring (Hipwell et al. 2008; Young et al. 1995). Moreover, research indicates that depression among fathers increases the likelihood of spanking their one-year-old infants (Davis et al. 2011) and they are more likely to express aggravation and stress in parenting, when compared with non-depressed fathers (Bronte-Tinkew et al. 2005). This is of significance as emotional and behavioural difficulties can manifest themselves because of severe discipline from the caregiver towards the child (Bayer et al. 2008). Depressive disorders may also impact on quantity and quality of paternal-offspring interaction time a father spends with their child (Paulson et al. 2006; Ramchandani & Psychogiou, 2009).

Methodological Considerations

There are also methodological difficulties with this emergent literature. In particular, there is a lack of consensus regarding operationalized definitions of paternal perinatal depression and male postpartum depression (Pilyoung & Swain, 2007). Several studies have used the maternal postpartum depression definition to form the basis of a parental postpartum depression definition (Pilyoung & Swain, 2007). However, this rests on the proposition that perinatal maternal and paternal present in the same way.

In addition, there is evidence of variance in the measurement of paternal depression. It is possible that issues regarding methodology, and the broader questions regarding timing and transmission of risk could be best informed by focussing on prospective study designs.

Aim of the Current Review

Although there have been narrative reviews of the field (Ramchandani & Psychogiou, 2009), there has as yet, been no systematic review of prospective studies investigating the outcomes for offspring of depressed fathers. Therefore, this review sought to synthesise and critically evaluate prospective studies that have examined the association between paternal depression and offspring outcomes. Our primary aim was to establish the strength of association between paternal depressive symptoms or diagnosis of depression and negative child and adolescent outcomes. Secondary aims were to identify whether parenting behaviour was likely to mediate the associations between paternal depression and child and adolescent outcomes. We also sought to identify and evaluate potential methodological sources of bias in the literature.

Methods

Inclusion & Exclusion Criteria

Inclusion criteria were articles that included (i) an assessment of paternal depressive symptoms or diagnosis based on self-report or interview measures, (ii) a measure of offspring internalizing and/or externalizing behaviours, (iii) offspring sample of 21 years or younger, (iv) data which is presented and extractable on the association between paternal depression and offspring outcome (v) follow-up, cohort designs, (vi) were published in English.

Exclusion criteria were (i) studies that measured mental health but a depression score could not be derived from the data, (ii) book chapters, (iii) case studies, (iv) previous meta-analyses/systematic reviews.

Outcomes

Outcomes were defined as internalizing and externalizing behaviours assessed using reliable and validated parent-report/teacher-report/self-report measures.

Literature Search

The review followed PRISMA guidelines (BMJ, 2009). The Electronic databases searched were: OVID (EMBASE: 1980-April 2015, Medline: 1946-April 2015, PsycINFO: 1806-April 2015) and Google Scholar (1980- April 2015). Search terms ‘paternal OR father*’ were joined with the terms ‘depress*’ and ‘adj5’, and then combined with the Boolean operator ‘AND’, alongside the following terms; ‘infant*’, ‘child*’, ‘adolescen*’, ‘offspring’, ‘emotion*’, ‘behav*, ‘psychopatholog*’ and ‘development*’. The truncation [*] was employed to increase the sensitivity of the search. Once all duplicates were removed, inclusion and exclusion criteria were applied to titles, abstracts and full-texts to establish study eligibility. The reference lists and citing papers of the included full-text papers were also searched for additional relevant articles. The remaining articles were reviewed in full and included in the review provided they met the inclusion criteria. The first author conducted the search and identification of relevant studies. Where eligibility of inclusion in the review was unclear (n=6), texts were reviewed for inclusion by the second author and a consensus agreement was reached. Based on this consensus, n=6 full-text articles were excluded. Full details of the extraction process are given in Figure 1.

Quality Assessment

Quality assessment was conducted with an adapted version of the Agency for Healthcare Research and Quality (AHRQ) methodology checklist (Williams et al. 2010). The AHRQ checklist assesses methodological quality of papers (details in Supplemental Table 1). This checklist was adapted to create a bespoke 11-item quality criteria assessment. Potential outcome ratings for each question in the quality criteria were; ‘Yes’ (2 points), ‘Partially’ (1 point), ‘No’ (0 points), and ‘Not applicable’ (0 points) with a range of 0-22. An individual blind to the aims of this review rated 30% of the studies, with 91.5% agreement on criteria. Any studies in which discrepancies existed were reassessed. A supplementary checklist on reporting bias was also created using the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) checklist (Von Elm et al. 2007) (Supplemental Table 2). As it was predicted that the literature is heterogeneous, the application of a further checklist enabled evaluation of the quality of reporting within the literature.

3. Results

Characteristics of the Studies

All 21 of the studies included in the review employed a prospective cohort design; with the exception of Pilowsky et al. (2014) and Gross et al. (2008) which both reported secondary analyses of longitudinal Randomised Control Trials and Van Batenburg-Eddes et al. (2013) which used a cross-cohort design. Van Batenburg-Eddes et al. (2013) combined findings from the prospective Avon Longitudinal Study of Parents and their Children (ALSPAC; Fraser et al. 2013) cohort and the Generation R (Jaddoe et al. 2010) cohort. A further seven studies used data from the ALSPAC cohort (Gutierrez-Galves et al. 2015, Hanington et al. 2010; Hanington et al. 2012; Ramchandani et al. 2005; Ramchandani et al. 2008a; Ramchandani et al. 2008b; Ramchandani et al. 2010). Two studies analysed the Generation R cohort (Van Den Berg et al. 2009; Velders et al. 2011), with Reeb et al. (2010) and Reeb et al. (2015) both using the same, unnamed cohort. Finally, Fletcher et al. (2011) analysed data from the Longitudinal Study of Australian Children (LSAC; Sanson et al. 2002) cohort. Each of the remaining studies used a variety of different cohorts with follow-up information gathered at various stages throughout infancy, childhood and adolescence. With the exception of the Van Battenburg-Eddes et al. (2013) paper, studies are present developmentally. For a full breakdown of study characteristics, see Table 1.

Sample Population

The review identified a combined total of n=21970 fathers with samples ranging from n=9 (Pilowsky et al. 2014) to n=10975 (Ramchandani et al. 2008b). Based on studies where it was reported, the age range of fathers was from 28 to 43. A total of n=23,385 offspring were included, ranging from n=19 (Pilowsky et al. 2014) to n=10,494 (Hanington et al. 2010). Offspring age ranged from 1 month old (Carro et al. 1993) to 21 years old (Reeb et al. 2015). Only four studies examined offspring outcomes beyond 13 years old.

Measurement of Paternal Depression

Paternal depression (incorporating paternal depressive symptoms and clinical diagnoses of depression) was assessed using a variety of self-report or interview

measures with measurement points ranging from 18 weeks gestation to offspring age 21. The most commonly used assessment tool was the Edinburgh Postnatal Depression Scale (EPDS), which was utilized in 10 of the studies. Full details of paternal depression measures are outlined in Table 1, Appendix A

Measurement of Child and Adolescent Outcomes

Child and adolescent outcomes were measured at assessment points ranging from 2 months old to 21 years old. Externalizing and internalizing behaviours were assessed using a number of self-report, parent-report and/or teacher-report measurement tools.

Paternal Depression and Offspring Internalizing and Externalizing Behaviours

As can be seen in Table 2, results across studies indicate a pattern of weak to moderate significant negative associations between paternal depression and offspring outcomes. This association is observable when paternal depression is present in the antenatal stage, infancy, childhood or adolescence. However, the effect of this relationship is significantly reliant on various mediators.

Internalizing Behaviours

Offspring internalizing difficulties were found to be associated with paternal depression in 17 of the included studies (Carro et al. 1993, age = 2-3 years), (Cummings et al. 2013, age = 6-8 years), (Fletcher et al. 2011, age = 4-5 years) (Gross et al. 2008, age = 4 years), (Gutierrez-Galve et al. 2015, age = 42 & 81 months), (Hanington et al. 2010, age = 24 months), (Hanington et al. 2012, age = 42 months), (Keller et al. 2009, age = 6-8years), (Ramchandani et al. 2005, age = 42 months), (Ramchandani et al. 2008a, age = 81 & 91 months); Ramchandani et al. 2008b, age = 3.5 & 7 years), (Reeb et al. 2010, age = 13 & 14 years), Reeb et al. 2015, age 12 & 21 years), (Shelton & Harold, 2008, age = 11-13 years), (Smith et al. 2013, age = 51 months), (Van Den Berg et al. 2009, age = 2 months), (Velders et al. 2011, age = 3.5 years). Moreover, specific associations included excessive crying (OR 1.29, 95% CI: 1.09 –1.52) (Van Den Berg et al. 2009), child temperament (Beta=0.049; $p < 0.001$) (Hanington et al. 2010) and anxiety symptoms (Beta=0.134, p=0.002) (Reeb et al. 2015). Similarly, maternal depression has also been significantly associated with increased levels of offspring internalizing symptoms and negative affect (Goodman et al. 2011).

Externalizing Behaviours

The association was also found for externalizing problems in 9 of the included studies (Carro et al. 1993, age = 2-3 years), (Fletcher et al. 2011, age = 4-5 years), (Gutierrez-Galve et al. 2015, age = 42 & 81 months); Ramchandani et al. 2005, age = 42 months), (Ramchandani et al. 2008a, age = 81 & 91 months), (Ramchandani et al. 2008b, age = 3.5 and 7 years), (Smith et al. 2013, age = 51 months), (Van Batenburg-Eddes et al. 2013, age = 3-4 years), (Velders et al. 2011, age = 3.5 years). More specifically, conduct problems (2·66, 1·67–4·25) (Ramchandani et al. 2005); attention problems (OR 1.11, 95% CI 1.00–1.24) (Van Batenburg-Eddes et al. 2013) and oppositional defiant disorder (adjusted OR 1.94, 95% CI 1.04-3.61) (Ramchandani et al. 2008a) were all associated with paternal depression. These findings are similar to those in the corresponding literature, with small to moderate significant associations found between maternal depression and higher levels of offspring externalizing symptoms, negative behaviour and general psychopathology (Beck et al. 1999; Goodman et al. 2011).

Timing of Depression

These findings also suggest that timing of paternal depression can negatively impact upon many stages of offspring development. Although only measured in 5 of the included studies, antenatal paternal depression associated with emotional problems in all 5 studies and behavioural problems in 4 of 5 included studies, in children aged between 2 months and 7.5 years (Hanington et al. 2012; Ramchandani et al. 2008b; Van Batenburg-Eddes et al. 2013; Van Den Berg et al. 2009; Velders et al. 2011). In the postnatal period, paternal depression was also associated with both internalizing (11 included studies) and externalizing problems (7 included studies) in early to late childhood, with associations present in offspring from 2 months old to 8 years old (Carro et al. 1993; Cummings et al. 2013; Fletcher et al. 2011; Gross et al. 2008; Gutierrez-Galve et al. 2015; Hanington et al. 2010; Hanington et al. 2012; Keller et al. 2009; Ramchandani et al. 2005; Ramchandani et al. 2008a; Ramchandani et al. 2008b; Smith et al. 2013; Velders et al. 2011). Finally, in 3 studies, paternal depression was also associated with an increased risk of negative adolescent functioning, specifically internalizing symptoms, in offspring ages 11 to 21 years old (Reeb et al. 2010; Reeb et al. 2015; Shelton & Harold, 2008). These results are echoed

in the literature examining maternal depression and offspring development, where exposure at earlier developmental stages increases the strength of the associations between the two variables (Goodman & Gotlib, 1999; Goodman et al. 2011).

Van Batenburg-Eddes et al. (2013) (Generation R cohort) and Ramchandani et al. (2013) (age 3 & 12 months) found no significant association between paternal depression and offspring attention and externalizing problems, respectively. Ramchandani et al. (2010) also found no significant association for children aged 6 and 81 months, however paternal symptoms predicted less offspring prosocial behaviours and finally, the association was no longer significant following adjustment for covariates in both the Smith et al. (2013) (age = 51 months) and the Velders et al. (2011) (age = 3.5 years) studies. Additionally, 4 papers (Hanington et al. 2012; Gross et al. 2008; Keller et al. 2009; Shelton & Harold, 2008) found no association between paternal depression and offspring (age range = 3.5 years to 8 years) externalizing behaviours. Numerous mediators impacted on the strength and formation of the associations observed in the studies.

Indirect Effect of Paternal Depression

Although the above evidence suggests a consistent pattern of weak to moderate associations between paternal depression and negative offspring outcomes, the reviewed studies indicate the presence of multiple potential mediators that also contribute to this association. This suggests a multi-factorial model of offspring risk, with direct and indirect effects on offspring outcomes. (See figure 2).

Mediators

The most common mediators of risk were parenting behaviours (paternal hostility, father involvement, father's negative expressiveness), with this association arising in 7 of the included studies and marital conflict, which was apparent in 5 of the included studies. Paternal depressive symptoms increased the risk of child and adolescent internalizing and externalizing problems as a function of paternal hostility (Reeb et al. 2010; Velders et al. 2011). Ramchandani et al. (2010) indicated that prosocial and problem behaviours were less common with high levels of father involvement but were more likely with higher levels of paternal depression. Gutierrez-Galve et al. (2015) showed fathers' non-involvement accounted for 5.4% of the total effect of paternal depression on child development at age 3.5 and 8.4% at age 7. Fathers'

negative expressiveness significantly mediated the impact of paternal symptoms on children's internalizing symptoms (Cummings et al. 2013; Keller et al. 2009; Shelton & Harold, 2008), externalizing symptoms (Shelton & Harold, 2008) and emotional insecurity (Cummings et al. 2013). Analogous to this, parenting difficulties as a result of depression play an important role in mediating the impact of maternal depression on offspring development (Murray & Cooper. 1997; Stein et al. 1991). Marital conflict accounted for 27.4% of the total effect of paternal depression on child outcomes at age 3.5 years and 27.2% of the total effect at follow-up (Gutierrez-Galve et al. 2015). Similarly, Hanington et al. (2012) found that marital conflict increased the impact of fathers' symptoms on offspring outcomes by 17.6%. Marital conflict also strongly mediated the formation of complex associations between paternal depression and child emotional and behavioural difficulties (Keller et al. 2009; Shelton and Harold, 2008 Smith et al. 2013). These findings are again consistent with those in the literature examining maternal depression and offspring outcomes (Cummings & Davies, 1994).

It should be noted, in each study that controlled for maternal depression, paternal depression remained independently associated with increased risk of negative offspring outcomes. Although maternal depression is substantial in heightening the risk, significance of fathers' depression remains in the studies whether maternal depression was present or not.

Study Quality

Study methodological quality scores are presented in Table 3. Scores ranged from 11 to 18. The most common methodological issues arose in relation to justification of sample size, with only a single study conducting a power analysis whilst also having a sufficiently large sample size to detect a clinically significant difference of 5% (Smith et al. 2013). The blinding of the assessors was unaccounted for consistently across the included studies, with again only one study employing partial blinding of the researchers (Pilowsky et al. 2014). This may have been prevented with the introduction of external assessors blind to participants' clinical status. Finally, bias in cohort selection existed, with only five studies outlining one or both of the inclusion/exclusion criteria (Cummings et al. 2013; Keller et al. 2009; Ramchandani

et al. 2013; Shelton & Harold, 2008; Smith et al. 2013). However, we note that the majority of the literature (10 papers) in this area are derived from analyses of three large cohort designs such as ALSPAC, Generation R and LSAC, and as such rely on these cohort protocols for these details. In addition to this, two studies employed bias in their sample selection techniques, with the use of flyers to obtain participants (Cummings et al. 2013; Keller et al. 2009). The highest rating achieved was 18 points (Ramchandani et al. 2008b; Smith et al. 2013), suggesting that these two studies are strongest methodologically. Question 2 of the quality criteria was not applicable to the majority of the studies due to their designs.

Regarding the statistical analyses utilized, the majority of the studies provide a comprehensive outline of effect sizes and confidence intervals. Just five of the studies refer to power calculations in their papers (Hanington et al. 2012; Keller et al. 2009; Ramchandani et al. 2008b; Ramchandani et al. 2010; Van Den Berg et al. 2009), however if the sample size is large, or the sample size is referred to as a limitation, it could be reasonably determined that power issues have been considered and addressed appropriately.

Concerning the validation and reliability of the measurement tools used to assess paternal depression, all studies scored highly on this criterion, however the reporting of testing for this validity is not always clear (Cummings et al. 2013; Fletcher et al. 2011, Gross et al. 2008; Keller et al. 2009; Pilowsky et al. 2014; Shelton & Harold, 2008). This issue also arose when reporting offspring measurements (Gross et al. 2008; Gutierrez-Galve et al. 2015; Pilowsky et al. 2014; Ramchandani et al. 2005; Shelton & Harold, 2008; Van Den Berg et al. 2009).

All but one of the studies had at least one follow-up point a minimum of 6 months after collection of baseline data (Van Den Berg et al. 2009). Yet, the length of these follow-up points varied considerably, meaning the influence of time is unclear. With regards to attrition rates, drop-out figures are reported in all but two of the studies (Fletcher et al. 2011; Gross et al. 2008) and the calculation of missing data to account for any potential bias of the dropouts is reported in a minority of studies (Cummings et al. 2013; Gross et al. 2008; Ramchandani et al. 2008a; Reeb et al. 2015; Shelton & Harold, 2008; Smith et al. 2013; Van Batenburgh-Eddes et al. 2013; Van Den Berg et al. 2009; Velders et al. 2011).

The assessment of confounding variables and their inclusion in analyses was relatively strong throughout the included studies. Consideration of at least of one of maternal depression, parenting behaviours or marital conflict were considered by all of the studies.

In terms of quantifying study quality, the mean total score was 14, with only one study rated more than two points below this. The remaining studies scored within two points of this mean, with twelve rated equal to, or above this. Based on this pattern of results we suggest that the quality of the literature is relatively consistent with evidence that the findings are methodologically robust.

Discussion

The results of our systematic review indicate a pattern of associations between paternal depression and increased risk of internalizing and externalizing behaviours in offspring. This association is evident across the developmental frame from offspring age of 2 months to 21 years old. Associations were stronger and more common in early childhood, suggesting this may be a particularly sensitive period of development. This is consistent with previous literature examining the impact of maternal depression on child outcomes, which states that earlier exposure can result in an increased vulnerability to atypical development (Goodman & Gotlib, 1999; Goodman et al. 2011). The reported association was evident both for depression measured at antenatal and postnatal time-points. Negative offspring outcomes incorporated a broad range of marker of suboptimal outcomes from increased risk for psychiatric disorders, oppositional defiant and conduct disorders (Ramchandani et al. 2008a), to excessive infant crying (Van Den Berg et al. 2009). Associations remain significant across offspring development at follow-up and into young adulthood, however fewer studies examined outcomes beyond the age of 13. Furthermore, the associations between paternal depression and offspring outcomes remained significant after controlling for key covariates, such as maternal depression. This does not necessarily suggest a direct causal link for transmission of risk, as the direct of paternal depression on offspring problem behaviours was of a relatively modest size. Therefore any transmission of psychopathological risk is likely to be subject to a number of indirect mechanisms to mediate the strength of this association, including

parenting behaviours and marital conflict. In addition, there is also a lack of specificity over the magnitude and timing of exposure, with duration of observation and assessment varying widely in the studies. Although the association is smaller and less direct, these findings are similar to those established in the corresponding literature on maternal depression and offspring outcomes.

In addition, we note that our results also need to be integrated with the substantial literature on sensitive periods within the child developmental neuroscience literature (Cichetti, 2015). Our findings support the proposition from multiple domains of analysis that exposure to stress early in life had a dose-dependent effect on increasing the risk of later psychopathology, particularly in the domain of internalizing behaviours (Andersen 2015). That said, much of this literature is based on either animal models or in relation to the mother-infant dyad. Therefore, the review's findings sit within a translational developmental psychopathology paradigm, whilst highlighting the relevance of paternal psychopathology in this line of enquiry.

Mechanisms of Transmissions of Risk

Fathers who exhibit depressive symptoms during the postnatal period have been found to obtain a lesser sense of parenting efficacy and increased levels of parental distress (Demontigny et al. 2013). These findings may be linked to the impact that depression also has on subsequent parenting behaviours and father involvement. In such a case, this would likely impact on the father-child relationship. One possibility is that deficits in the father-child bond increase the risk of childhood insecure attachment patterns, in response to stress. Research suggests that maternal depression decreases the likelihood of secure attachment and increases the risk for avoidant and disorganized attachment (Martins & Gaffan, 2000; Teti et al. 1995). These types of attachment have commonly been linked to a catalogue of internalizing and externalizing behaviours (Colennessi et al. 2014; Groh *et al.* 2012; Jinyao *et al.* 2012). If this same potentiation of risk applies to paternal attachment then this could constitute one developmental pathway from paternal depression to offspring vulnerability. Consistent with this model, father interaction and father non-involvement are both associated with child internalizing and externalizing behaviours (Wilson & Durbin, 2010) and these are two primary mechanisms through which paternal depression can negatively impact upon child outcomes.

We also observed that in several studies (N=7), marital conflict significantly mediates the association between paternal depression and offspring development. For example, the presence of paternal depression increases the risk for marital conflict and this conflict has been suggested to increase by up to 50% between the antenatal and postnatal period in families with depressed parents, resulting in a large risk increase for children (Hanington et al. 2012). This supports the suggestion that paternal depression exerts its influence by acting as the catalyst of a series of actions that combine to negatively impact upon their children. This method is considerably different to how maternal depression impacts offspring, with the influence much more direct and less reliant on contextual mechanisms (Gutierrez-Galve et al. 2015). This finding corresponds with the ACE literature which notes that marital conflict can be considered an adverse childhood experience which may result in suboptimal development for offspring (Chartier et al. 2010).

Limitations

We note several limitation of the review. Firstly, as discussed above, although there are consistent associations between paternal depression and offspring outcomes, these are of a small to moderate magnitude and subject to multiple mediators. A potential explanation for this may be that depressive symptoms of those who participated in the study at follow-up were lower than those who dropped out (Ramchandani et al. 2010; Van Den Berg et al. 2009). Children of fathers with higher symptoms were less likely to partake in the follow-up and this sub-group had higher rates of anxiety disorder (Ramchandani et al. 2008a). Therefore, we cannot comment on the impact of higher or more complex levels of paternal psychopathology on offspring outcomes. This suggests that clinical or more severe cases of depression may increase the negative effect on offspring development. We also note a potential Hawthorne Effect, given that most of these data arise from longitudinal cohorts with multiple follow-up points. Consequently, the act of being monitored within a cohort study may act as a form of additional support, buffering the effect for paternal symptoms, which again may explain the modest associations.

The timing of assessments may also have affected the results. This is reflected in differing rates for offspring emotional and behavioural based on whether paternal depression is assessed antenatally or postnatally. We also note that only 5 of the 21

included studies measured paternal antenatal depression. However, fathers who had both antenatal and postnatal depression posed the greatest risk to their offspring (Ramchandani et al. 2008b). This suggests that the literature needs a greater focus on antenatal depression measurement, in order to establish the reliability of findings, particularly across multiple developmental measurement points.

Paternal depression was almost exclusively measured using self-report questionnaires introducing a potential reporting bias. Indeed, men have been found to under-report their levels of depression to a greater extent than females (Allen-Burge et al. 1994; Eaton et al. 2000), which may contribute to the small effect, found relating to paternal depression and offspring outcomes. Additionally, there was a high degree of heterogeneity amongst depression measures used. The most frequent measure utilized was the EPDS, which although validated for use in men, also conflates depressive and anxiety symptoms (Green, 1998; Stuart et al. 1998). This also raises the possibility that paternal anxiety confounds the association between father and child characteristics.

Furthermore, owing to the restriction of including English-only papers, the review may not be open to cross-cultural comparisons. However, studies that were included examined cohorts in the UK, the USA, the Netherlands and Australia, meaning there is more than an element of generalizability for this review. There is also potential bias from over-representation of certain samples, given the included literature includes multiple reports from the ALSPAC, Generation R and LSAC cohorts.

In addition, several studies used parent reporting of offspring internalizing and externalizing behaviours. These self-report measures may introduce subjective bias as cognitive-affective aspects of paternal depressive symptoms may impact on fathers' perceptions of both the father-child relationship and their child's problem behaviours (Treutler & Epkins, 2003). This underscores the need to conceptualize associations between paternal mental health and child outcomes with appropriate reference to mediating mechanisms, particularly interpersonal factors such as paternal

involvement. Moreover, although evidence regarding the existence of bidirectional relationships of paternal depression and offspring characteristics may be limited (Gross et al. 2008), this may also impact on fathers' interpretation and subsequent rating of child symptoms. In addition, studies were only conducted in UK, Europe, Australia and USA limiting cross-cultural generalizability of the findings. The role of the father differs across cultures, with father-involvement varying in degree throughout the world and as such the effect of paternal depression may not be as strong on children in other cultures (Munroe & Munroe, 1992).

Finally, we note that the primary aim of this systematic review was to examine the association between paternal depressive symptoms or diagnosis of depression and negative child and adolescent outcomes looking across the published literature. Our findings suggest that a critical next step is to further clarify effects using meta-analysis or similar statistical modelling, in order to address cross-study effects. Our review highlights that meta-analytic approaches would need to incorporate methodological aspects within the existing literature, including heterogeneity of measures, time periods and outcomes across different studies.

Implications for Future Research

A clear finding from the review is the need for more detailed examination of mediating factors between paternal depression and offspring outcomes – particularly with regard to environmental associations between paternal depression and offspring outcomes is necessary. Whilst the review has identified several potential mechanisms, it is still unclear *how* paternal depression enables or obstructs the intergenerational transmission of psychopathology. More detailed research would create a theoretical basis for treatments during the perinatal period. We also note that the data on severity of paternal depression is relatively sparse; therefore future researching that explores stratification of samples by depression severity would be a welcome addition to the area.

The research suggests that offspring differ in their susceptibility to paternal depression, with no clear pattern with regard to gender specific risk factors between girls or boys (Hanington et al. 2010; Reeb et al. 2010; Ramchandani et al. 2005). This is puzzling, given the well-known observation of differing patterns of risk to specific

forms of developmental psychopathology between genders, with girls more likely to exhibit internalizing disorders, and boys to present with externalizing disorders (Zahn-Waxler, Shirtcliff and Marceau, 2008). The existing literature proposes that these gender differences reflect a confluence of biological and environmental factors, operating at multiple domains of enquiry. To further investigate these gender difference in the case of paternal depression, one possibility would be to explore gene-environment interactions, with the potential for multi-level analyses as has been applied to the literature on conduct disorder (Meier et al., 2011, Moffit & Caspi, 2001). Another approach would be to use different study designs to inform potential associations, such as twin-studies (Kim-Cohen et al. 2005) or adoption studies (Tully et al. 2008), which may shed more light on this topic.

With regard to methodological improvements, blind research measurement of fathers' depressive symptoms would provide a more objective assessment. Furthermore, greater uniformity of assessment tools and timings would also facilitate cross-study comparisons and may improve quality of evaluating child outcomes across developmental periods. We also highlight a paucity of research on antenatal paternal depression. Further research on fathers' with depression during the gestation period and subsequent offspring behaviours will provide supplementary information on risk transmission.

Implications for Clinical Practice

The review highlights the potential value of mental health screening of both parents during pregnancy. Women are consistently screened for postnatal depression but this was until recently, rarely the case for men (Goodman, 2004). Both professional and public acknowledgement that men can experience depression during pregnancy and beyond and the negative implications this may have for offspring development presents an opportunity for the development of preventative interventions. This may also prove to be cost efficient, as paternal postnatal depression is resulting in augmented community care costs for primary care, mental health groups and hospital services (Edoka et al. 2011). In parallel to this, there would be value in considering what protective factors may be specific to the father-child relationship, in addition to more general protective factors such as attachment security (Brown et al, 2013).

In addition, these findings suggest that, from a more holistic perspective, interventions for paternal depression may be improved by engaging with the whole family, focussing on parenting behaviours, parent-child interaction and marital conflict as this could greatly reduce the negative impact of paternal depression. The assessment of the family environment and functioning may enhance couple and family relationships. This has the potential to improve family life, communication and reduce depressive symptoms in fathers (Pilyoung & Swain, 2007). This is especially important as the risk of depression co-existing in partners is higher, and normal paternal mental health can buffer the effects of maternal depression on offspring outcomes (Gere et al. 2013). Finally, the review highlights the complex interactional nature of parenting relationships, particularly when viewed longitudinally. Intervention frameworks may therefore need to accommodate an awareness of dynamic, bidirectional interactions within the family unit. For instance, an infants' constitutional difficulties at any given time (e.g. colic, dysregulated sleep patterns) may contribute to family stress, which in turn may contribute to increased levels of parental sleeplessness, distress and low mood, which can affect parenting, and lead to babies becoming more uncomfortable/difficult (Gross et al., 2008; Hestbaek et al., 2014).

Conclusion

This review synthesises existing data demonstrating that paternal depression can play a significant role in negatively impacting upon infant, child and adolescent development. The strength of the association varied in the literature, however it supports the findings of recent literature that fathers can have a significant impact on offspring outcomes (Ramchandani & Psychogiou, 2009). This review has also identified and quantified the many mechanisms in which paternal symptoms exert their influence. This primarily occurs through family environment mechanisms. Further research is needed on mechanism of transmission in order to fully understand the extent of this association. Early intervention, which can identify and address both parents' mental health needs, is potentially required to provide the optimum opportunity for child development.

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Figure 1. Study extraction process

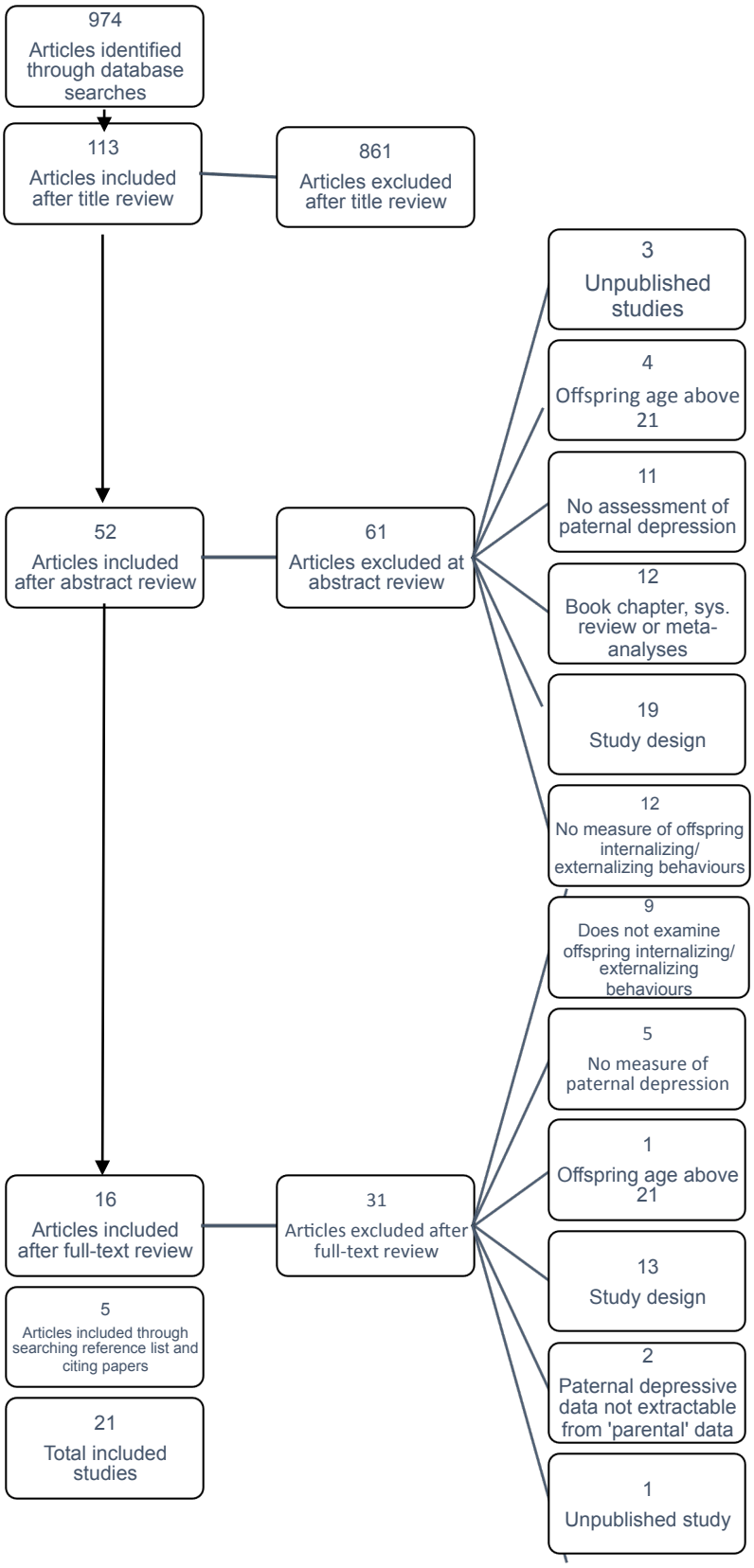

Figure 2. Hypothetical Mediational Model Outlining Transmission of Risk

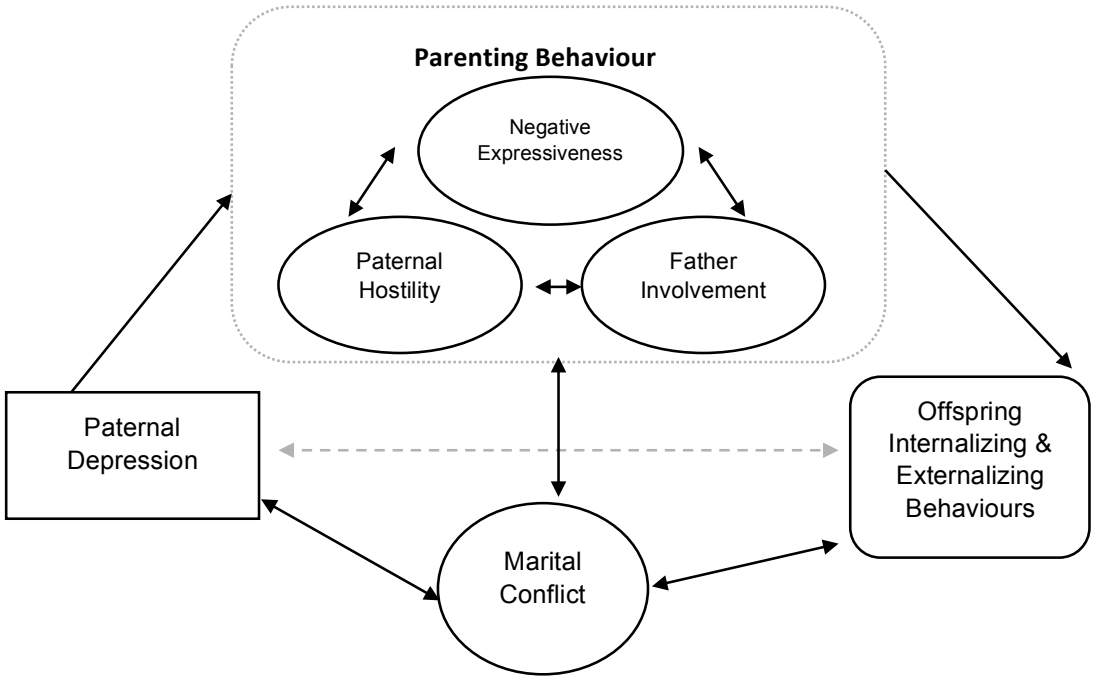

Table 1. Characteristics of included studies

First Author Year Location	Paternal Characteristics N, age	Child Characteristics N, age, gender	Paternal Depression Assessment Timing of Assessment	Child & Adolescent Outcomes Measure Assessment Timing	Covariates
Van Batenburg-Eddes (2013) Netherlands & UK†	ALSPAC N=3442 Generation R N=2280	ALSPAC N=3442 Generation R N=2280	ALSPAC EDPS 18 weeks gestation Generation R BSI 20 weeks gestation	ALSPAC SDQ Generation R CBCL/1½– 5	ALSPAC Infant gender, birthweight, maternal education, family income, parent ethnicity, maternal smoking and alcohol use. Generation R Maternal education, alcohol and smoking intake during pregnancy, family income, child ethnicity
Hanington (2010) UK	ALSPAC N=7168 (T1) 6170 (T2)	N=10494 (T1) 10317 (T2)	EPDS 8 months postpartum (T1) 24 months postpartum	*Temperament* CTSs 6 months old(parent) 24 months old(parent)	Child sex, ethnicity, parent sex, ethnicity, parent education, social class & MDep
Ramchandani (2010) UK	ALSPAC N=8431	N=5064 Range=6 and 81 months b=2586, g=2478	EPDS 18 months postpartum	*Temperament* ITQ 6months old(parent) *Prosocial and problem behaviour in children* SDQ 81months old(parent)	Parental involvement, father's highest educational level, child sex.
Ramchandani (2005) UK	ALSPAC N=8431	N=10024	EPDS 8 weeks postpartum 21 months postpartum	*Emotional/behavioural development* Rutter's revised preschool scale 42 months(parent)	Child sex, MDep, paternal social class, parental education.
Gutierrez-Galve (2015) UK	ALSPAC N=8431	N=10024 at 42 months 8401 at 81 months	EPDS 8 weeks postpartum 8 months postpartum	*Emotional or behavioural problems* Rutter's Revised Preschool Scale 42 months old (parent) SDQ 81 months old(parent)	MDep, couple conflict, paternal non-involvement, father's antisocial traits, alcohol & cannabis misuse, parental age, parental education.
Hanington (2012) UK	ALSPAC N=9846	N=9910	EPDS 18 Weeks gestation (T2) 8 months postpartum (T2)	*Emotional/behavioural problems* Rutter's Revised Preschool Scale 42 months old(parent)	Marital conflict, child age, gender, ethnicity. Parent age, ethnicity, social class, MDep symptoms.
Ramchandani (2008)b UK	ALSPAC N=7601 Mean age=28.8(SD=9.75)	N=6449 B=3297, g=3152	EPDS 18weeks gestation 8weeks postbirth	*Emotional/behavioural development* Rutter's Revised Preschool Scale 3.5 years old(parent)	Father's age at childbirth, number of children in family home at childbirth, paternal education, paternal ethnicity, social class, marital status, past history of

				Development and Wellbeing DAWBA 7 years old(parent & teacher)	severe depression.
Ramchandani (2008)a UK	ALSPAC N=10975	N=8401 Range=6-7 years old	EPDS 18 weeks gestation 8 weeks postpartum 8 months postpartum 21 months postpartum	*Emotional/behavioural development* SDQ(parent) 81 months old *Development and wellbeing* DAWBA(parent) 91 months old	Parental anxiety, history of severe depression, family history of depression, father's age at childbirth, paternal education, degree of crowding in family home, number of children in family at childbirth.
Van Den Berg (2009) Netherlands	Generation R N=5463	N=4426	BSI 20 weeks gestation	*Excessive Infant Crying* Wessel's Criteria 2 months old(parent)	MDep, age, education, ethnicity, number of children, child birthweight, gender, ethnicity, father education, ethnicity, age, parent smoking and alcohol use.
Velders (2011) Netherlands	Generation R N=2698	N=2698	BSI 20 weeks gestation	*Behaviour problems* CBCL for ages 1½–5 years 3 years old(parent)	Prenatal and postnatal parent psychological symptoms, prenatal family functioning, parent ethnicity, infant birthweight, gender, ethnicity, birth order, parental age at childbirth, parental education. Maternal smoking and alcohol use during pregnancy, prenatal scores on BSI subscales.
Ramchandani (2013) UK	N=192 Mean age=35 (SD=5.7)	N=168 b=78, g=90	EPDS 7 weeks postpartum	*Behaviour problems* CBCL for ages 1½–5 years 1 year old(parent) *Temperament* GRS 3 months old(parent)	Infant sex, paternal education, age, social class, SES, father-infant interactions, maternal sensitivity, MDep, paternal antisocial behaviour, paternal age, paternal employment.
Fletcher (2011) Australia	LSAC N=2620	N=5104 Age=assessed at 3-19 months, 2-3 years and 4-5 years	K6 Offspring age 4-5	*Emotional and behavioural functioning* SDQ(parent) *Overall development and wellbeing* DOIs(parent)	Socioeconomic position, maternal education, MDep, later paternal depression, child sex.
Carro (1993) USA	N=70 Mean age=30.73 years (SD=4.28)	N=70? (M = 2.67 years, SD=0.47). Age range=2-3 years	BDI 1 month postpartum	CBCL 2-3 years old(parent)	MDep symptoms, marital satisfaction, parental perceived stress, parental perceived social support
Gross* (2008) USA	N=297	N=731 Mean age=29.9 months (SD=3.2) at T1 Age range=2-4 years	CES-D T1 (offspring age 2) T2 (offspring age 3) T3 (offspring age 4)	CBCL 4 years old(parent)	Family demographics, child noncompliance, MDep
Smith (2013) UK	N=705 Mean age=34	N=705	EPDS 3 months postpartum GHQ 36 months postbirth	*Emotional/behavioural development* SDQ 51 months old(parent)	Infant sex, parent SES, marital status, parent's occupational status, maternal mental health, marital discord, mother lives with partner, paternal ethnicity, parental age.
Cummings	N=206	N=235 (b=106, g=129)	CES-D Scale	*Emotional Insecurity*	Child ethnicity, gender, age. Parental age,

(2013) USA	Age=mean 36.84 (SD=6.14)	Age=mean 6.0 (SD=0.45)	T1 (offspring age 6) T2 (offspring age 7) T3 (offspring age 8)	SIMS-PR 8 years old(parent) *Internalizing Symptoms* CBCL 6 & 8 years old(parent) TRF 6 & 8 years old(teacher)	ethnicity, marital status, parental education, income, marital conflict, MDep symptoms, parental negative expressiveness
Keller (2009) USA	N=235	N=235 Age=mean 6.0 (SD=5 B=106, g=129	CES-D T1 (offspring mean=age 6)	*Internalizing and externalizing symptoms* CBCL 6 years old & T3(parent)	MDep, marital conflict, marital negativity & child sex
Pilowsky* (2014) USA	N=9 Mean age=43.6	N=19 Mean age:11.89 (SD=3.21) Range=7-17	HAMDEP17 Baseline and 1, 2, 3, 6 and 9 months after	*Emotional/behavioural symptoms* CDI(self-report) MASC(self-report) CIS(self-report) C-GAS(assessor) K-SADS-PL(parent & self-report)	Parent gender, time of assessment, age & sex of child, number of child siblings.
Shelton (2008) UK	N=352	N=352 B=179, g=173 Age range=11-13 Mean age=11.67 (SD=0.47)	BDI T1 (offspring age=11-13)	*Internalizing symptoms* Withdrawn and Anxious–Depressed subscales of CBCL(self-report) CDI(self-report) at 11-13 years & 13-15 years *Externalizing symptoms* Aggression subscale of CBCL (self-report) Trait measure of antisocial behavior by Buss and Durkee(self-report) Aggression subscale of CBCL(teacher report) at 11-13 years & 13–15 years	MDep symptoms, parental relationship insecurity, inter-parental conflict, child gender.
Reeb (2015) USA	N=395 Mean age=38	N=395 Mean age=12.61(SD=0.55) at T1 Mean age=20.66(SD=0.55) at T2 Age range=20-22	SCL-90-R Depression Subscale Time 1(1989)	*Internalizing symptoms* SCL-90-R (depression subscale) Time 1(1989)(self-report) SCL-90-R (anxiety subscale) Time 2(1997)(self-report)	Family income, offspring sex, parents' age, parental education, physical health, parent anxiety, marital instability, hostile parenting, marital discord, economic hardship, MDep symptoms.
Reeb (2010) USA	N=451	N=451 Mean age=13.2 b=215, g=236	SCL-90-R Depression Subscale Year 1 (1989) Year 2 (1990)	*Depressive symptoms* SCL-90-R Depression Subscale Mean age 13.2(Self-report) Year 2(self-report)	Social class, parent age, family income, parents' education, MDep mood, offspring gender, paternal hostility, interaction effects.

Notes: b: boy, BDI: Beck Depression Inventory, BSI: Brief Symptoms Inventory, C-GAS: Child Global Assessment Scale, CBCL: Child Behaviour Checklist, CBQ: Conflict Behaviour Questionnaire, CDI: Children's Depression Inventory, CES-D: Center for Epidemiologic Studies- Depression, CIS: Columbia Impairment Scale, CTS: Carey Temperament Scale, DAWBA: Development and Well-Being Assessment, DOIs: Derived Outcomes Indices, EPDS: Edinburgh Postnatal Depression Scale, g: girl, GHQ: General Health Questionnaire, GRS: Global Rating Scale, HAMDEP17: Hamilton

Rating Scale for Depression, HSCL-10: Hopkins Symptoms Checklist, IBQ: Infant Behavioural Questionnaire, ITQ: Infant Temperament Questionnaire, K-SADS-PL: Kiddie Schedule for Affective Disorders and Schizophrenia for School-Age Children- Present and Lifetime Version, K-SADS: Kiddie Schedule for Affective Disorders and Schizophrenia for School-Age Children, K6: Kessler Psychological Distress Scale, LIFE: Longitudinal Interval Follow-up Evaluation, MASC: Multi-dimensional Anxiety Scale for Children, MCS: Mental Component Scale, MDep: Maternal Depression, MFQ: Mood and Feelings Questionnaire, N: Number, PHQ-2: Patient Health Questionnaire, PHQ: Patient Health Questionnaire, SCID-NP: Structural Clinical Interview for DSM-IV Axis Disorders-Non-Patient Version, SCID: Structural Clinical Interview for DSM-IV Axis Disorders, SCL: 90-R: Symptoms Checklist 90- Revised, SD: Standard Deviation, SDQ: Strengths and Difficulties Questionnaire, SF-12: Short Form 12, SIMS-PR: Security in the Marital Subsystem, T1: Time 1, T2: Time 2, T3: Time 3, TRF: Teacher Report Form; *Denotes secondary analysis of RCT; †Denotes cross-cohort design

Table 2. Key findings from included studies.

First Author Year Location	Main Findings	Internalizing Behaviours	Externalizing Behaviours	Covariates
Van Batenburg-Eddes (2013) UK	ALSPAC: Antenatal PDep associated with increased risk of child attention difficulties Gen. R: No association found	‡	ALSPAC: PDep associated with increased risk of child attention problems (OR 1.11, 95% CI 1.00–1.24).	PDep anxiety symptoms were not significantly associated with child adjustment problems in either cohort.
Hanington (2010) UK	PDep symptoms lead to more difficult temperament at follow-up. Only significant for boys.	PDep at T1 significantly predicted child mood (Beta=0.049; p < 0.001) and intensity (Beta=0.038; p = 0.003) at T2.	‡	Significance remained when controlling for earlier mood/intensity and later PDep.
Ramchandani (2010) UK	No direct effects for PDep and infant reactivity were found.	Infant reactivity (6months) was correlated with father involvement (*r*=0.06; *p* < .01) and PDep symptoms (*r*=0.03; *p*=0.04).	Higher PDep predicted fewer prosocial behaviours in offspring. Effects were found for total behavioural problems; for father involvement (*Beta*= -0.11; *p* < .01), PDep (*Beta*=0.10; *p* < .01), and infant reactivity (*Beta*=0.04; *p* < .01).	Infant reactivity (6 months) correlated with father involvement (r=0.06; p < .01) and PDep symptoms (r=0.03; p=0.04). Prosocial behaviour main effects were found for father involvement (Beta=0.06; p < .01), PDep (Beta=−0.06; p < .01), infant reactivity (b=0.07; p < .01.)
Ramchandani (2005) UK	Postnatal PDep was associated with poorer emotional and behavioural outcomes in offspring (3-5 years old) (AOR 2·09, 95% CI: 1·42–3·08).	PDep strongly associated with increased risk for high scores on emotional problems scale, before controlling for covariates.	Higher risk of conduct problems in boys (2·66, 1·67–4·25).	Effects persisted after controlling for postnatal MDep (2·09, 1·42–3·08), later PDep (conduct problems (1·73, 1·06–2·85), hyperactivity (1·96, 1·12–3·43) but not emotional problems.
Gutierrez-Galve (2015) UK	PDep was associated with negative child outcomes.	Postnatal PDep effect on child (42 months old) total psychological problems= 0.168 (95% CI: 0.133–0.202; P<.001). Direct effect=34.5%. 81 months old: PDep effect on total psychological problems=0.130 (95% CI: 0.098–0.161; P< .001). Direct effect=32.1%	Postnatal PDep effect on child (42 months old) total psychological problems= 0.168 (95% CI: 0.133–0.202; P<.001). Direct effect=34.5%. 81 months old: PDEP effect on total psychological problems=0.130 (95% CI: 0.098–0.161; P< .001). Direct effect=32.1%	At 42 months: 3 indirect effects: MDep= 32.7% (0.055/0.168); couple conflict= 27.4% (0.046/0.168) paternal non-involvement= 5.4% (0.046/0.168) At 81 months: MDep= 32.3% (0.042/0.130); couple conflict= 27.2% (0.035/0.130); paternal non-involvement= 8.4% (0.011/0.130)
Hanington (2012) UK	Marital conflict mediated the association between PDep and child outcomes.	Postnatal PDEP (OR 2.20, 95% CI 1.47–3.28) predicted total child problems at 42 months. Antenatal PDep predicted later child problems (OR 2.34, 95% CI 1.70–3.23)	Paternal depression did not predict conduct problems.	Marital conflict attenuated score by 17.6% but effect of PDEP remained strong (OR 1.98, 95% CI 1.31–2.99). Significance remained for antenatal PDep when marital conflict included (OR 2.17, 95% CI 1.54–3.05).
Ramchandani (2008b) UK	Prenatally and postnatally depressed fathers children had the greatest risk of psychopathology (3½ years) (OR 3.55; 95% CI: 2.07, 6.08) and psychiatric diagnosis (7 years) (OR 2.54; 1.19, 5.41).	Children of prenatal-only group had increased likelihood of scoring higher on emotional symptoms scale than non-depressed group (19.1% vs. 12.9%; unadjOR 1.60 (1.04, 2.47)).	Sons of postnatal-only group had increased scores for conduct problems age 3½ years (OR 2.14; 1.22, 3.72), boys of the prenatal group did not (OR 1.41; .75, 2.65).	Little change in associations when controlling for MDep and other covariates.
Ramchandani (2008a) UK	Postnatal PDep was significantly associated with child psychiatric disorder 7 years later (aOR 1.72, 95% CI 1.07-2.77)	Postnatal PDep was strongly associated with later child psychiatric diagnoses 7 years later (aOR 1.72, 95% CI 1.07-2.77)	Strongest associations were found between PDep and oppositional defiant/conduct disorders (aOR 1.94, 95% CI 1.04-3.61).	Controlling for paternal eductaion and MDep, a 66% increase remained in likelihood of psychiatric diagnosis (OR 1.66; 95% CI: 1.05Y2.63; p=0.030) and 50% increase of oppositional defiant/conduct disorders (OR 1.97; 95% CI 1.08Y3.58; p=0.026) for children of postnatally depressed fathers.

Van Den Berg (2009) Netherlands	PDep was significantly associated with increased risk of excessive infant crying (OR 1.29, 95% CI: 1.09 –1.52)	PDep was significantly associated with increased risk of excessive infant crying (OR 1.29, 95% CI: 1.09 –1.52)	‡	Significance remained after adjusting for confounders, including MDep.
Velders (2011) Netherlands	PDep symptoms increased risk of child problems. Paternal postnatal hostility explained this increase.	Prenatal PDep symptoms predicted offspring internalizing problems (OR 1.15, 95% CI: 1.05-1.26). (Not significant when paternal hostility added).	Prenatal PDep symptoms predicted higher offspring externalizing problems (OR 1.12, 95% CI: 1.02; 1.23). (Not significant when paternal hostility added).	Postnatal paternal hostility (OR= 1.30, p < 0.001) independently associated with child internalizing and externalizing problems.
Ramchandani (2013) UK	No significant association between PDep and child externalising behaviours.	‡	PDep (Beta=0.102 t(1.163, p < .247)) was not associated with child externalizing behaviours.	Paternal age (Beta=.214, t(2.521) p<0.013) and paternal remoteness (Beta=0.175, t(2.001) p<.048) were significantly associated with child externalizing behaviours.
Fletcher (2011) Australia	Early PDep was a significant predictor of numerous poor child outcomes.	Early PDep was associated with higher risk of a low DOI score (OR, 2.70; 95% CI, 2.44–2.98)	Early PDep was associated with increased risk for behavioural difficulties (OR, 3.34; 95% CI, 3.06–3.65).	Controlling for early MDep and later PDep, significance remained. (aOR behavioural difficulties=1.93 (95% CI, 1.75–2.14); OR, DOI score=1.65 (95% CI, 1.48–1.85).
Carro (1993) USA	PDep symptoms 1 month postpartum predicted child internalizing and externalizing problems at 2-3 years old.	Postpartum PDep symptoms approached significance for child internalizing problems when entered before *(p*=0.06) and after *(p*=0.10) mothers' symptoms.	Postpartum PDep symptoms were significantly related to externalizing problems at entry *(sr2*=0.07, *p*=0.02) but less significant when entered after mothers' symptoms *(sr2*=0.05, *p*=0.07).	Postpartum PDep symptoms significantly predicted child outcome before *(sr2*=0.14, *p* < .001) and after *(sr2=0.*11, *p*=0.004) entering MDep.
Gross (2008) USA	Higher levels of PDep symptoms at age 2 were related to increased internalizing difficulties at age 4.	Significant associations found between PDep symptoms and mother-reported age 4 internalizing behaviour (*Beta*=0.31, *SE*=0.10, *p* < .01).	No association found.	Child noncompliance (age 2) and maternal education were associated with increased internalizing and externalizing behaviours.
Smith (2013) UK	PDep symptoms (3 & 36 months) predicted more father-reported child problems (51 months) but not mother-reported problems.	PDep symptoms significantly predicted child socio-emotional problems.	Postnatal PDep was a predictor for father-reports of child behaviour but was not significant when controlling for marital discord	Marital discord mediated the relationship between PDep symptoms and later child problems.
Cummings (2013) USA	PDep symptoms predicted offspring internalizing symptoms through fathers' negative expressiveness and child's emotional insecurity.	No direct effect of PDep on child internalizing symptoms.(non-significant standardised parameter estimate of 0.06)	‡	PDep symptoms were significantly associated (0.39, p<0.001) with paternal negative expressiveness. This was significantly associated with child emotional insecurity (0.19, p<0.01). This was significantly associated with child internalizing behaviours (0.25, p<0.01).
Keller (2009) USA	Fathers' covert negativity alongside mothers' overt conflict behaviours mediated the link between PDep and child internalizing difficulties.	PDep symptoms at T1 were associated with more overt destructive conflict behaviours at T2 (β=0.17, *p* < .05). This was associated with offspring internalizing symptoms at T3, (β=0.16, *p* < .05).	PDep symptoms were not associated with offspring externalizing behaviours.	PDep symptoms were significantly associated with covert marital negativity (β= 031, *p* < .01). Covert marital negativity was associated with increased offspring internalizing symptoms (β=0.33, *p* < .05).
Pilowsky (2014) USA	Depressed fathers' offspring were less likely to have internalizing or externalizing problems and a psychiatric disorder than offspring of depressed mothers (11% vs .37%; p=0.012).	Depressed fathers offspring were more likely to have lower prevalence of K- SADS symptoms, by child (0.2 vs. 1.2 symptoms; p=0.063) and parental report (0.6 vs 2.0 symptoms; p=0.075) than offspring of depressed mothers.	Depressed fathers offspring were less impaired (CIS of 6.5 vs. 11.6; p=0.009) than offspring of depressed mothers.	Most offspring symptoms decreased with treatment of MDep but not for offspring of treated depressed fathers.

Shelton (2008) UK	PDep was associated with negative child outcomes as a function of numerous mediators.	PDep symptoms were associated with interparental conflict. Interparental conflict was associated with child adjustment problems (r=0.00, p>0.10, to r=0.22, p>0.05).	No relations found between PDep and offspring externalizing symptoms.	PDep, MDep, parental insecurity, marital discord, parent-child relationship and child perceptions all resulted in various different outcomes regarding offspring internalizing and externalizing scores.
Reeb (2015) USA	PDep symptoms during early adolescence (13) predicted depression and anxiety symptoms in later adolescence (21).	PDep symptoms made significant contributions to children's depressive (H1; Beta=0.117, p=0.030) and anxiety (H2; Beta=0.134, p=0.002) symptoms at follow-up.	‡	MDep symptoms did not moderate the effects of PDEP symptoms on offspring depressive symptoms (R^2=0.001), t(384)=0.52, ns, or anxiety symptoms (R^2=0.003), t(384)=1.11, ns.
Reeb (2010) USA	PDep symptoms strongly predicted adolescent outcomes. Daughters experienced more depressive symptoms than sons (*Beta*=-0.249; $p < .05$).	PDep strongly predicted adolescent depressive symptoms.	‡	Paternal hostility increased the effect of PDep symptoms (β=0.159; $p < .01$).

Notes: MDep=Maternal Depression, PDep=Paternal Depression. ‡ denotes this outcome was not measured

Supplementary Table 1. Quality Rating for Included Studies

Author Year	Unbiased selection of cohort	Selection minimizes baseline differences in prognostic factors	Sample size calculation & Number	Analysis appropriate	Adequate description of the cohort	Validated method for ascertaining paternal depression	Validated method for ascertaining clinical outcomes	Outcome assessment blind to paternal status	Adequate follow-up period	Completeness of follow up	Control of confounders	Total Score
Van Batenburg-Eddes (2013)	Partial	N.A.	Partial	Yes	Partial	Yes	Yes	No	Yes	Yes	Partial	14
Hanington (2010)	Partial	N.A.	Partial	Yes	Yes	Yes	Yes	No	Yes	Partial	Partial	14
Ramchandani (2010)	Partial	N.A.	Partial	Yes	Partial	Yes	Yes	No	Yes	Partial	Yes	14
Ramchandani (2005)	Partial	N.A.	Partial	Yes	Partial	Yes	Yes	No	Yes	Partial	Yes	14
Gutierrez-Galve (2015)	Partial	N.A	Partial	Yes	Partial	Yes	Yes	No	Yes	Yes	Yes	15
Hanington (2012)	Partial	N.A.	Partial	Yes	Yes	Yes	Yes	No	Yes	Partial	Partial	14
Ramchandani (2008b)	Partial	Yes	Partial	Yes	Yes	Yes	Yes	No	Yes	Yes	Yes	18
Ramchandani (2008a)	Partial	N.A.	Partial	Yes	Partial	Yes	Yes	No	Yes	Partial	Yes	14
Van Den Berg (2009)	Partial	N.A.	Partial	Yes	Partial	Yes	Yes	No	Partial	Yes	Partial	13
Velders (2011)	Partial	N.A.	Partial	Yes	Partial	Yes	Yes	No	Yes	Partial	Partial	13
Ramchandani (2013)	Yes	Partial	Partial	Yes	Partial	Yes	Yes	No	Yes	Yes	Yes	17
Fletcher (2011)	Partial	N.A.	Partial	Partial	Partial	Yes	Partial	No	Yes	No	Yes	12
Carro (1993)	Partial	N.A.	No	Yes	Partial	Yes	Yes	No	Yes	Partial	Yes	13
Gross (2008)	Partial	N.A.	Partial	Yes?	Partial	Yes	Yes	No	Yes	Yes	No	13
Smith	Yes	N.A.	Yes	Yes	Yes	Yes	Yes	No	Yes	Yes	Yes	18

(2013)												
Cummings (2013)	Partial	N.A.	Partial	Yes	Yes	Yes	Yes	No	Yes	Yes	Yes	16
Keller (2009)	Partial	N.A.	Partial	Partial	Yes	Yes	Yes	No	Yes	Yes	No	13
Pilowsky (2014)	No	Yes	No	Partial	No	Yes	Yes	Partial	Yes	Partial	No	11
Shelton (2008)	Yes	N.A.	Partial	Yes	Partial	Yes	Yes	No	Yes	Yes	Yes	16
Reeb (2015)	No	N.A.	Partial	Yes	Yes	Partial	Yes	No	Yes	Yes	Partial	13
Reeb (2010)	No	N.A.	Partial	Partial	Yes	Yes	Yes	No	Yes	Yes	Partial	13

N.A. = This question was not applicable to this study design

PRISMA Checklist

Section/topic	Checklist item	Reported on page #
TITLE Title	Identify the report as a systematic review, meta-analysis, or both.	1
ABSTRACT Structured summary	Provide a structured summary including, as applicable: background; objectives; data sources; study eligibility criteria, participants, and interventions; study appraisal and synthesis methods; results; limitations; conclusions and implications of key findings; systematic review registration number.	1
INTRODUCTION Rationale	Describe the rationale for the review in the context of what is already known.	2-4
Objectives	Provide an explicit statement of questions being addressed with reference to participants, interventions, comparisons, outcomes, and study design (PICOS).	3 & 4
METHODS Protocol and registration	Indicate if a review protocol exists, if and where it can be accessed (e.g., Web address), and, if available, provide registration information including registration number.	4
Eligibility criteria	Specify study characteristics (e.g., PICOS, length of follow-up) and report characteristics (e.g., years considered, language, publication status) used as criteria for eligibility, giving rationale.	4
Information sources	Describe all information sources (e.g., databases with dates of coverage, contact with study authors to identify additional studies) in the search and date last searched.	4
Search	Present full electronic search strategy for at least one database, including any limits used, such that it could be repeated.	5 & 6
Study selection	State the process for selecting studies (i.e., screening, eligibility, included in systematic review, and, if applicable, included in the meta-analysis).	6
Data collection process	Describe method of data extraction from reports (e.g., piloted forms, independently, in duplicate) and any processes for obtaining and confirming data from investigators.	42
Data items	List and define all variables for which data were sought (e.g., PICOS, funding sources) and any assumptions and simplifications made.	42
Risk of bias in individual studies	Describe methods used for assessing risk of bias of individual studies (including specification of whether this was done at the study or outcome level), and how this information is to be used in any data synthesis.	7,19,35-41
Summary measures	State the principal summary measures (e.g., risk ratio, difference in means).	NA
Synthesis of results	Describe the methods of handling data and combining results of studies, if done, including measures of consistency (e.g., I^2) for each meta-analysis.	NA

Risk of bias across studies	Specify any assessment of risk of bias that may affect the cumulative evidence (e.g., publication bias, selective reporting within studies).	19, 40 & 41
Additional analyses	Describe methods of additional analyses (e.g., sensitivity or subgroup analyses, meta-regression), if done, indicating which were pre-specified.	NA
RESULTS Study selection	Give numbers of studies screened, assessed for eligibility, and included in the review, with reasons for exclusions at each stage, ideally with a flow diagram.	6
Study characteristics	For each study, present characteristics for which data were extracted (e.g., study size, PICOS, follow-up period) and provide the citations.	9-12
Risk of bias within studies	Present data on risk of bias of each study and, if available, any outcome level assessment (see item 12).	40&41
Results of individual studies	For all outcomes considered (benefits or harms), present, for each study: (a) simple summary data for each intervention group (b) effect estimates and confidence intervals, ideally with a forest plot.	NA
Synthesis of results	Present results of each meta-analysis done, including confidence intervals and measures of consistency.	NA
Risk of bias across studies	Present results of any assessment of risk of bias across studies (see Item 15).	40&41
Additional analysis	Give results of additional analyses, if done (e.g., sensitivity or subgroup analyses, meta-regression [see Item 16]).	NA
DISCUSSION Summary of evidence	Summarize the main findings including the strength of evidence for each main outcome; consider their relevance to key groups (e.g., healthcare providers, users, and policy makers).	20 & 22, 23 & 24
Limitations	Discuss limitations at study and outcome level (e.g., risk of bias), and at review-level (e.g., incomplete retrieval of identified research, reporting bias).	22&23
Conclusions	Provide a general interpretation of the results in the context of other evidence, and implications for future research.	23
FUNDING Funding	Describe sources of funding for the systematic review and other support (e.g., supply of data); role of funders for the systematic review.	With submission