Perinatal mental disorders are associated with increased risk of psychological and developmental disturbances in children. However, these disturbances are not inevitable. In this Series paper, we summarise evidence for associations between parental disorders and offspring outcomes from fetal development to adolescence in high-income, middle-income, and low-income countries. We assess evidence for mechanisms underlying transmission of disturbance, the role of mediating variables (underlying links between parent psychopathology and offspring outcomes) and possible moderators (which change the strength of any association), and focus on factors that are potentially modifiable, including parenting quality, social (including partner) and material support, and duration of the parental disorder. We review research of interventions, which are mostly about maternal depression, and emphasise the need to both treat the parent’s disorder and help with associated caregiving difficulties. We conclude with policy implications and underline the need for early identification of those parents at high risk and for more early interventions and prevention research, especially in socioeconomically disadvantaged populations and low-income countries.

Introduction

A substantial body of evidence now exists that shows an increase in a range of psychological and developmental disturbances in children. However, disturbances are not inevitable and effect sizes for these associations are mostly moderate or small. Therefore, why an association exists between a particular parental disorder and child outcome, and in what situations the risk is accentuated or ameliorated, is important to understand. Most investigators have tried to answer this question with attempts to elucidate possible mechanisms of transmission, that is, the role of mediating variables underlying associations between parent psychopathology and outcomes in their children, and possible moderators that change the strength of any association.

In this Series paper, we summarise the evidence about the different domains of development that are affected by perinatal mental disorders, and describe mediating and moderating variables, interventions, and implications of policies.

We mainly focus on depression and anxiety disorders during the perinatal period, but also assess the evidence for bipolar disorder, other psychoses, personality disorders, and eating disorders, although little research has been done about these disorders in relation to child outcomes. We prioritise findings from longitudinal studies (especially meta-analyses of such studies) for which, by contrast with cross-sectional designs, the temporal sequence of exposure and outcome is known. This knowledge helps to increase the potential for causal inference, although still not as clearly as experimental designs. We report evidence from studies that use reliable and valid measures of mental disorders of either self-report symptom questionnaires. Questionnaires are feasible in large population-level studies, often have well established thresholds to suggest clinically significant levels of symptoms, and can be used as continuous scales. We take a developmental perspective, report associations between perinatal disorders and offspring outcomes, beginning with fetal and proceeding through to adolescent outcomes. Although the focus of this Series is maternal disorders, when deeming the effect on the child, paternal disorders also need to be taken into account.
Fetal and neonatal outcomes
Two meta-analyses1,2 have assessed the association between antenatal depression and fetal and neonatal outcomes. Both reports showed that symptoms of antenatal depression are associated with an increased risk for premature delivery (<37 weeks’ gestation). One reported that studies controlling for women taking antidepressant drugs or smoking generated small (and non-significant) odds ratios,1 whereas the second2 concluded that “the summary relative risk was comparable for depressed women treated and not treated with antidepressants”2. Antidepressants or smoking can be markers for more severe depression1 and the effects of antenatal depression on prematurity (and low birthweight) were strong in studies where depression was defined by a disorder,2 suggesting that severity of disorder is important.

A discrepancy was noted between the findings relating to depression and low birthweight; one meta-analysis1 reported a modest association whereas the other reported a non-significant association.1 However, in the meta-analysis reporting no association,1 studies from low-income and middle-income countries (LMICs) were excluded from analyses. In geographically diverse studies incorporated within the second meta-analysis,2 moderator analyses showed that the association with low birthweight was higher in LMICs than in high-income countries (HICs). Low birthweight might therefore be a substantial factor in LMICs, but not in HICs, except possibly in socio-economically disadvantaged communities.2

Antenatal depression was not associated with pre-eclampsia, Apgar scores, or admission to neonatal intensive-care units; for intrauterine growth restriction, antenatal depression was associated with an increased risk but only in LMICs.1,2 Additional researchers in a meta-analytic review4 examined associations between these outcomes and use of antidepressants during pregnancy, but reported weak associations after accounting for confounding.4

Some fetal and neonatal outcomes have been investigated mostly in association with the use of antidepressants rather than with a diagnosis of depression1 usually because of the availability of prescription data in large population databases studies,1 making it difficult to disentangle the effect of antidepressants, life style confounders, and depression.

Evidence is inconsistent for associations between antenatal anxiety and adverse fetal outcomes. A meta-analysis5 reported small, non-significant correlations for most outcomes other than pregnancy-related anxiety and young gestational age at birth. A meta-analysis6 showed slight associations between anxiety and both preterm birth and low birthweight, although birthweight was not significant when adjusting for confounders.6 Both meta-analyses were limited by the paucity of studies and the frequent comorbidity of anxiety with depression and life stressors, which could confound associations.7

Other mental health problems during pregnancy have been associated with fetal and neonatal outcomes. Several studies suggest that maternal anorexia nervosa (active or past) is associated with low birthweight, although associations are inconsistent with high rates of prematurity.8 Schizophrenia has also been associated with an increased risk of low birthweight, preterm delivery, stillbirth, and infant death within 1 year after birth.9,10 This risk might be partly attributable to environmental factors associated with adversity, including smoking, poverty, poor nutrition, and substance misuse.9 Risks of such adverse outcomes do not substantially differ between infants according to whether their mothers had a history of a psychiatric admission for schizophrenia or affective disorders. Risks for both groups with either schizophrenia or affective disorders are lower than they are for infants of mothers with substance misuse disorders.9

Search strategy and selection criteria
<table>
<thead>
<tr>
<th>Study</th>
<th>Time of first exposure</th>
<th>Exposure measure (maternal)</th>
<th>Outcome measure (child)</th>
<th>Age of children at follow-up</th>
<th>Results</th>
<th>Strengths and limitations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gerardin 2011; n=164, France</td>
<td>Antenatal</td>
<td>Clinical interviews, DSM-IV</td>
<td>NBAS</td>
<td>ITSEA</td>
<td>12 months</td>
<td>Antenatal depression was associated with increased externalising and internalising symptoms on the ITSEA and poor motor and regulation skills on NBAS</td>
</tr>
<tr>
<td>Blair 2011; n=120, USA</td>
<td>Antenatal</td>
<td>Not reported</td>
<td>STAI; PSA</td>
<td>Not reported</td>
<td>ECIQ</td>
<td>2 years</td>
</tr>
<tr>
<td>Velders 2011; n=2698, Netherlands (generation-R)</td>
<td>Antenatal</td>
<td>Not reported</td>
<td>BSI</td>
<td>Not reported</td>
<td>CBCL (cutoff)</td>
<td>3 years</td>
</tr>
<tr>
<td>Davis 2012; n=178, USA</td>
<td>Antenatal</td>
<td>Salivary cortisol</td>
<td>PSS; CES-D; STAI; PSA</td>
<td>Not reported</td>
<td>CBCL anxiety scale</td>
<td>6–9 years</td>
</tr>
<tr>
<td>Barker 2011; n=2298, UK (ALSPAC)</td>
<td>Antenatal</td>
<td>Not reported</td>
<td>EPDS, CROWN- CRISP index</td>
<td>DAWBA, internalising disorders</td>
<td>Not reported</td>
<td>8 years</td>
</tr>
<tr>
<td>Leis 2013; n=2891, UK (ALSPAC)</td>
<td>Antenatal</td>
<td>Not reported</td>
<td>EPDS, CROWN-CRISP index</td>
<td>Not reported</td>
<td>SDQ mother and teacher report</td>
<td>10–11 years</td>
</tr>
<tr>
<td>Pawlby 2009; n=127, UK</td>
<td>Antenatal</td>
<td>Pregnancy: clinical interview schedule; at age 16 years: SADS-L</td>
<td>Depression, diagnosis clinical interview DSM-IV, CAPA</td>
<td>Not reported</td>
<td>11 years and 16 years</td>
<td>Antenatal depression was associated with increased risk of depression for child at age 16 years (OR 4.70 [95% CI 1.60–13.86])</td>
</tr>
<tr>
<td>Kershaw 2012; n=192, Finland</td>
<td>Antenatal</td>
<td>Not reported</td>
<td>EPDS</td>
<td>Not reported</td>
<td>CBCL, YSR</td>
<td>16 years or 17 years</td>
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(Table 1 continues on next page)
(Continued from previous page)

<table>
<thead>
<tr>
<th>Time of first exposure</th>
<th>Exposure measure (maternal)</th>
<th>Outcome measure (child)</th>
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<tr>
<td>Antenatal</td>
<td>Not reported</td>
<td>EPDS</td>
<td>18 years</td>
<td>Association between 1 SD increase in maternal EPDS score and child depression at age 18 years: antenatal EPDS (OR 1.23 [95% CI 1.03–1.44]); postnatal EPDS in mothers with low education (OR 1.26 [95% CI 1.06–1.50]) or high education (OR 1.09 [95% CI 0.88–1.36]).</td>
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<td>Depression diagnosis</td>
<td>Not reported</td>
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<td>clinical interview</td>
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<td>CIS-R, ICD10</td>
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<tr>
<td>Postnatal</td>
<td>Not reported</td>
<td>Still Face</td>
<td>3–8 months</td>
<td>Postnatal anxiety was associated with more distress to novelty and reported emotional problems</td>
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<td>Paradigm, Cortisol</td>
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<td>reactivity in response</td>
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<td>to Still Face procedure*</td>
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Series

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Emotional (internalising) difficulties and social development

Children’s emotional and behavioural difficulties are often conceptualised as being either internalising or externalising. Internalising difficulties include symptoms or diagnoses of depression and anxiety (eg, separation anxiety and phobias). By social development, we refer to a child’s development of social skills, such as perspective taking, empathy, and cooperation.

Longitudinal studies have shown that antenatal depression is associated with an increased risk for child emotional problems,13,15,18 self-reported symptoms and depressive disorder are associated with an increased risk of clinical depression in late adolescence.9,20

Increased risks of emotional problems in children of mothers with postnatal depression have long been noted.15 Detailed records of mother and infant behaviours showed that infants of mothers with postnatal depression have an increased risk of difficulties in early emotional regulation and social behaviour.8,15 Longitudinal studies provide evidence for associations between postnatal depression and emotional outcomes across domains and age ranges, including internalising disorders, poor social competence in school years, and an increased risk of depression during adolescence (table 1).25,27,29,36,39

Risks are associated with both symptoms of depression and depressive disorder, although effect sizes are generally large in children of mothers diagnosed with depressive disorder (table 1).

In view of the high extent of the association between depression in the antenatal and postnatal periods, large numbers of participants are required to provide sufficient power to assess whether the risks associated with antenatal and postnatal depression are independent of each other.21,32 One study31 reported independent associations between symptoms of antenatal and postnatal depression and offspring depression at age 18 years. Maternal level of education moderates the association between symptoms of antenatal and postnatal depression and emotional problems in the offspring (table 1).

Fewer investigations have been done to assess associations between antenatal and postnatal anxiety and child emotional difficulties than have for perinatal...
<table>
<thead>
<tr>
<th>Time of first exposure</th>
<th>Exposure measure (maternal)</th>
<th>Outcome measure (child)</th>
<th>Age of children at follow-up</th>
<th>Results</th>
<th>Strengths and limitations</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Clinical interview or objective assessment</td>
<td>Questionnaire (parent or child reported)</td>
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<tr>
<td>Pemberton 2010; n=361, USA</td>
<td>Antenatal Not reported</td>
<td>BDI Not reported</td>
<td>CBCL 27 months</td>
<td>For the birth mother, prenatal-postnatal depressive symptoms were associated with externalising (β 0.09, p=0.10); for an adoptive mother postnatal depressive symptoms associated with child externalising (β 0.14, p=0.05)</td>
<td>Openness in adoption, sex of child, parent age, prenatal complications, birth, adoptive parent antisocial behaviour, and infant characteristics</td>
</tr>
<tr>
<td>Velders 2011; n=2698, Netherlands (generation-R)</td>
<td>Antenatal Not reported</td>
<td>BSI Not reported</td>
<td>CBCL 3 years</td>
<td>Evidence that child externalising difficulties were associated with antenatal depression (OR 1.19 [95% CI 1.09–1.30])</td>
<td>See table 1</td>
</tr>
<tr>
<td>Van Batenburg-Eddes 2013; n=2280, Netherlands (generation-R); UK (ALSPAC)</td>
<td>Antenatal Not reported</td>
<td>EPDS Not reported</td>
<td>SDQ, CBCL 3 years</td>
<td>Child attention problems were associated with antenatal depression: generation-R (OR 1.23 [95% CI 1.05–1.43]); ALSPAC (OR 1.33 [1.19–1.48]); and antenatal anxiety: generation-R (OR 1.24 [1.06–1.46]); ALSPAC (OR 1.32 [1.19–1.47])</td>
<td>Sex of child, birthweight, birth order, ethnic origin, and age at questionnaire; maternal smoking and alcohol use during pregnancy, and maternal level of education; family income; and adjusted for depression or anxiety of a partner during pregnancy</td>
</tr>
<tr>
<td>Barker 2011; n=3298, UK (ALSPAC)</td>
<td>Antenatal Not reported</td>
<td>EPDS Not reported</td>
<td>DAWBA: externalising disorders</td>
<td>Child externalising behaviour was associated with antenatal depression (no association with anxiety; β 0.09, p=0.05); and postnatal depression (β 0.09, p=0.05)</td>
<td>Adjusted for a cumulative risk score derived from SES, marital status, teenage mother, substance use, criminal background, and antenatal and postnatal anxiety and depression, mutually adjusted</td>
</tr>
<tr>
<td>Korhonen 2012; n=192, Finland</td>
<td>Antenatal Not reported</td>
<td>EPDS Not reported</td>
<td>CBCL, YSR 16-17 years</td>
<td>Antenatal and postnatal depression was associated with higher externalising score on the YSR, but not the CBCL</td>
<td>Later maternal depression at child age of 16-17 years</td>
</tr>
<tr>
<td>Hay 2010; n=120, UK</td>
<td>Antenatal Clinical interview, ICD-9 CIS</td>
<td>Not reported</td>
<td>DSM-IV: conduct disorder or antisocial behaviour</td>
<td>Associations between antenatal depression and offspring antisocial behaviour (OR 2.46 [95% CI 1.15–5.30])</td>
<td>Associations were similar when including antenatal anxiety, smoking, drinking, postnatal and later depression, and social adversity</td>
</tr>
<tr>
<td>Conroy 2012; n=200, UK</td>
<td>Postnatal Clinical interview, DSM-IV</td>
<td>Not reported</td>
<td>ITSEA (SD 9) 18 months</td>
<td>Postnatal depression was associated with infant dysregulated behaviour (β 5.12 [95% CI 1.19–21.47])</td>
<td>Occupation, ethnic origin, partner status, later maternal depression, sex of infant, maternal sensitivity towards their infant</td>
</tr>
<tr>
<td>Galera 2011; n=2057, Canada</td>
<td>Postnatal Not reported</td>
<td>CES-D scale Not reported</td>
<td>Not reported 17 months to 8 years, trajectories of ADHD symptoms</td>
<td>Postnatal depression was associated with an increased risk of high trajectories of hyperactivity-impulsivity and inattention symptoms through childhood (OR 1.35 [95% CI 1.18–1.54])</td>
<td>Prematurity and birthweight of child; prenatal smoking, drug and alcohol use; family structure, maternal age, income, maternal education, parenting factors, and paternal depression</td>
</tr>
</tbody>
</table>

*(Table 2 continues on next page)*
depression. Depression and anxiety are substantially comorbid, and thus associations attributed to one, might include causes associated with the other. Several studies showed self-reported symptoms of antenatal anxiety are associated with internalising symptoms in childhood. After symptoms of anxiety for both antenatally and postnatally were accounted for, no independent effect of antenatal depression occurred.

A systematic review reported associations between postnatal symptoms of anxiety and child emotional difficulties. Comparisons between depression and anxiety disorders in a few studies were assessed by diagnostic interviews; some specificity was noted in the nature of the early effects of anxiety disorders on infant distress to novelty (which is linked to behavioural inhibition), but impairment in fear regulation was restricted to infants of mothers with depression. Infants of mothers with post-traumatic stress disorder have an increased risk of difficulties in emotional regulation even after adjustment for symptoms of depression.

Although research for other disorders is scarce, one report shows that children of mothers admitted to mother and baby units with severe postnatal disorders are at an increased risk of a psychiatric (mainly emotional) disorder in adulthood compared with siblings who were not exposed to a postnatal episode.

**Behavioural (externalising) difficulties**

Externalising difficulties include attention deficit hyperactivity disorder, oppositional defiant disorder, and conduct disorder, or symptoms of any of these. Several studies have reported associations between antenatal depression and a child’s externalising behaviour. When a child is adopted, including when a child is adopted, a small study reported an association between antenatal depressive disorder and

### Table 2: Behavioural outcomes

<table>
<thead>
<tr>
<th>Exposure measure (maternal)</th>
<th>Outcome measure (child)</th>
<th>Age of children at follow-up</th>
<th>Results</th>
<th>Strengths and limitations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time of first exposure</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Clinical interview or objective assessment</td>
<td>Self-report</td>
<td>Objective assessment or interview</td>
<td>Questionnaire (parent or child reported)</td>
</tr>
<tr>
<td></td>
<td>Postnatal depression</td>
<td>Pitt depression inventory</td>
<td>Not reported</td>
<td>Richman child behaviour scale</td>
</tr>
<tr>
<td></td>
<td>Postnatal depression</td>
<td>EPDS</td>
<td>Not reported</td>
<td>Rutter revised preschool scales</td>
</tr>
<tr>
<td></td>
<td>Postnatal depression</td>
<td>BDI</td>
<td>Not reported</td>
<td>Teacher report social competence, CBCL</td>
</tr>
</tbody>
</table>

Antenatal depression is associated with disorganised attachment (a form of insecure attachment), independently of postnatal depression. In two meta-analyses, 65,66 postnatal depression was associated with an increased risk of insecure (especially disorganised) mother–infant attachment. This association is low or non-significant in community samples in relation to clinical samples and when depression is measured by self-report, in relation to diagnostic interviews. 41,66

In a small sample of mothers with severe psychopathology and admitted to hospital postnatally, infants of mothers with unipolar depression, but not manic disorders, were more likely to show insecure attachment at aged 12 months than were infants of mothers without a perinatal disorder.67

**Cognitive development**

Antenatal depression (both self-reported symptoms and the disorder) is associated with low levels of general cognitive development, including IQ scores in childhood. However, effect sizes are generally small65,66 and not all studies showed a significant association.41 Postnatal depression has shown consistent associations (including studies from LMICs40–42) with a range of cognitive outcomes in early childhood, including infant ability to learn, achievement of developmental milestones, and language and general cognitive development. 26,37,38,14 Persistence of postnatal depression seems to be of particular importance in relation to cognitive development (table 4).69–71

**Attachment**

Attachment is when a young child uses a caregiver as a secure base from which to explore and, when necessary, as a haven of safety and source of comfort. 64 This attachment is based on early experiences with caregivers’ extent of responsiveness with the child.

<table>
<thead>
<tr>
<th>Time of first exposure</th>
<th>Exposure measure (maternal)</th>
<th>Outcome measure (child)</th>
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<th>Results</th>
<th>Strengths and limitations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antenatal</td>
<td>Diagnostic interview DSM-IV, SCID-IV</td>
<td>BDI-II</td>
<td>Strange situation*</td>
<td>12 months</td>
<td>Antenatal depression was associated with disorganised attachment ($\beta$ 0.21 [SE 0.09], $p&lt;0.05$; OR 1.23)</td>
</tr>
<tr>
<td>Postnatal</td>
<td>Diagnostic interview DSM-IV, SCID-IV</td>
<td>Not reported</td>
<td>Strange situation*</td>
<td>18 months</td>
<td>Postnatal depression was associated with insecure attachment style (OR 2.98 [95% CI 1.26–7.01])</td>
</tr>
</tbody>
</table>

**Table 3:** Attachment outcomes

<table>
<thead>
<tr>
<th>Exposure measure (maternal)</th>
<th>Outcome measure (child)</th>
<th>Age of children at follow-up</th>
<th>Results</th>
<th>Strengths and limitations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical Interview</td>
<td>Self-report</td>
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<td>or objective assessment</td>
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DSM=diagnostic and statistical manual. SCID=structured clinical interview for DSM-IV. BDI=Beck depression inventory. CIDI=composite international diagnostic interview. BSI=brief symptom inventory. EPDS=Edinburgh postnatal depression scale. *An observed experimental procedure where an infant’s reactions to separation and reunion with their mother are used to categorise so-called security of mother-infant attachment.

antisocial behaviour in adolescence that was independent of postnatal depression. Several large longitudinal studies, including from LMICs, provided supportive evidence that symptoms and disorders of postnatal depression are associated with a child’s externalising behaviour, particularly symptoms of attention deficit hyperactivity disorder, up to age 16 years.23–25 In a large study of antenatal and postnatal maternal symptoms of depression, associations between antenatal symptoms and persistent childhood attention deficit hyperactivity disorder symptoms were diminished after postnatal symptoms were accounted for.12

Self-reported symptoms of maternal anxiety both antenatally21,26 and postnatally27 are associated with externalising disorders in childhood.64 We found no studies that used clinical diagnostic interviews therefore associations between specific anxiety disorders and children’s externalising difficulties remain to be established.

Few studies have investigated the effect of other perinatal psychopathology on behavioural outcomes. Results of a small study46 suggested that infants of mothers with comorbid postnatal depression and personality disorder had reported dysregulated behaviour.

**Attachment**

Attachment is when a young child uses a caregiver as a secure base from which to explore and, when necessary, as a haven of safety and source of comfort.4 This attachment is based on early experiences with caregivers’ extent of responsiveness with the child.
Studies of the longer term effects of postnatal depression on cognitive functioning have been inconsistent. Results from a large study\(^a\) showed an association between symptoms of postnatal depression and very small decreases in IQ scores at age 8 years, which became non-significant after accounting for maternal depressive symptoms after the postnatal period. Another large study\(^7\) reported no association between postnatal depression and low maths achievement at age 11 years and a small UK study\(^8\) showed an association between postnatal depressive disorder and academic achievement in adolescence.

Several studies have investigated perinatal depression and anxiety by use of self-reporting. Associations with poor child cognitive outcomes were specific to symptoms of depression; these included studies of antenatal\(^9\) and postnatal\(^10,11\) symptoms. Other studies\(^12\) have reported that symptoms of antenatal anxiety, rather than depression, were associated with poor exam achievements at age 11 years. An association was reported between antenatal symptoms of anxiety and impaired executive function abilities.\(^14\)

We found no studies that reported associations between other disorders in mothers during the perinatal period and their child’s cognitive development.

**Child physical growth and development**

Evidence is emerging that poor perinatal maternal mental health, especially in women at a socioeconomic disadvantage, is linked to poor infant growth. Although some studies, especially those done in HICs, have not noted such associations or only in subgroups,\(^69-71\) increasing data from populations living in LMICs suggests that perinatal depression is associated with underweight and stunting in infancy,\(^15-24\) with effects persisting up to school age of 5 years.\(^25-27\) Children of mothers with chronic depression (multiple episodes) might be particularly at risk of stunting or being underweight in LMICs.\(^26,27\) By contrast, a systematic review\(^69\) of studies in HICs reported chronic depression after childbirth was associated with the child being overweight.\(^7\) Postnatal depression in LMICs is associated with high rates of diarrhoeal diseases in children,\(^28\) which could contribute to their poor growth. Of the few studies of other perinatal disorders, some evidence exists that children of mothers with eating disorders, mainly those with anorexia nervosa, are at increased risk of poor growth.\(^40\)

**Feeding, eating habits, and attitudes**

Children of mothers who had an eating disorder during pregnancy or in post partum are susceptible to difficulties during infancy, such as mealtime conflict.\(^46\) Two longitudinal studies\(^47,48\) reported an increased risk for negative outcomes in children of mothers with postnatal eating psychopathology. One of these studies\(^41\) reported increased mealtime conflict at 5 years of age, and concerns about body shape and weight and use of dietary restraint at 10 years of age. The second study showed that eating disturbance at age 5 years was predicted by many postnatal maternal variables, such as body dissatisfaction and the internalisation of the thin ideal\(^42\) and at age 8 years, high postnatal maternal dietary restraint predicted high body dissatisfaction and dieting behaviours only in girls.\(^43\)

**Fathers**

Traditionally, the mother’s mental health received most attention. However, recognition of the importance of father’s mental health is increasing.\(^44\) Fathers can affect their children directly via quality of their interactions or genetic effects, or indirectly via their support to the mother and family environment. A large Norwegian population study\(^44\) reported an association between symptoms of paternal antenatal depression and poor socioemotional and behavioural development of children at age 36 months;\(^44\) postnatal symptoms were not assessed. However, another study\(^47\) reported no evidence that paternal symptoms of antenatal depression were associated with child depression at age 18 years, rather only an association with paternal symptoms of depression postnatally.\(^21\)

Symptoms of paternal postnatal mental disorders are associated with an increased risk of emotional and behavioural disorders for young children,\(^15,26\) difficulties with their language development,\(^45\) and depression at age 18 years.\(^7\) Paternal and maternal depression in the postnatal period seem to have similar effects on behavioural outcomes, whereas maternal depression has a greater risk for emotional difficulties.\(^15,32,55\) In a meta-analysis\(^46\) of associations between both maternal and paternal disorders and their child’s internalising or externalising difficulties across childhood, the younger the age of the child at the time of study, the greater the effect sizes for associations with maternal depression, but the reverse was reported for paternal depression.\(^46\)

**Mechanisms**

**Overview**

The figure shows a model summarising possible mechanisms underlying the associations between parental psychiatric disorders and child outcomes. Biologically mediated effects of antenatal disorders (through in-utero effects) would be specific to mothers, whereas effects occurring postnatally and genetic effects might occur if either parent is affected by a disorder.

**Genetic factors**

Shared genetic risk factors probably account for part of the association between parental mental disorders at any time (including the perinatal period) and child susceptibilities. However, evidence exists for a substantial environmental contribution in the cause of mental disorders.\(^49\) Correlations
<table>
<thead>
<tr>
<th>Time of first exposure</th>
<th>Exposure measure (maternal)</th>
<th>Outcome measure (child)</th>
<th>Age of child at follow-up</th>
<th>Results</th>
<th>Strengths and limitations</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Koutra 2012; n=223; Greece (Rhea study)</strong></td>
<td>Antenatal not reported</td>
<td>EPDS, personality trait (EPQ-R)</td>
<td>18 months</td>
<td>Lower cognitive Bayley score was associated with antenatal depression ($\beta = -5.45 [95% CI -10.44$ to $-0.46]$), and postnatal depression ($\beta = -5.80 [-11.65$ to $-0.05]$); no effect of trait anxiety for cognitive development, no associations with maternal symptoms and Bayley language scores</td>
<td>Maternal age, maternal education, gestational age, quality of assessment, sex of child, and duration of breastfeeding, parent employment status</td>
</tr>
<tr>
<td><strong>Tse 2010; n=1030; USA</strong></td>
<td>Antenatal not reported</td>
<td>EPDS</td>
<td>3 years</td>
<td>No evidence for an association between antenatal depression and PPVT score ($\beta = -0.7 [95% CI -3.6$ to $2.3]$); Bayley III ($\beta = -0.5 [-1.7$ to $1.3]$)</td>
<td>Maternal race or ethnic origin, age, and education, parity, household income, pregnancy intention, partnership status, partner education, alcohol use and smoking, birthweight for gestational age</td>
</tr>
<tr>
<td><strong>Barker 2011; n=2998; UK (ALSPAC)</strong></td>
<td>Antenatal not reported</td>
<td>EPDS and Crown Crisp index</td>
<td>8 years</td>
<td>Low IQ was associated with antenatal depression ($\beta = -0.31$, p&lt;0.05), and postnatal depression ($\beta = -0.04$, p=0.05); no association with anxiety</td>
<td>Personality traits associated with infant neurodevelopmental scores</td>
</tr>
<tr>
<td><strong>Evans 2012; n=6735; UK (ALSPAC)</strong></td>
<td>Antenatal not reported</td>
<td>EPDS</td>
<td>8 years</td>
<td>Antenatal depression was associated with a small decrease in IQ points, which attenuated after adjustments ($\beta = -0.64 [95% CI -1.68$ to $0.40]$); no association between postnatal depression independent of other time periods</td>
<td>Not reported</td>
</tr>
<tr>
<td><strong>Hadley 2008; n=431; rural Ethiopia</strong></td>
<td>Postnatal not reported</td>
<td>HSCL</td>
<td>3 months and 24 months</td>
<td>Maternal total symptoms were associated with low developmental scores ($\beta = -0.003 [95% CI -0.001$ to $0.000$]; separating symptoms into depression and anxiety, depression ($p=0.01$) and anxiety ($p=0.01$) was cause of the noted association</td>
<td>Not reported</td>
</tr>
<tr>
<td><strong>Galler 2000; n=226; Barbados</strong></td>
<td>Postnatal not reported</td>
<td>Zung scale*</td>
<td>6 months</td>
<td>Negative correlation between postnatal mood at age 7 weeks and total Zung score at age 6 months ($r=0.32$, p&lt;0.05)</td>
<td>Socioeconomic and home environment factors derived from Socioeconomic and Home Environment Questionnaire gestational age, parity, and birth order, infant weight at 3 months, length at 3 months and 6 months</td>
</tr>
<tr>
<td><strong>Patel 2003; n=171; India</strong></td>
<td>Postnatal not reported</td>
<td>EPDS</td>
<td>6 months</td>
<td>Postnatal depression was associated with greater risk of poor development in infant (OR $3.3 [95% CI 1.2-8.8]$)</td>
<td>Birthweight and maternal education</td>
</tr>
<tr>
<td><strong>Hamadani 2012; n=488; Bangladesh</strong></td>
<td>Postnatal not reported</td>
<td>EPDS</td>
<td>6 months and 12 months</td>
<td>Weak correlations between postnatal depression and impaired mental development ($r=0.04$, p=0.05) and milestones ($r=0.08$, p=0.05)</td>
<td>Association attenuated once family care indicators were included</td>
</tr>
</tbody>
</table>

*Table 4 continues on next page*
(Continued from previous page)

<table>
<thead>
<tr>
<th>Time of first exposure</th>
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<tr>
<td>Quevedo 2012; n=296; Brazil 49</td>
<td>Clinical interview, DSM-IV, MINI</td>
<td>Bayley III language score</td>
<td>Not reported</td>
<td>Postnatal depression duration (depressed on no, one, or two occasions in first year) associated with lower language Bayley scores (β = -2.87 [95% CI -5.01 to 0.64])</td>
<td>Maternal age, sex of child, parity, primary caregiver; Duration of exposure of postnatal depression associated with more impaired language</td>
</tr>
</tbody>
</table>

Kaplan 2011; n=134; USA 49

| Time of first exposure | Clinical interview, SCID, DSM-IV | Infant learning by observed conditioned attention task | Not reported | Infants aged 1 year of currently depressed mothers with relatively longer-duration depressive episodes (ie, perinatal onset) showed significantly poorer learning than infants aged 1 year of currently depressed mothers with relatively shorter duration depressive episodes (non-perinatal onset) | Not reported |

Conroy 2012; n=200; UK 48

| Time of first exposure | Clinical interview, DSM-IV, SCID-I, NP | Bayley II, MDI (mental subscale) ITSEA | Not reported | Postnatal depression associated with impaired mental development (β = -7.26 [95% CI -13.04 to -1.47], p<0.05) | Occupation, ethnic origin, partner status, later maternal depression, sex of infant, maternal sensitivity |

Sutter-Dalray 2011; n=598; France 50

| Time of first exposure | Clinical interview, DSM-IV, MINI | Bayley II (MDI) | Not reported | Sex of child, maternal age, maternal education, income, parity | Mediated by later depression in mother |

Kersten-Alvarez 2012; n=142 (29 with depression); Netherlands 49

| Time of first exposure | Clinical interviews; DSM-IV | PPVT-R | Teacher report social competence; CBCL | Early school (child average age of 5 years) | See table 1; postnatal depression was associated with low verbal intelligence only in girls |

Letourneau 2013; n=1033 (age 4-5 years); n=247 (age 11 years); Canada 50

| Time of first exposure | Clinical interviews; DSM-IV | Language, maths achievement | Not reported | Association between postnatal depression and low language at age 5 years (OR 1.39, p<0.05) and low maths achievement at age 11 years (OR 1.17, p<0.05) | SES, income adequacy, mothers’ years of education, family structure, and family functioning |

Galler 2004; n=92; Barbados 50

| Time of first exposure | Not reported | Zung scale* | Exam scores at school entry (Eleven-Plus examination) | Not reported | Maternal anxiety (not depression) at 7 weeks correlated with overall exam score (r = -0.25, p<0.05) |

Murray 2010; n=89; UK 50

| Time of first exposure | Clinical interviews SPI, SADS-L | Bayley scales MDI at 18 months, McCarthy scale at age 3 years, WISC-V at age 8 years, GCSE exam results at the end of school age 15 years | Not reported | Postnatal depression associated with lower exam grade points (GCSEs) in children, particularly in boys whose mothers have postnatal depression than in boys whose mothers did not have postnatal depression | Maternal IQ and cognitive support, social class considered but not included in final model |


Table 4: Cognitive outcomes
Epigenetics

Epigenetic change (modification of gene expression, such as through methylation, without changing the genetic sequence) is a mechanism proposed to explain the long-lasting effects of early life experiences, including the perinatal environment, on biological and behavioural phenotypes.

Investigations have mainly been derived from animal studies; however, preliminary human studies report that antenatal stress and anxiety increases glucocorticoid

<table>
<thead>
<tr>
<th>Time of first exposure</th>
<th>Exposure measure (paternal)</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Kvaløvaag 2013; Norway (MoBa study)</td>
<td>Antenatal</td>
<td>SCL-5</td>
<td>Not reported</td>
<td>SDQ, ITSEA, CBCL (operationalised into three summary scores: behavioural difficulties, emotional difficulties, and social functioning)</td>
<td>3 years</td>
</tr>
<tr>
<td>Velders 2011; Netherlands (generation-R)</td>
<td>Antenatal</td>
<td>BSI</td>
<td>Not reported</td>
<td>CBCL (internalising problem binary)</td>
<td>3 years</td>
</tr>
<tr>
<td>Pearson 2013; UK (ALSPAC)</td>
<td>Antenatal</td>
<td>EPDS</td>
<td>CIS-R</td>
<td>Not reported</td>
<td>18 years</td>
</tr>
<tr>
<td>Fletcher 2011; Australia</td>
<td>Postnatal</td>
<td>K6</td>
<td>Not reported</td>
<td>SDQ (binary)</td>
<td>4–5 years</td>
</tr>
<tr>
<td>Ramchandani 2008; UK (ALSPAC)</td>
<td>Postnatal</td>
<td>EPDS</td>
<td>DAWBA, DSM-IV</td>
<td>Not reported</td>
<td>6 years and 7 years</td>
</tr>
</tbody>
</table>


Table 5: Outcomes for fathers
receptor (involved in stress responses) methylation (ie, silencing) in children.2 Postnatally, maternal mental disorders might change maternal caregiving, which in animal studies lead to epigenetic changes in offspring.19 Thus, epigenetic pathways might mediate the association between maternal disorders and child outcomes, although this hypothesis has not been formally tested.

Timing of perinatal mental health problems
Mental disorders or symptoms in pregnancy often continue after birth, thus raising questions about the contribution of antenatal and postnatal exposure and the extent to which their contributions are distinct, interactive, or cumulative. In particular whether antenatal effects are due to the direct effect on fetal development or because antenatal symptoms continue postnatally.

Mediating and moderating factors
Overview
A mediator is part of the causal pathway whereas moderators change the strength of association between exposure and outcome, which is sometimes referred to as effect modification.46 Some factors can act as either mediators or moderators.

Fetal programming
Animal studies provide evidence that antenatal stress leads to maladaptive cognitive and behavioural changes in rodent offspring.44 Often these changes are attributed to the effect of increased cortisol concentrations or other biological results due to stress on the offspring’s brain development in utero.45 However, findings of positive associations are inconsistent in human beings between antenatal depression or anxiety and increased cortisol concentrations,22 implicating a more complex mechanism. For example, anxiety in pregnancy has been associated with the downregulation of the enzyme that metabolises cortisol and protects the baby in utero from excessive cortisol concentrations.26 Thus, decreased expression of this enzyme might be a mechanism by which antenatal anxiety affects the fetus, even in the presence of normal concentrations of maternal cortisol. Importantly, although the present model is based on adverse effects of antenatal stress, clinical data suggests the possibility of a non-linear association between antenatal depression and fetal stress.27 This postulation is consistent with some animal models, which indicates that some exposure to stress might be advantageous, with levels at both extremes leading to more negative fetal responses.28

Maternal programming
Mental disorders during pregnancy might disrupt neurocognitive changes in the mother that prepare women to respond towards their infants, known as maternal-programming.26 Antenatal depression is associated with reduced maternal responsiveness towards infants independently of postnatal depression.25 In rodents, antenatal stress reduces nurturing behaviour of maternal care.26 This disruption to maternal programming might account for evidence that antenatal depression is associated with insecure attachment styles independently of postnatal depression29 and an increased risk of children being exposed to maltreatment.25

Chronic exposure
Several studies have shown that effects of perinatal mental disorders are mediated by continued or recurrent exposure to the disorder during the child’s life (tables 1—5).29,54,58 That is, some variance in the association between perinatal disorder and child outcomes results from the persistence of the parent’s disorder.

Interparent conflict
Perinatal mental disorders are associated with an increased risk of interparent conflict, relationship breakdown, and domestic violence61 which, in turn, negatively affects children.100 Evidence shows that interparent and family conflict mediates the association between symptoms of postnatal depression and child externalising behaviours.56

Parenting
A body of evidence suggests that the most important potential mediator is the quality of parenting. Data for each component of this mediating pathway shows first, perinatal disorders and symptoms can compromise the quality of parenting; second, compromised early parenting is associated with disturbances in child development; and third, disrupted parenting mediates the association between perinatal mental health and child outcomes. Symptoms of mental disorders can affect a person’s ability to respond to their environment, and thereby their parenting capabilities. For example, rumination and mood disturbance make it difficult for parents to focus their attention on, and provide contingent responsiveness towards, their infant’s cues.112 Several aspects of parenting are associated with postnatal disorders, including disengagement and withdrawal; missing of infant cues; poor responsiveness and particularly insufficient contingent responsiveness; intrusiveness;15,54 and difficulty in thinking about and appreciating their children’s perspectives, thoughts, and feelings.108 Often, the term “lack of sensitivity” is used to incorporate these sometimes overlapping parenting capacities.100 Most of this research has included maternal depression, but some evidence shows that different disorders are associated with specific disruption to maternal parenting. Mothers with eating disorders are more likely to use over-controlling and intrusive parenting, especially during mealtimes90 and anxious mothers use more intrusive parenting101 than do mothers without a psychiatric disorder. Mothers admitted to psychiatric mother and baby units (particularly those with either schizophrenia...
and personality disorder) often display practical difficulties in baby care, poor emotional responsiveness, and intrusive behaviour with their infants.87,104,105 

Parental difficulties in focusing their attention to infant signals and poor contingent responsiveness have been negatively associated with development of children’s attention and cognitive functioning in studies with mothers with depression.101 An infant's ability to control attention and cognitive functioning in turn, predictive of intellectual abilities.106,107 An important task for a parent is to support their infant when distressed, to maintain or recover their emotional equilibrium. Insufficient parental warmth, difficulties in regulation of an infant distress (so-called emotional scaffolding), and intrusiveness during stressful situations are negatively associated with child emotional regulation and behaviour.101 

Two aspects of parenting are associated with a child’s attachment security. First, disruptions to parental availability and appropriate responsiveness to attachment cues108 and second, the parent’s capacity to treat their child as a psychological agent with thoughts, feelings, and intentions.109 Disorder specific behaviours, such as speech and affect, manifested by a parent can be modelled by or transmitted to the child, leading to outcomes in the child that resemble the parent’s disorder. Infants of mothers with depression are more likely to show sad affect and vocalise less, although these behaviours are probably in part due to infant distress as a result of the parent's insufficient appropriate responsiveness.108,109 Infants of socially anxious mothers display similar responses to their mother, such as fear and avoidance of strangers.110 

Observed parental sensitivity mediates the association between both antenatal and postnatal depressive disorders and attachment security.106,107 and the association between postnatal depressive disorder and child emotion regulation during infancy10 and depression at age 16 years.10 Furthermore, parental responsiveness, cognitive support, and book reading to the child mediates the association between symptoms of postnatal depression and poor cognitive development.11,12 Experimental evidence suggests that activation of negative cognitions, both in the context of postnatal anxiety and depressive disorders, diminishes the quality of parenting, which is associated with negative infant behaviour.102 

Consistent with a role of modelling of disorder specific behaviours, mother and offspring depressogenic cognitions are correlated and partly mediate the association between perinatal maternal depression and the child’s depression at age 18 years.101 

Assumptions should not be made that the direction of effects is only from parent to child (figure). For example, some children are more difficult to raise because they frequently cry, sleep poorly, and are emotionally reactive. High irritability in young infants is strongly associated with the onset of maternal depression by 8 weeks’ postpartum.100 These infant characteristics might evoke mood changes in carers, potentially setting a cycle of bidirectional effects between mother and child.

Moderating factors

Moderation can occur at two levels: a variable can moderate the primary association—eg, sex of the child can directly modify associations between postnatal depression and child behaviour—or can occur at the level of the mediator. For example, the association between perinatal disorders and parenting (the mediator) is often moderated by socioeconomic circumstances. Important potential moderators of associations between perinatal disorders and child outcomes relate to the mother’s practical and financial support and socioeconomic status.105 Children whose mothers have the same extent of postnatal depression, but who are from a higher socioeconomic status, are less likely to be adversely affected. For example, in a large longitudinal study,19 maternal education moderated the association between symptoms of postnatal depression and offspring depression at age 18 years. Only children of mothers with a low level of education showed an increased risk of depression themselves.21 In a large community study,18 socioeconomic status moderated the association between postnatal depression and language development. Quality of parenting was also measured in this study, showing that the moderating effect occurred at the level of parenting.21 Evidence shows that a high socioeconomic status is protective of parenting in postnatal schizophrenia.21 Social and emotional support, including partner support, reduces associations between postnatal depression and early cognitive development.22 

In LMIC settings, the quality of parenting might have a greater role in a child’s physical wellbeing, because the environment is harsher in LMICs than in HICs. Poverty,
overcrowding, and poor sanitation are common, and with suboptimum maternal care, these factors potentially increase the risk to physical health of children.

Many additional factors accentuate the risk of poor outcomes in children of parents with perinatal disorders: single parenthood, teenage parenthood (in HICs), and family disharmony. The severity and duration of the disorder and particularly persistence after the postnatal period has consistently been shown to be an important moderator. Results of a moderating effect of sex are inconsistent although girls have been suggested to be more susceptible to emotional outcomes and boys to poorer behavioural and cognitive outcomes. Children who have specific temperaments, those who show high levels of negative emotions, are more affected by the quality of maternal care and might also be more amenable to interventions to help them.

**Treatment and prevention**

Although promising findings are emerging for the treatment and prevention of mental disorders during the perinatal period, few have investigated the potential benefit of such interventions for the wellbeing of the children. The extent to which children benefit from such interventions is important. For example, although reliable evidence shows that maternal depression can be successfully prevented and treated, amelioration of depressive symptoms alone has not been shown to improve mother–child interactions. Thus, in addition to provision of treatment for depression, efforts need to directly target improving mother–child interaction to potentially improve child outcome, in view of evidence for the mediating role of parenting. That is, ongoing stressors, poor parenting, or persistent parental symptoms might contribute to difficulties for children even in the absence of the mother’s disorder.

The few studies examining associations between treatment of maternal perinatal disorders and infant outcomes have reported the following findings. Mothers with postnatal depression who participated in psychotherapy (either non-directive supportive counselling, cognitive behavioural therapy, or psychodynamic psychotherapy) were associated with fewer toddler behavioural problems when aged 18 months than children of mothers assigned to routine care. However, no effect on cognitive development or attachment was noted. Mothers’ reduced levels of depression after 12 weeks of antidepressant drugs were associated with improvements in the quality of mother–infant interaction and infant play. Interpersonal therapy for mothers with postnatal depression decreased their level of depression, but was not associated with significant improvements in parenting or child outcomes. Finally, pregnant women with both diabetes and depression who participated in cognitive behavioural therapy had 6-month-old infants with better child development in psychomotor and behavioural functioning than did infants of mothers assigned to supportive counselling. Furthermore, improvements in depression symptoms in mothers treated with cognitive behavioural therapy correlated with infants’ developmental outcomes. Additional studies have also reported that antidepressant treatment in pregnancy might be associated with better infant developmental outcomes. Overall, this understudied question of associations has yielded a mixed pattern of results suggesting additional investigations are needed.

**Parenting interventions**

Interventions designed to provide parent education and improve parent–infant interactions for women with perinatal disorders have had some promising findings. Most interventions focused on postnatal depression, with no reported studies on interventions delivered prophylactically in pregnancy. Home-visiting programmes improve the quality of maternal–infant interactions in women with depression. Additionally, psychotherapeutic approaches for mothers who have depression, including a mother–infant psychotherapy group and interpersonal therapy, increases mother interactions with their infants compared with interactions of mothers with their infants in a wait-list control group. A meta-analysis concluded that the most effective parenting interventions for mothers with depression included infant massage, support groups, or interventions with more than one component.

Additional approaches have targeted mother–child interactions, often by use of individualised video-feedback which focused on improving the mothers’ sensitivity to infant cues. In women with depression, video-feedback interventions have been associated with improvements in quality of interactions, infants’ attachment security, and social competence compared with women who received little telephone parenting support. Video-feedback treatment provided to mothers with eating disorders led to improvements in mother–infant interactions and increased infant autonomy by comparison with women who received supportive counselling. An intensive (about once a week for 1 year) mother–toddler intervention for mothers of 20-month-old toddlers, who had had at least one major depressive episode since their child’s birth, was associated with improved cognitive development and secure attachment when compared with mothers who had not had depression since birth. In women with schizophrenia, approaches focusing on increasing maternal sensitivity are effective in improving mother–infant interactions. An assessment of video-feedback for mothers with acute postpartum psychiatric illness and their infants admitted to a mother and baby unit, reported improvements in mother–child interactions compared with mothers with psychiatric diagnoses living in the community who had not received inpatient care or video-feedback.
Several randomised controlled trials in LMICs showed that psychological interventions delivered by local community health workers can have a positive effect on parenting and aspects of child development. A study in Pakistan used cognitive-behavioural therapy with parenting support that began during pregnancy. Rates of postnatal depression were reduced, but no effect was noted on child growth. Parents reported a reduction in rates of children’s diarrhoea, increased rates of immunisation, and increased play with children, although the quality of parent–child interaction was not measured. In a socioeconomically disadvantaged South African community, although not exclusively the context of maternal depression, an intervention focused on helping mothers to attend to the details of the infant’s communication and to respond sensitively. This intervention led to improvements in the quality of mother–child interaction and increased rates of secure attachment. In Jamaica, an intervention targeting child rearing and parenting self-esteem led to improvements in both maternal depressive symptoms and infant global development compared with standard care. Determination of whether any improvements in mother and infant outcomes are sustained is an important question for future research.

Interventions involving fathers

Preliminary evidence shows that psychological interventions, such as cognitive-behavioural therapy, can be effective in reducing symptoms of depression in fathers during the postnatal period. However, no trials have assessed the effect of these interventions on parenting or child outcomes. Rather than focusing on treatment of maternal or paternal mental health in isolation, another approach is to move towards more inclusive perinatal mental health care, whereby the wellbeing of both parents are regarded and fathers are routinely involved. In view of the importance of the interparental relationship, correlations between maternal and paternal symptoms, and evidence that paternal involvement can buffer the effect of maternal disorder on the child, this inclusive approach could improve outcomes for the whole family. A preliminary assessment suggested that father inclusive information about perinatal mental health, provided by health-care systems, improved paternal response to the newborn baby.

Policy implications and conclusions

From a policy perspective, an essential first step is to identify both parents and children who are at an increased risk of adverse outcomes as a result of perinatal mental disorders to enable early treatment and prevention. Children also have needs that are not necessarily met by treating the parental disorder alone. Although much progress has been made to understand the mechanisms and pathways to both healthy and disturbed development of children, much remains to be done. Understanding of mechanisms, especially modifiable pathways, is key to development of interventions and identification of at-risk groups. For example, the quality of parenting is important and an understanding of the relationship between specific parenting behaviours and different child outcomes is crucial. Furthermore, if researchers can clarify the process that environmental factors, such as education and social support, mitigate the effects of perinatal disorders on the child, this knowledge could be used in prevention.

Development of evidence-based interventions and preventive strategies for these parents and children, including routine involvement of the father, is urgently needed. In view of increasing evidence that experiences in the early years of life are crucial for healthy development and productivity later in life and the number of people affected, interventions and prevention strategies should be a public health priority. Recognition is needed that addressing perinatal mental disorders can contribute substantially to the Millennium Development Goals and the post-2015 Millennium Development Goals agenda, specifically those related to child nutrition and early development, education, and maternal health (including antenatal care).

Although an association exists between children whose parents have mental disorders during the perinatal period and an increased risk of a range of adverse child outcomes, such negative outcomes are not inevitable. Most effect sizes for associations between disorders in parents and outcomes in children were moderate or small. Moreover, we identified evidence for moderation whereby factors, such as low socioeconomic status, absence of social support (including partner support), and persistence of the parental disorder, increased the child’s risk of adverse outcomes. Conversely, when disorders occur in the absence of social adversity and if they are of short duration, the risks to the child are generally low. Children in socioeconomically disadvantaged circumstances, especially in LMICs, are more likely to be both exposed to parental disorders and if their parent has a disorder to be affected than in children whose parents do not have a disorder, highlighting the need for global strategies that focus on integration of perinatal mental health and public health. Nonetheless, despite adversity many children in such situations develop normally and remain healthy, showing resilience of parental care and child development.

Contributors

AS and LMH developed the outline of this Review. RMP did the literature searches with ER and MMcC. RMP and MMcC created all tables. All authors contributed to the writing and editing of the manuscript. AS, RMP, and ER prepared the final version of the Series paper, which all authors approved.

Declaration of interests

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Wellcome Trust. ER was previously employed on a grant funded by the Wellcome Trust. LMH is chair of the NICE (update) guideline on Antenatal and Postnatal Mental Health. LMH is chief investigator of an National Institute of Health Research (NIHR) Programme Grant for Applied Research on the effectiveness of perinatal mental health services (grant number RP-RP-DG-108-10012) which also supports CMP, and has received funding from an NIHR Research Professorship (NIHR-RP-R3-12-003) on maternal mental health, and a grant from Tommy’s baby charity (with the support of a corporate social responsibility grant from Johnson and Johnson) on antipsychotics in pregnancy. LMH is also supported by the NIHR Mental Health Biomedical Research Centre at the south London and Maudsley NHS Foundation Trust and King’s College London. The views expressed are those of the authors and not necessarily those of the NHS, the NIHR, or the Department of Health. CMP has received research funding and consultancy fees from Eli Lilly, Servier, and Janssen, which are pharmaceutical companies involved in the development of antidepressants in the past 5 years. CMP is supported by the Medical Research Council and the European Commission; the NIHR and the NIHR Biomedical Research Centre for Mental Health at the south London and Maudsley NHS Foundation Trust and King’s College London; the Wellcome Trust, the NARSAD, and the Psychiatry Research Trust; and Eli Lilly and Janssen. We declare no other competing interests.

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**References**


