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Raised by Depressed Parents: Is it an Environmental Risk?

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Abstract

The mechanisms explaining how parental depression compromises healthy child development are complex and multifaceted, with genetic and environmental pathways intertwined. Reexamination of whether and how maternal and paternal depression serve as *environmental* risk factors is important because such an investigation can be helpful to identify modifiable mechanisms that are accessible to interventions. We review studies that have employed designs that isolate the effects of the environment from genetic influences, including adoption studies and children of twins studies. Findings indicate that maternal depression is an environmental risk factor for the emotional, behavioral, and neurobiological development of children. Although more studies are needed, preliminary findings suggest that paternal depression appears to be a weaker environmental risk as compared to maternal depression, at least during infancy and toddlerhood. Implications for theory and future research are discussed.

Keywords

Parental Depression; Environment; Child Development; Adoption; Children of Twins

Much has been written about children of depressed parents. There is no question that parental depression, particularly maternal depression, is a potent risk factor that compromises optimal child development. In a recent meta-analysis of family studies, Goodman et al. (2011) reported a small yet meaningful effect linking maternal depression and child functioning, including child internalizing problems (weighted mean r = .23),

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externalizing problems (weighted mean $r = .21$), and negative (weighted mean $r = .15$) and positive (weighted mean $r = -.10$) affect and behaviors. The "infectious" nature of maternal depression is not just a Western phenomenon, where most of the research has been conducted; associations between maternal depression and negative child outcomes have been observed worldwide (Wachs et al., 2009).

The mechanisms underlying the association between parental depression and child outcomes are highly complex. The intergenerational transmission of parental depression involves environmental risk factors, heritable components of depression (e.g., McGuffin and Katz, 1989; Rice et al., 2002; Sullivan et al., 2000; Thapar et al., 2012), and most likely the interplay of the two. Particularly pertinent to this review, proving that parental depression has true *environmental* risk effects on offspring outcomes turns out to be quite challenging. One important consideration is that genetic transmission of depression may confound the environmental effects of parental depression. Given that each parent and child share 50% of their genes, including those that influence psychopathology, the associations between the parent and child could be explained by genetic factors as well as environmental factors. Therefore, it is possible that genetic transmission may account for the associations observed in family studies that do not allow for the parsing of genetic and environmental effects. Still, it is important to isolate environmental mechanisms operating in the familial aggregation of depression from inherited pathways because such investigation can help to identify the modifiable systems that may be accessible targets of psychosocial intervention programs for families of depressed parents.

In this report, we aim to (a) review existing findings on associations between parental depression and child outcomes in studies that can disaggregate the environmental aspects of these associations from genetic transmission of depression across generations; (b) identify promising research topics for future investigation; and (c) discuss implications based on the reviewed findings. In this review, we focus on how parental depression functions as an *environmental* risk factor. Guided by the tenets of developmental psychopathology in general, and the concept of multifinality in particular (Cicchetti and Rogosch, 1996), this review considers multiple types of child and adolescent behaviors as outcomes of parental depression. Additionally, our review includes a mixture of studies using clinical diagnosis and subclinical symptoms of depression, although certain aspects of depression may be qualitatively different from diagnosable depression (Beach and Amir, 2003). For broader theories and findings on parental depression in general, interested readers are referred to existing meta-analyses (Goodman et al., 2011) and reviews (e.g., Cummings and Davies, 1994; Downey and Coyne, 1990; Gelfand and Teti, 1990; Goodman et al., 2011).

Evidence From Designs That can Isolate Environmental Effects

Despite the fact that numerous theories and studies presume the effects of parental depression on child outcomes to be environmentally driven (e.g., Cicchetti et al., 1998; Cummings and Davies, 1994; Shaw et al., 2009), only a few studies have conducted a direct test of whether and how parental depression influences child development through environmental mechanisms above and beyond genetically-driven influences. The paucity of this line of research likely reflects the difficulty and special care required in designing a

study that is capable of directly testing environmental effects. Most of the research on parental depression is based on data from biological families (i.e., parent(s) and child are genetically related), but isolating the environmental aspects of the association between parent and child functioning in such data is complicated by the challenges of partitioning genetic from environmental influences. The heritability of depression (McGuffin and Katz, 1989; Rice et al., 2002; Sullivan et al., 2000) creates a situation in which a depressed parent may not only pass down the genotype associated with depression to his or her child but may also create a rearing environment for the child that is influenced by the parent's own heritable characteristics, including depression. In this way, the genes that the child inherits and the home environment in which the child is raised are correlated. This association, known as passive gene-environment correlation (Plomin et al., 1977), cannot be eliminated from biological family designs, and makes it challenging to infer "pure" environmental linkage between parental depression and child outcomes (Rutter et al., 2001).

There are specific study designs, however, that can tease apart these effects, and they have begun to delineate the contributions of environmental influences in families of depressed parents to child outcomes. These designs include (a) adoption studies of parents rearing children to whom they are genetically unrelated and (b) children of twins studies. The utility of these designs in detecting environmental effects have been discussed elsewhere (Rutter et al., 2001). Given that the number of studies utilizing these two genetically/environmentally-sensitive designs is on the rise, this is an opportune time for a review of recent advances in the field. Table 1 summarizes the major findings pertinent to this report, as reviewed in the two sections below.

Adoption Studies

The classic parent-offspring adoption study design relies on a naturally occurring phenomenon whereby children are raised by genetically unrelated parents. Based on the quasi-experimental nature of the classic adoption design, some researchers have referred to it as a "natural experiment" (Rutter, 2006; Rutter et al., 2001). The adoption design, especially when children are adopted at or near birth, provides a powerful tool to disentangle environmental effects of parental depression from genetic influences. Because a parent and a child in a nonrelative adoptive family do not share genes, associations between adoptive parent depression and child outcomes represent "environmental" effects that are not attributable to shared genetic influences (Rutter et al., 2006; Rutter et al., 2001). When birth parents are also assessed, genetic influences can also be estimated within the adoption design. With detailed assessments of both adoptive and biological parents, as well as of the adopted child, a more nuanced and complete understanding of the interplay between genes and environment is possible. Additionally, when information on the prenatal environment is available, it is also possible to disentangle the effects of prenatal and postnatal environments on child outcomes.

There are variants of the adoption design, including the *adoption-at/near-birth design* and the *adoption-at-conception design* (Harold et al., 2013). The former, as described above, refers to a study design in which a child is placed for adoption at birth or near birth. The latter takes advantage of the increase in artificial reproductive technologies, including *in*

vitro fertilization (IVF), in which rearing mothers and fathers may or may not be genetically related to the child. Because the focus of this review is on disentangling the environmental effects of parental depression from genetic influences, this report is limited to a review of adoption studies in which the child was placed into an adoptive family at birth (or before the age of 2 years) or was conceived via IVF. Additional studies that have focused on children adopted from foster care or from orphanages after birth (e.g. the English and Romanian Adoptees Study [Rutter et al., 2007]; the Bucharest Early Intervention Project [Zeanah et al., 2003], and LONGSCAN [Runyan et al., 1998]) are excluded from this review. Although these studies have made significant contributions to the literature on resilience and depression, children within these studies often experience high levels of environmental instability (i.e., multiple caregivers and families) or early deprivation prior to study entry. As a result, distinguishing the effects of parental depression from these other environmental stressors is challenging.

Adoption-at-birth studies

In an investigation from the Sibling Interaction and Behavior Study (SIBS), Tully, Iacono, and McGue (2008) report on the first parent-child adoption study to examine the environmental effects of maternal and paternal depression on psychiatric disorders in adolescent children. Their findings indicated that depression in adoptive mothers was significantly associated with an increased incidence of various psychopathologies in adopted adolescents, including depression, oppositional defiant disorder, and conduct disorder. A follow-up of the sample revealed that this association remained in emerging adulthood; that is, major depressive disorder in adoptive parents was associated with major depressive disorder in adopted children as adults (Marmostein et al., 2012).

A second *adoption-at-birth* study, the Colorado Adoption Project (CAP), reported a significant, albeit modest ($r = .13$, $p < .05$), association between neuroticism in adoptive mothers, a known correlate of depression (Eaves et al., 1989), and maternal report of depression in adopted children (Eley et al., 1998). In comparison, the correlation between maternal neuroticism and children's depression was .05 for birth mothers and adopted children, and .17 for birthmothers and their biologically-related children living together. When contrasted with the correlation in adoptive families (.13), the slightly inflated correlation in family studies based on samples of biologically-related family members living together (.17) suggests that the environmental effects of maternal depression reported in biologically-related families could be confounded with genetic influences. Still, environmentally-driven mechanisms underlying the intergenerational transmission of depression seem present and potent, as indicated by the magnitude (albeit relatively modest) of the correlation between maternal neuroticism and child depression in adoptive families.

Although the aforementioned adoption studies suggest that parental depression affects functioning in children via environmental routes, several questions remain unanswered. First, the findings are from older children (middle childhood to late adolescence). It is widely acknowledged that older children of depressed parents display adult-equivalent psychopathologies (Goodman et al., 2011). However, parental depression influences a wide array of social, emotional, and biological functioning in children, even in those too young to

develop and/or manifest clinically recognized psychopathologies (Cicchetti and Rogosch, 1996). Early emerging risks and abnormalities in children of depressed parents may serve as intermediate phenotypes, or endophenotypes, of depression that provide clues to how parental depression may be transmitted to the child.

Furthermore, most studies have not considered paternal depression, with a few exceptions from the SIBS project (Marmostein et al., 2012; Tully et al., 2008). This issue of "father absence" is pervasive not only in depression research, but also in developmental research in general, particularly in studies of early childhood (Phares, 1992; Phares and Compas, 1992). In addition, prenatal environment is considered distinct from postnatal environment, and is often neglected in studies examining the impact of parental depression on child outcomes, including adoption studies. It is important to consider prenatal exposure to maternal depression, however, because it is known to have adverse effects on the physiological and socioemotional functioning in neonates (Field et al., 2006; Lundy et al., 1999; Monk et al., 2012) and in children up to 8 and 9 years of age (Luoma et al., 2001). Openness in adoption is another potential confound in the adoption design. For example, when birth parent(s) and an adoptive family interact (e.g., sending photos, birthday cards and Christmas gifts, calling over the phone, and making visits), the separation (broadly speaking) of "nature" and "nurture" pieces in the adoption design is muddled.

The third *adoption-at-birth* study, The Early Growth and Development Study (EGDS) has attempted to address these limitations. The EGDS is an ongoing prospective adoption-at-birth study, following children, birth families, and adoptive families since 4 months of age (Leve et al., 2013). A series of findings from the EGDS has confirmed and extended those from the aforementioned adoption studies with older children; that is, maternal depression is a potent environmental risk factor in a wide array of child outcomes during early childhood. Findings from the EGDS to date have shown that the effects of adoptive mothers' depressive symptoms were evident beginning as early as 9 months of age and continuing through toddlerhood (see details below) in the development of affect regulation (Leve et al., 2010), emotional/behavioral adjustment (Kerr et al., 2013; Laurent et al., 2013a; Natsuaki et al., 2010; Pemberton et al., 2010), and cortisol regulation (Laurent et al., 2013b). It is noteworthy that these results were obtained using a variety of methods to assess child outcomes, including observation, adoptive parents' reports of child behavior, and neurobiological measures (e.g., cortisol via saliva samples).

Affective regulation is one of the domains in child development that is affected by parental depression via environmental pathways during early childhood. In the Leve et al. (2010) study, 9-month old infants were seated in a high chair and presented with a stimulating toy. The toy, however, was presented behind a clear Plexiglas barrier that the infant could see but not reach, generating frustration. Infants whose adoptive mothers had higher levels of affective dysregulation (including depressive symptoms) persisted in this frustrating task and displayed difficulties in disengaging from a source of frustration in the barricade task. Further, this pattern was accentuated among infants whose biological mothers showed signs of heightened externalizing problems, suggesting a gene-environment interaction (GxE). Failure to shift attention away from a frustrating situation reflects an inability to suppress dominant, maladaptive behavior (Crockenberg et al., 2008). Such difficulty in diverting

attention away from an uncontrollable event might share similar features with ruminative coping, a known correlate of depression among older individuals, whereby persistence and dwelling on negative events and emotions result in a depressed mood (Nolen-Hoeksema et al., 1994). However, as children were only 9-months old when this study was undertaken, it remains to be seen whether there would be continuity between early difficulties in managing attention during an uncontrollable event and later internalizing symptoms or correlates of depression (e.g., rumination).

Additionally, adoptive mothers' depressive symptoms appear to influence early emerging emotional and behavioral outcomes in infancy and toddlerhood. An early emerging behavior that is known to correlate with later internalizing and externalizing problems is toddlers' distressed, fussy, affectively charged behavior. Natsuaki and colleagues (2010) documented that adoptive mothers' depressive symptoms (self-reported) at 9 months of age were associated with increased child fussiness at 18 months of age (rated by adoptive fathers) after controlling for preexisting fussiness and birth mothers' major depressive disorder (Natsuaki et al., 2010). Kerr et al. (2013) found that adoptive mothers' depressive symptoms were concurrently associated with the externalizing and internalizing symptoms of their children at age 18 months, a relationship that had previously been established among biological parents and their offspring but awaited corroboration from an adoption study design (Olino et al., 2008). This effect was found above and beyond birth mothers' major depressive disorder and antisocial behavior. Similarly, Pemberton et al. (2010) observed that chronicity of adoptive mothers' depression in infancy was predictive of externalizing problems at 27 months after accounting for birth mothers' depressive symptoms during pregnancy and after the birth of the child. Taking full advantage of multiple assessments of maternal depression over time, Laurent et al. (2013a) found that *changes* in maternal depressive symptoms are an equally important environmental liability, with changes in depressive symptoms of adoptive mothers associated with changes in levels of children's internalizing symptoms between ages 18 to 54 months. Taken together, these findings suggest that the mean level, chronicity, and changes in the depressive symptoms of adoptive mothers are all important predictors of emotional and behavioral outcomes in young children. However, it is also noteworthy that adoptive mothers' depressive symptoms at 9 months were not predictive of child anger at 18 months after controlling for other environmental risk factors in adoptive homes, including marital hostility and harsh parenting in adoptive parents (Rhoades et al., 2011).

Further, exposure to parental depressive symptoms appears to alter young children's vulnerability to stress, a putative mechanism underlying the development of psychopathology. Laurent and colleagues (2013b) reported that birth mothers' elevated depressive symptoms during pregnancy and postnatal exposure to adoptive mothers' depressive symptoms contributed to reduced cortisol levels in the child (Laurent et al., 2013b). Cortisol dysregulation, including lower cortisol, has been linked to elevated depression (Ehlert et al., 2001; Guerry and Hastings, 2011).

As noted earlier, there is little research on how depressive symptoms in fathers impact child outcomes. To date, EGDS studies that have examined paternal depression (e.g., Natsuaki et al., 2010; Rhodes et al., 2011) suggest that, in comparison to the depressive symptoms of

adoptive mothers, the depressive symptoms of adoptive fathers play a less consistent role as an environmental liability, at least in early childhood. There is only one study (Pemberton et al., 2010) that observed a direct effect of adoptive fathers' depression on child adjustment. This study showed the time-limited effect of paternal depressive symptoms, such that paternal depressive symptoms at 9 months were associated with children's externalizing problems at 27 months (Pemberton et al., 2010). This weak and inconsistent association between paternal depression and child outcomes in early childhood is aligned with the general picture depicted by previous studies of parental depression in early childhood using data from biologically-related family members (Connell and Goodman, 2002). Theoretically, paternal psychopathology increases its salience as children get older because fathers are often more involved in parenting older children (Connell and Goodman, 2002).

Although paternal depression alone might not be a reliable risk factor for child problem behavior relative to maternal depression in infancy and toddlerhood, it may serve as an amplifier of maternal depression. Specifically, in the Laurent et al. (2013b) study, evidence of E (maternal depression) x E (paternal depression) was found; the double dose of parental depressive symptoms from both adoptive fathers and adoptive mothers was related to lower cortisol levels in the child, which then was linked to higher levels of child internalizing problems. In addition, Pemberton et al. (2010) reported that adoptive fathers' depressive symptoms were associated with adoptive mothers' depressive symptoms during early childhood, suggesting the family-level clustering of parental depressive symptoms. These findings suggest the importance of considering paternal depressive symptoms within the broader level of the family context rather than only within individual parent-child dyads. In fact, emerging evidence from meta-analysis shows that paternal depression and maternal depression tend to co-occur in early years of child life – as early as prenatal and postpartum phases (Paulson and Bazemore, 2010).

Adoption-at-conception studies

This second type of adoption design includes children who were conceived via different types of IVF (Thapar et al., 2007). As a consequence, adoption-at-conception studies include children who vary in regard to their genetic relatedness to their parents. Specifically, children are genetically related to both parents (homologous IVF or surrogacy), to the mother only (sperm donation), to the father only (egg donation), or to neither parent (embryo donation). As such, when combined with a detailed assessment of the postnatal environment, this *adoption-at-conception design* clarifies whose genes the child inherited (mother, father, or third party), who provides what kind of prenatal environment (biological mother who raises the child or nonbiological mother who raises the child, or nonbiological surrogate mother who does not raise the child), and postnatal environments (the rearing mother and father). Such designs facilitate an examination of the genetic, prenatal, and postnatal environmental influences, extending what the *adoption-at-birth design* aims to do.

Using this design, Harold and colleagues (2011) showed that maternal and paternal depression were directly associated with child depressive symptoms during childhood (ages 4–10) in genetically-unrelated families. The environmental transmission of depressive symptoms independent of inherited effects was stronger for girls than for boys, and the

effects of life events, income, and SES shared among family members did not account for the observed environmental effects of parental depression (Lewis et al., 2011). This finding is consistent with those from the aforementioned adoption-at-birth studies, showing the importance of environmental factors in the transmission of depressive symptomatology across generations.

It is noteworthy that the IVF design is particularly powerful in disentangling two sources of environmental influences: prenatal and postnatal environments. This is an important issue to attend when researchers are interested in parental depression because maternal distress serves as prenatal and postnatal adversity to developmental outcomes in children through alterations in epigenetic and environmental pathways (Monk et al., 2012). The ability of the IVF studies to parse out these two environments stems from the design feature that allows variations in the contributions of rearing mothers to child development: genetic and postnatal environmental influences but no intrauterine environmental influences (surrogacy), intrauterine and postnatal environments but no genetic influences (egg and embryo donation), or intrauterine environment, postnatal environment, and genetic influences (homologous IVF and sperm donation). The special case of “neither genetic nor intrauterine environment” belongs to adoption-at-birth design, as discussed above.

Using this unique design feature of IVF, Rice et al. (2010) found that prenatal exposure to maternal distress was significantly associated with child anxiety and antisocial behavior in families whose mother and child were genetically unrelated (Rice et al., 2010). Interestingly, the association between prenatal exposure to maternal distress and child anxiety (but not child antisocial behavior) was mediated by maternal internalizing symptoms assessed when children were elementary school aged (ages 4–10), such that mothers who had higher levels of distress during pregnancy tended to show elevated depression and anxiety later, which contributed to children’s anxiety. Thus, two environmental mechanisms appear to be involved in the development of child anxiety: prenatal and postnatal exposure to maternal internalizing psychopathology.

Children of Twins Studies

Children of Twins studies (COT) are an extension of the twin study design. COT studies take advantage of the naturally occurring quasi-experiment that is the result of twins having children (D’Onforio et al., 2003). In the COT studies, the focus is the comparison of offspring of monozygotic (MZ) and dizygotic (DZ) twins who are differentially exposed (to parental depression, for example; Horwitz and Neiderhiser, 2011). Because the parents are twins, their children share approximately 25% of their genes on average in the case of children of MZ twin parents (like half siblings), and 12.5% of their genes in the case of children of DZ twin parents (like any cousins). This design is sensitive to environmental effects within the family because the offspring of twin parents experience different nuclear family-level factors such as parental depression (when twin parent pairs are discordant for depression), but share extended family-level factors, including genetic factors shared among cousins through twin parents.

Perhaps, the most intuitive approach to analyzing the COT data is to compare offspring outcomes of twin parents who are discordant in behaviors (D'Onforio et al., 2003; McAdams et al., in press). For example, when twin mother pairs are discordant for depression, offspring can be categorized into four groups: children of depressed (Group 1) vs. nondepressed MZ mothers (Group 2), and those of depressed (Group 3) vs. nondepressed DZ mothers (Group 4). If the rate of depression is similar across Groups 1 and 2, genetic influences are implicated; both children inherit genetic risk for depression from maternal side because the mothers of both children are genetically identical. In contrast, if the rate of depression is higher in Group 1 than Group 2 (and in Group 3 than Group 4), it supports the intergenerational transmission of depression via environmental pathways, specifically through environmental factors that are unique to the nuclear family. This is because children in Group 1 are exposed to the presence of a depressed mother at home (environmental exposure) while children in Group 2 are not. Further, if the rates of depression across four groups are similar, the role of environments shared by extended families is implicated because the common thread that makes cousins similar – regardless of differences in genetic relatedness or genetic risk toward depression – is environmental factors that are shared among extended family members.

Finding from the COT studies from the United States (Silberg et al., 2010), Australia (Singh et al., 2011), and Sweden (Class et al., 2012) have supported environmental mechanisms explaining the familial aggregation of depression. Applying this unique design to a Swedish sample, Class et al. (2012) recently reported that associations between paternal depressive symptoms and adolescent offspring-perceived self-competence (a known correlate of depression) most likely reflect nuclear family-level environmental factors. The detected effect of paternal depression as an environmental risk factor is consistent with the findings from the aforementioned adoption study with older children (Tully et al., 2008). Taken together, the findings from COT studies are consistent with those from adoption studies in two regards; that both types of study support an environmental mechanism for the intergenerational transmission of depression, and that paternal depression emerges as an environmental risk factor for older children.

Summary

Although many studies have identified significant associations between parental depression and child outcomes, fewer have explored the extent to which these links are explained by environmental mechanisms after accounting for genetic effects. This review focused on studies designed to separate environmental and genetic contributions to child adjustment, including adoption, IVF, and children of twins studies. These studies indicate that maternal depression is associated with children's outcomes through *environmental* pathways. In the studies reviewed, maternal depression was consistently associated with a wide array of child outcomes beginning in early childhood, including child psychiatric disorders (e.g., depression, anxiety, conduct disorders), adjustment problems (internalizing and externalizing problems), academic and peer problems, and early correlates of psychopathology (attention control, fussiness, vulnerability to stress) beginning as early as 9 months of age. Some evidence suggests that the environmental effects of maternal depression can persist into young adulthood. Most importantly, these associations were

present even when potential genetic and prenatal confounds were absent or controlled. Therefore, maternal depression is a non-specific environmental risk factor affecting a multitude of developmental outcomes in children starting in very early childhood. This observation is consistent with the idea of multifinality, whereby a single risk factor might lead to heterogeneous patterns of adaptation and/or maladaptation (Cicchetti and Rogosch, 1996). Interestingly, associations between paternal depression and child adjustment via environmental pathways during the first 5 years of life were less consistent relative to the effects of maternal depression, but they appear to be increasingly detectable as children become older. However, this conclusion needs to be taken with caution given the paucity of research on paternal depression in studies that is designed to highlight environmental effects.

Discussion: Limitations, Future Directions, and Implications

Based on the results reviewed above, at least three theoretically important lines of future research on parental depression are warranted. First and foremost, investigations into the mechanisms underlying the environmental associations between parental depression and child developmental outcomes are needed. Although this review detailed evidence supporting an environmental effect of parental depression, we do not yet fully understand *how* parental depression facilitates or impedes the intergenerational transmission of (mal)adaptation. Previous work has identified potential mediators, such as alterations in the child's biological systems (e.g., cortisol regulation, epigenetic processes), changes in the repertoire of behaviors and cognition through learning and modeling, and/or attachment insecurity (for reviews, see Cummings and Davies 1994; Downey and Coyne 1990; Gelfand and Teti 1990; Goodman and Gotlib 1999; Monk et al., 2012). Parental depression is also known to generate other contextual challenges, such as disruption and disengagement in parent-child relationships, disorganized home environments, interruptions in interparental relationships, and extra-family context adversity (e.g., stressful life events, employment, and social economic status: Cummings and Davies, 1994; Downey and Coyne, 1990; Gelfand and Teti, 1990; Goodman et al., 2011; Shaw and Shelleby, in press). Therefore, it is likely that chains of secondary environmental risks may partially explain the environmental effect of parental depression. With our increasing access to evidence from studies that are capable to parse out environmental effects of parental depression from genetic confounds, future research is well positioned to empirically and rigorously test these environmental hypotheses.

Additionally, there is still the open question as to children's differential susceptibility to the environmental aspects of parental depression. Individuals differ in their degree of sensitivity to environmental inputs (Belsky et al., 2007; Belsky et al., 2009; Boyce and Ellis, 2005). Thus, it is reasonable to assume that each child responds to the exposure to parental depression differently. One potentially fruitful avenue is to examine the possibility of gene-environment interaction (GxE). A recent perspective of human plasticity suggests that individuals differ in their degree of susceptibility to the environment – positive or negative environments — because of individual differences in genetic makeup (Belsky et al., 2009). An example that is consistent with the framework of GxE comes from the Leve et al. (2010) finding that infants of biological mothers with elevated externalizing problems had more difficulty in disengaging attention from a frustrating task when depressed/anxious adoptive

mothers raised them. Designs that are not only environmentally but also genetically informative, such as adoption and COT designs, provide an additional push to move beyond main effects of parental depression on child development.

Finally, the field still needs to further understand the impact of paternal depression on the lives of children. In particular, it is important to contextualize the effect of paternal depression within children's age and developmental milestones. In this review, we observed effects of paternal depression in older children (Class et al., 2012; Harold et al., 2011) but not in younger children (Leve et al., 2010; Natsuaki et al., 2010; Rhoades et al., 2011). The age-dependent association between paternal depression and child outcomes has been noted in previous meta-analysis (Connell and Goodman, 2002), but we lack the direct answer as to why this might be the case. Furthermore, depression in mothers and fathers is often treated independently in a parallel fashion, though in reality, depression of one parent may serve as a context of the depression of other parent, and mental health of both parents may be mutually interactive. A snapshot of this interactive process between fathers' and mothers' depression was observed in Laurent et al. (2013b). Results from this study found that even though paternal depression alone was not related to young children's functioning, its effect became significant when combined with maternal depression. Studies that take a family systems approach may be helpful in unpacking the role of paternal depression in the lives of children in various ages.

Implications

The confirmation that parental depression operates as an environmental risk factor for children's well-being should be treated as encouraging news, as it suggests the existence of modifiable risks in the familial transmission of depression. While we would refrain from making specific suggestions until we further delineate the detailed environmental mechanisms linking parental depression and child outcomes, it is warranted to conclude that successful treatment of parental depression would not only benefit the patients themselves, but also their children. Certainly, an accumulation of evidence suggests that treatment of depression is a family matter.

Additionally, our review reveals that parental depression exerts its adverse effects on child development not only when its severity reaches clinical levels (i.e., major depressive disorders), but also when it is at much more subtle, subclinical levels. This view is reflected in the tenets of developmental psychopathology; that is, atypical development is a deviation or distortion from normality, and the progression from normality to clinically diagnosable disorders takes multiple pathways and evolves over time (Cicchetti, 1993; Sroufe and Rutter, 1984). An important clinical implication of this notion is that screening methods and interventions that aim exclusively to serve severely depressed parents may be merely treating the tip of the iceberg. Parents with elevated depressive symptoms (but not clinically diagnosed as major depressive disorders) and their offspring may also benefit from intervention efforts.

Conclusion

Depression has a clear pattern of intergenerational transmission. Using studies conducted over the past 5 to 10 years with a variety of novel and genetically-informed research designs, this review provides evidence that parental depression (particularly maternal depression) is an *environmental* risk factor that influences the development of children as early as infancy and toddlerhood. Parental depression influences both typical and atypical child development, affecting a multitude of domains of functioning in children through environmental mechanisms. Future research is encouraged to further clarify *how* this environmental risk factor influences child development and the intergenerational transmission of depression. For this investigative endeavor, environmentally sensitive and genetically informative designs, such as longitudinal adoption and twin studies, will be powerful tools.

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Table 1

Studies That Isolate Environmental Effects of Parental Depression from Genetic Effects

Studies	E (parental depression)	Child outcomes	Age of child	Findings on maternal depression	Findings on paternal depression
		Adoption Studies			
Tully et al. (2008)	AP MDD	MDD, ODD, CD, ADHD, SUD	14 yrs	Main effect on MDD, ODD, and CD	Main effect on ADHD
Marmostein et al. (2012)	AP MDD	MDD, ND, AUD, CUD	19 yrs	Main effect on MDD	*ns*
Eley et al. (1998)	Neuroticism	Anxious/depressive symptoms	7–12 yrs	Main effect on maternal report of child symptoms	N/A
Leve et al. (2010)	AP Affective Dysregulation (depressive and anxiety symptoms)	Attention	9 mo	Main effect $G_{(BM\ externalizing\ problems)}$ x E	*ns*
Natsuaki et al. (2010)	AP Depressive symptoms	Fussiness	18 mo	Main effect	*ns*
Pemberton et al. (2010)	AP Depressive symptoms	EXT	27 mo	Main effect	Main effect
Laurent et al. (2013b)	AP Depressive symptoms	INT and EXT	18–54 mo	Main effect for INT E x cortisol for EXT	E x Cortisol for EXT E x Cortisol for INT
Laurent et al. (2013a)	AP Depressive symptoms	Cortisol	54 mo	$E_{(AM\ depression)}$ x $E_{(AF\ depression)}$	
Kerr et al. (2013)	AM Depressive symptoms	INT and EXT	18–54 mo	Main effect for INT and EXT at age 18 mo	N/A
Rhodes et al. (2011)	AP Depressive symptoms	Anger	18 mo	*ns*	*ns*
Harold et al. (2011)	Depressive symptoms for parents genetically unrelated to the child	Depressive symptoms	6 yrs	Main effect (not accounted for by parenting)	*ns*
Lewis et al. (2011)	Depressive symptoms for parents genetically unrelated to the child	Depressive symptoms	6 yrs	Main effect (not accounted for by family-shared adversity	*ns*
Rice et al. (2010)	Postnatal exposure to maternal depressive/anxiety symptoms for parents genetically unrelated to the child	Anxiety symptoms	4–10	Main effect (also mediating the link between prenatal stress and child anxiety)	*ns*
		Children of Twins Studies			
Silberg et al. (2010)	Depressive symptoms	Depressive symptoms	14 yrs	Support for environmental transmission of depression (no distinction between maternal and paternal depression)	
Singh et al. (2011)	MDD	MDD	25 yrs	Support for environmental transmission of depression (no distinction between maternal and paternal depression)	
Class et al. (2012)	Depressive symptoms	Self-competence	16 yrs	Shared genetic explained the mother-offspring associations	The father-offspring associations independent of genetic or extended family-level factors

Note. AP = adoptive parent(s); AM = adoptive mother; AF = adoptive father; BP = biological parent(s); E = environment; G = genetic influences; MDD = major depressive disorder; ODD = oppositional defiance disorder; CD = conduct disorder; ADHD = attention deficit disorder; SUD = substance use disorder; ND = nicotine dependence; AUD = alcohol use disorder; CUD = cannabis use disorder; INT = internalizing symptoms; EXT = externalizing symptoms.