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The Functioning of Offspring of Depressed Parents: Current Status, Unresolved Issues, and Future Directions

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Abstract

Although the intergenerational transmission of risk for depression is well documented, the mechanisms and moderators involved in this transmission of risk from depressed parents to their offspring are not clear. In this review, we discuss the progress that has been made over the past two decades in studying offspring of depressed parents and describe the maladaptive characteristics of these offspring in a diverse range of domains, including clinical, cognitive, and biological functioning. Despite recent advances in this area, there are unresolved questions that warrant further investigation involving the nature of risk transmission from parent to offspring, the specificity of findings to depression, and the role of factors that often accompany depression. We discuss these issues and offer directions for future research that we believe will move the field forward in gaining a better understanding of the relation between parental depression and altered psychobiological functioning in their offspring.

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DEPRESSION

Major depressive disorder (MDD) is among the most prevalent and recurrent of all psychiatric disorders (Am. Psychiatr. Assoc. 2013); almost 20% of the US population, or more than 30 million adults, will experience a clinically significant episode of depression, with most having multiple episodes of MDD over their lifetime (Kessler & Wang 2008). Further, rates of MDD and depressive symptoms have been increasing in both children and adults, particularly during the COVID-19 pandemic (Gotlib et al. 2021, Racine et al. 2021) and more strikingly in females than in males (Cent. Dis. Control 2023). MDD has enormous personal, familial, and societal costs, including adverse effects on physical health, interpersonal relationships, educational attainment, and financial security (Kessler & Wang 2008, Luppino et al. 2010). It is not surprising, therefore, that the World Health Organization (2017) has projected that depression will be the single most burdensome and costly disease in the world in terms of disability-adjusted years in the twenty-first century.

Given the increasing prevalence and rising costs of depression, the consistency with which researchers have documented adverse effects of parental depression on the functioning of offspring is especially alarming; in particular, a large literature has confirmed the high rates of depression in offspring with a depressed parent. Specifically, children and adolescents of parents with MDD are two to four times more likely to develop depression than are their peers with no family history of the disorder (Apter-Levy et al. 2013, Goodman & Garber 2017). Further, first episodes of depression appear to be occurring at increasingly younger ages (Lebrun-Harris et al. 2022); indeed, early-onset depression has been found to be associated with pervasive dysfunction over the life course (Hill et al. 2004). Given that over 10% of children in the United States are now exposed to parental depression each year (Ertel et al. 2011), it is clear that these high rates of depression in offspring with a family history of the disorder are a significant public health concern.

Although studies documenting high levels of depressive symptoms and an elevated prevalence of diagnosed depression in offspring of depressed parents are important in highlighting a specific adverse consequence of parental MDD, over the past two decades researchers have been building

on scientific advances to identify other types of difficulties and forms of altered or maladaptive functioning that characterize offspring of depressed parents. Thus, in addition to high rates of depression, there is now evidence that offspring of depressed parents are characterized by negative biases in their processing of information, impaired cognitive development, and problematic interpersonal relationships and exhibit altered neural function, structure, and connectivity, dysregulated hypothalamic-pituitary-adrenal (HPA) axis functioning, high levels of inflammation, and accelerated biological aging.

Several review articles have now been published describing the effects of parental depression on offspring. Perhaps most notably, Goodman & Gotlib (1999) formulated a comprehensive, integrative model of intergenerational transmission of risk for MDD focused on genetic, contextual, regulatory, and social-cognitive-demographic factors as mechanisms and moderators of risk and resilience for depression as outcomes in offspring of depressed mothers.[1] Goodman & Gotlib drew several conclusions and caveats from their review, noting the lack of research in particular areas and the tenuous links between psychobiological difficulties in offspring of depressed mothers and the subsequent development of depression. Although their review was comprehensive, Goodman & Gotlib focused primarily on clinical symptoms and diagnoses as the outcomes of having a depressed mother; they conceptualized other maladaptive characteristics as mediators of the association between maternal depression and offspring depression. More recently, Gotlib et al. (2020) also focused on clinical symptoms and diagnoses in offspring of depressed parents and identified mechanisms that might underlie the intergenerational transmission of risk for depression, underscoring findings that maternal depression is associated with a range of other risk factors such as poverty, marital conflict, and other environmental stressors. To date, however, there has not been a detailed consideration of the range of functioning that appears to be affected in offspring with a family history of depression and how this altered functioning may lead to the development of depression and/or other forms of psychopathology in offspring.

The purpose of this article is to synthesize the current literature examining the intergenerational effects of parental depression, elucidating the diverse domains and ways in which offspring of depressed parents exhibit maladaptive functioning. We begin by presenting recent data concerning symptoms and psychiatric diagnoses in offspring of depressed parents. Next, we describe cognitive and social difficulties in offspring of depressed parents and discuss alterations in their biological functioning. We discuss the likelihood that difficulties or alterations in specific domains do not occur in isolation but instead almost certainly influence each other and interact, leading to higher rates of disorder. In this context, therefore, we describe findings involving mediation and/or moderation of the association of parental depression with disorder in offspring and note examples of interactions between different domains of functioning. We also discuss the importance of conducting research with non-White participants and describe findings concerning the effects of the COVID-19 pandemic on pregnancy and the relation between parental depression and offspring functioning. Finally, we raise issues that warrant further investigation and offer directions for future research that we think will help make significant progress in understanding

[1] Given that, as we note above, the prevalence of depression is twice as high in women as it is in men, combined with the fact that most studies of offspring of depressed parents have focused on depressed mothers, it is understandable that Goodman & Gotlib (1999) discussed clinical outcomes in offspring of depressed mothers. Klein et al. (2005), Lewinsohn et al. (2005), and Gutierrez-Galve et al. (2019) examined the effects of maternal versus paternal depression on offspring's functioning and found that both were associated with adverse effects in offspring and that there were inconsistent differences between the effects of maternal versus paternal depression. In this review we broadly discuss the effects of parental depression on offspring and try to note differences, when available, between maternal and paternal depression.

the nature of the relation between parental depression and altered psychobiological functioning in offspring.

MALADAPTIVE PSYCHOBIOLOGICAL CHARACTERISTICS OF OFFSPRING OF DEPRESSED PARENTS

Psychiatric Diagnosis

One of the most consistent findings in clinical psychology and psychiatry is that offspring of depressed parents are at increased risk for developing depression (Hammen 2018). The link between parental depression and increased rates of psychopathology has been reported in children as young as 6 years of age, with over 60% meeting the full criteria for a psychiatric disorder, particularly an affective disorder (Priel et al. 2019). Given the consistency of this finding and our space constraints, we do not go into details of specific studies here but will note that in an exceptional longitudinal study, Weissman and colleagues (1982) significantly advanced our knowledge of the extent of the impact of parental depression on offspring by following a sample of depressed parents, their offspring, and now the offspring's children for almost 40 years. In 1982, Weissman and her colleagues recruited 91 families with and without a depressed parent, assessed psychopathology in the 276 offspring of these parents, and documented elevated rates of depression in these offspring. In 2005, Weissman et al. assessed the grandchildren of the initial probands and found that, even at a mean age of only 12 years, almost 60% of the grandchildren in families in which two generations had experienced depression already had a psychiatric disorder (Weissman et al. 2005). And in 2021, Weissman et al. reported a 38-year follow-up of these families and found that offspring of depressed parents were at increased risk not only for developing MDD and other psychiatric disorders but also for death by suicide or overdose (Weissman et al. 2021). Similar findings were reported by van Dijk et al. (2021) in the Adolescent Brain Cognitive Development sample and by Josefsson et al. (2019) in a Swedish cohort study. Finally, in the largest study in this area, Gronemann et al. (2023) followed a sample of almost three million people (all Danish citizens born between 1960 and 2003 with known parental identity) from their fifteenth birthday until they were diagnosed with depression, censored, or reached December 31, 2018. They found that for both men and women, exposure to maternal or paternal depression was associated with a two-times higher risk for developing MDD. Further, onset of maternal MDD before age 70 (for female offspring) and before age 30 (for male offspring) was associated with higher risk for MDD.

Cognitive Functioning

Depressed persons have consistently been found to have poorer and/or less adaptive cognitive functioning than do neurotypically functioning people. Importantly, similar differences have also frequently been observed in their offspring. For example, in 3- to 5-year-old children, maternal depressive symptoms were associated with a bias toward interpreting puppets as being sad (Martin et al. 2015). Compared with their peers without a family history of disorder, late childhood– to early adolescent–aged children of depressed parents have been found to have a more negative interpretation bias (Dearing & Gotlib 2009, Sfärlea et al. 2019), to have more difficulty identifying emotional facial expressions (Joormann et al. 2010, Székely et al. 2014), and to attend selectively to negative facial expressions (Joormann et al. 2007, Montagner et al. 2016). Interestingly, better cognitive functioning has been found to be protective of depressive symptoms in high-risk children. Davidovich et al. (2016) found that 9- to 17-year-old children of depressed parents with better inhibitory control and cognitive flexibility had fewer symptoms of depression than did children with poorer cognitive performance. Thus, cognitive functioning may offer an opportunity for targeted interventions for children at familial risk for depression.

Social Relationships

Depression often involves dysfunction in social relationships, difficulties that have also been observed in offspring of depressed parents. For example, in a meta-analysis, Barnes & Theule (2019) found that rates of insecure infant attachment were higher in offspring of depressed mothers than in controls. Further, prenatal maternal depressive symptoms have been associated with more peer relationship problems and less prosocial behavior in 4-year-old children (Koutra et al. 2017). Henry et al. (2020) found that symptoms of anxiety and depression were highest in adolescents who experienced more peer relationship stress and whose mothers had more symptoms of depression. Kujawa et al. (2020) found that the quality of the mother–child relationship at age 3 mediated the association of maternal depression with both child peer stress and neural response to social rejection at age 12. Finally, experiences of peer victimization have been found to mediate the association between maternal depressive symptoms and suicidal ideation in adolescent girls (Tsypes & Gibb 2015). Thus, like depressed adults, offspring of depressed parents appear to consistently experience difficulties in their interpersonal relationships.

Biological Characteristics of Offspring of Depressed Parents

Over the last 20 years researchers have made significant advances in our understanding of biological anomalies in offspring of depressed parents, involving progress in studying fetal development, neuroimaging, stress biology, inflammation, and biological aging. Many of the observed biological alterations have been implicated in the development of psychopathology, including depression. Thus, findings of studies in this area may provide information about mechanisms that underlie the intergenerational transmission of risk for this disorder. Recent reviews have provided comprehensive summaries of the literature organized by various biological methods and measures focused on elucidating specific mechanisms underlying familial risk for the development of psychopathology (e.g., Burkhouse & Kujawa 2022). In this section we build on these advances to describe differences in biological functioning between offspring of depressed versus nondepressed parents.

Epigenetic alterations. Maternal depression occurring during the gestational period may alter the development of the fetus, setting offspring on a long-term course of vulnerability to the onset of depression. The Developmental Origins of Health and Disease hypothesis posits that early life experiences, including the prenatal environment, can have lifelong implications for mental and physical health (Gluckman et al. 2007). Researchers examining neonatal tissues (e.g., placenta, umbilical cord blood) have begun to elucidate the ways in which maternal depression during pregnancy affects the functioning of offspring. For example, Conradt et al. (2013) found that higher levels of methylation of the glucocorticoid receptor gene *NR3C1* in the placenta of mothers with depression predicted lower self-regulation scores in their newborn infants. Similarly, Zhang et al. (2018) found that maternal prenatal depressive symptoms were associated with increased infant negative affect, but only in infants with decreased *HSD11B2*, *NR3C1*, and *NR3C2* (HPA-axis regulatory genes) placental gene expression. Further, Liu et al. (2012) found that depressive symptoms during pregnancy were associated with higher methylation at the *MEG3* differentially methylated region, which has been implicated in altered fetal development (Heijmans et al. 2009) and internalizing symptoms in infancy (Fuemmeler et al. 2016). Collectively, these findings point to a likely cascade of epigenetic alterations related to maternal depression that contribute to risk for the development of depression in offspring, although additional research on the epigenome and replication of relevant findings are clearly needed to gain a more comprehensive understanding of the major pathways that are affected by prenatal depression.

Brain structure, function, and connectivity. Offspring of depressed parents have consistently been found to exhibit alterations in their brain structure, function, and connectivity, some of which emerge as early as infancy in the context of prenatal maternal depression. For example, in a sample of 1-month-old infants, maternal prenatal depressive symptoms were associated with reduced right frontal white matter microstructure, particularly in female infants (Dean et al. 2018). In newborns with a genomic profile indicating elevated risk for MDD, maternal prenatal depressive symptoms were positively associated with larger right hippocampal volume and shape, right amygdala shape, and thicker orbitofrontal and ventromedial prefrontal cortices (Qiu et al. 2017); these findings suggest that MDD-relevant genes moderate the effects of prenatal maternal depressive symptoms on fetal development of brain regions implicated in cognitive-emotional functioning. Borchers et al. (2021) found that prenatal maternal depressive symptoms were associated with poorer white matter integrity in infancy. Prenatal maternal depressive symptoms have also been found to predict reduced cortical thickness in child offspring (Sandman et al. 2015). Interestingly, reductions in cortical thickness have been found to mediate the link between prenatal maternal depression and children's externalizing problems (Sandman et al. 2015), which in turn have been demonstrated to increase risk for internalizing problems during childhood (Oh et al. 2020) and depression during adolescence (Nilsen et al. 2013).

Longitudinal findings suggest that the effects of perinatal maternal depression on neurodevelopment in offspring are long-lasting and that chronic exposure to maternal depression is particularly impactful. For example, in a large birth cohort study, Zou et al. (2019) found that consistently high levels of perinatal maternal depression were associated with reduced gray and white matter volume in offspring at age 10. Finally, using resting-state functional magnetic resonance imaging, researchers have documented altered functional connectivity in infants exposed to prenatal depression, including greater positive functional connectivity of the amygdala with the left insula, bilateral anterior cingulate, and ventromedial prefrontal cortices (Qiu et al. 2015), and greater negative functional connectivity of the amygdala with dorsal prefrontal cortices (Posner et al. 2016).

Thus, the effects of perinatal depression on structural and functional brain alterations in infancy, some of which may persist through childhood and adolescence, may contribute to the intergenerational transmission of risk for depression. This perspective is consistent with the Developmental Origins of Health and Disease hypothesis that early life is characterized by rapid neurobiological development and, thus, is a sensitive period of increased plasticity and openness to the effects of maternal depression (Gluckman et al. 2007). As Goodman & Gotlib (1999) posited over two decades ago, an altered prenatal environment related to maternal depression, which might include increased fetal exposure to inflammation and cortisol, may shape offspring neurodevelopment in ways that increase risk for the development of depression (Wang et al. 2022).

Several researchers have also examined relations between parental depression and neural responsivity of offspring to various tasks in the scanner. Given that disruptions in individuals' responses to rewarding experiences have been implicated in diminished positive affect in depression (Forbes & Dahl 2012), many of these efforts have focused on reward processing. As is the case with depressed adults (Fox & Lobo 2019), offspring of depressed parents show altered neural function when processing rewarding stimuli. Blunted reward-related neural responses have also been observed in children of depressed mothers at as young as 5 years of age (Wiggins et al. 2017); similarly, Kujawa et al. (2014) found reduced neural differentiation between the receipt of gains and losses in 9-year-old children of depressed mothers. Because adolescence is a period of both heightened risk for the onset of depression and heightened striatal responses to the receipt of rewards (Luking et al. 2016), much of the research examining the brain basis of reward processing with offspring of depressed parents has focused on adolescents and young adults. For example,

in an early study Gotlib et al. (2010) found that never-depressed daughters of depressed mothers exhibited diminished activation in the putamen and left insula and heightened activation in the right insula when anticipating monetary gains. Adolescents and young adult offspring of depressed parents have also been found to exhibit decreased activation in the orbitofrontal cortex (McCabe et al. 2012) and the nucleus accumbens (Monk et al. 2008) in response to reward. More recently, using electroencephalography, Freeman et al. (2022) found that a maternal history of depression was related to reduced neural responses to the receipt of social rewards in adolescent girls. Collectively, therefore, this literature indicates that offspring of depressed parents are characterized by a blunted neural response in reward contexts, in both cortical and striatal regions.

Finally, researchers have posited that amygdala hyperactivity in response to negative stimuli is a useful biomarker of risk for the development of depression (Mattson et al. 2016). Indeed, investigators have found higher amygdala response to negative facial expressions in 6- to 9-year-old children of mothers who had prenatal depression (van der Knaap et al. 2018), in 8- to 14-year-old children of depressed parents (Chai et al. 2015), and in adolescents of depressed parents (Monk et al. 2008). Joormann et al. (2012) found stronger amygdala activation during a sad mood induction task in 9- to 14-year-old daughters of remitted depressed than of never-depressed mothers. In a larger sample of adolescents with a depressed parent or grandparent, left amygdala response to negative facial expressions increased over a 2-year interval compared with those without a family history of depression (Swartz et al. 2015). Given that amygdala activation has been shown to predict response to antidepressant treatment (Williams et al. 2015), it may be an important target for assessment and intervention in offspring of depressed parents.

Stress biology. Findings of studies examining the functioning of the HPA axis and autonomic nervous system indicate that offspring of depressed parents are characterized by dysregulated stress biology. Infants (Feldman et al. 2009), children (Apter-Levi et al. 2016), and adolescents (Gotlib et al. 2015) of depressed parents have all been found to have elevated levels of basal cortisol as well as potentiated increases in cortisol in response to stressors (Klimes-Dougan et al. 2022). For example, compared with adolescent daughters of mothers without a history of depression, daughters at familial risk for depression have elevated diurnal cortisol and cortisol reactivity to stress (Foland-Ross et al. 2014, Gotlib et al. 2015). Further, offspring of depressed parents exhibit patterns of autonomic activity that have been posited to underlie decreased or ineffective self-regulation. For instance, infants of depressed mothers have been found to exhibit elevated heart rate and decreased vagal tone both at rest and during maternal interactions (Propper & Holochwost 2013). Similar findings have been reported in older offspring: Relative to adolescent offspring of nondepressed mothers, adolescent offspring of depressed mothers are characterized by elevated resting heart rate (Nelson et al. 2021) and increased autonomic reactivity to sad faces (Burkhouse et al. 2014). Thus, as early as infancy, offspring of depressed mothers show signs of increased arousal and hyperactivity in stress biology systems.

Inflammation. Perhaps not surprisingly given that inflammation has been implicated in the pathophysiology of depression (Toenders et al. 2022), researchers have documented altered levels of inflammation in offspring of depressed parents. For example, toddlers of depressed mothers have been found to have heightened inflammation, indexed by a composite measure of proinflammatory cytokines (Measelle & Ablow 2018). Similarly, Wolf et al. (2008) found that parental depressive symptoms (primarily in mothers) were linked with increases in their children's inflammatory markers (e.g., interleukin-4) over a 6-month period. These findings were extended both by O'Connor et al. (2020), who found that maternal depressive symptoms in childhood predicted elevated levels of C-reactive protein in their offspring almost a decade later, and by Plant et al. (2016), who demonstrated that prenatal maternal depression predicted elevated levels

of C-reactive protein in offspring 25 years later. It is noteworthy that maternal depression and depressive symptoms have also been linked in offspring to physical health problems that have a strong inflammatory component. For example, prenatal maternal depressive symptoms have been found to be associated with infant infections (Schuez-Havupalo et al. 2018); similarly, autoimmune disorders, such as arthritis, psoriasis, and type 1 diabetes, are more common in children of parents with depression than in children of parents with no history of psychopathology (Nevriana et al. 2022). Thus, exposure to parental depression appears to alter the development of the immune system in offspring in ways that contribute to the expression of a maladaptive proinflammatory phenotype that may increase risk for the development of depression.

Biological aging. Offspring of depressed parents have been found to show signs of accelerated biological aging, conceptualized as a more rapid decline in the integrity and function of biological systems than occurs solely with increasing chronological age. Biological aging has been studied using a variety of measures across multiple domains (Colich et al. 2020), although it appears that different measures capture different aging or developmental processes (Miller et al. 2020). At the cellular level, researchers have perhaps most frequently assessed telomeres—protein caps at the end of chromosomes that shorten with cell division (Blackburn 2000); shorter telomere length has been used as a cellular measure of more rapid biological aging. In this context, adolescent offspring of depressed mothers have been found to have shorter telomeres than do their peers with nondepressed mothers (Gotlib et al. 2015, Nelson et al. 2021). Shorter telomere length has also been reported in children of mothers with elevated levels of depressive symptoms (Nelson et al. 2018). Although it is tempting to posit on the basis of these findings that shorter telomere length is a risk factor for the development of depression, it is important to recognize that shorter telomere length might instead be a consequence of other factors involved in the intergenerational transmission of risk for depression. For example, dysregulated stress biology, including increased HPA-axis and sympathetic nervous system activation, are putative mechanisms of processes, such as inflammation and oxidative stress, that contribute to telomere shortening (Jiang et al. 2019). Indeed, changes in maternal depressive symptoms have been indirectly linked to shorter telomere length in toddlers via altered infant HPA-axis functioning (Nelson et al. 2018). Importantly, findings from longitudinal research suggest that symptoms of psychopathology in adolescents predict telomere shortening over time, but not the reverse (Humphreys et al. 2020, Wade et al. 2020). Thus, shorter telomere length in children and adolescents of depressed parents may reflect a consequence of other psychobiological processes involved in familial risk for depression. These diverse findings involving telomere length in the context of other biological processes highlight the complexity of the construct of biological aging and underscore the need for more research in this area and replication of relevant findings.

Mediators, Moderators, and Interacting Variables

Although offspring of depressed parents are clearly at elevated risk for the development of depression, the mechanisms by which this risk is transmitted intergenerationally are still unclear. We describe above alterations in the functioning of offspring of depressed parents in several diverse domains, any of which may represent the mechanisms underlying the relation between family history and the development of depression. It is almost certain, however, that there is not a single mechanism underlying the intergenerational transmission of risk for depression but rather a constellation of contributing and likely interacting factors. Most researchers have focused on a single domain of risk for depression, such as anomalous neural characteristics, negative cognitive biases, or altered HPA-axis functioning. While the results of these investigations are informative, it is almost certain that many of these characteristics co-occur, and it is becoming increasingly clear

that the strongest advances in the understanding of risk for depression will come from studies that integrate assessments of psychobiological characteristics of offspring of depressed parents. Below, we briefly consider how the variables described above may interact with each other and with other factors to contribute to the development of depression in offspring with a depressed parent.

Caregiving behaviors. Suboptimal caregiving behaviors may increase risk for depression in offspring of depressed parents. Children of depressed parents have been found to report receiving fewer positive parenting behaviors from their parents (Loechner et al. 2020). Mechanistically, depression may lead parents to be less sensitive to their children's needs and/or to engage in less warm parenting, perhaps attributable to the negative cognitive biases, social withdrawal and lack of warmth, or increased irritability that characterizes depressed individuals (Lovejoy et al. 2000), impairing their ability to be aware of and respond to their children's needs. For example, mothers with prenatal depression have been found to be less responsive to their infants' distress when experiencing a heel lance and to engage in less affectionate touch in response to infant crying (Mercuri et al. 2023). Similarly, Salo et al. (2020) found that parents with high depressive symptoms exhibited less cognitive, emotional, and affective empathy toward their 1- to 3-year-old children.

Importantly, researchers have consistently found that problematic caregiving behaviors and parent–child relationships mediate associations between parent symptoms and child functioning. For example, harsh parent–child interactions at 2–3 years of age were found to partially mediate the association between early postnatal maternal depressive symptoms and child executive functioning at 4 years of age (Gueron-Sela et al. 2018). Similarly, maternal parenting at 18 months of age mediated the relation between maternal depressive symptoms at 9 months postpartum and child externalizing symptoms at 2 years of age (Taraban et al. 2019). Notably, Kuckertz et al. (2018) found that although parenting behaviors mediated the association of maternal depression with internalizing symptoms at 9 years of age, the relation between maternal and child symptoms was bidirectional. Maternal depression in the first year of life has been found to be associated with decreased mother–child synchrony during play and child oxytocin levels at 6 and 10 years of age, both of which mediated the relation between maternal depression and subsequent child internalizing and externalizing symptoms (Priel et al. 2019). Psychogiou et al. (2020) found that poorer parent–child relationships reported by 16-year-old offspring of depressed parents mediated the associations of parental depressive symptoms with child academic performance and mental health.

Sex differences. Perhaps not surprisingly given the well-documented sex differences in the prevalence of depression, some (but not all) researchers have also found sex differences in the magnitude of the association between parent depression and offspring outcomes. For example, researchers have found that maternal prenatal symptoms of depression were related to smaller amygdala volume in newborn males, but not females (Lehtola et al. 2020), and to poorer white matter microstructure in 1-month-old females, but not males (Dean et al. 2018). Similarly, Letourneau et al. (2019) found that maternal postnatal depressive symptoms mediated the association between maternal early life stress and internalizing and externalizing symptoms for 2-year-old boys, but not girls, and Swales et al. (2018) found that higher levels of prenatal maternal depressive symptoms were related to greater child emotional reactivity in preschool-aged girls, but not boys.

The timing of exposure to depressive symptoms may also be an important consideration in understanding sex differences in vulnerability to depression due to familial risk. In this context, Nolvi et al. (2019) found that whereas maternal depression that occurred only during the prenatal period was associated with elevated fear in 6-month-old girls but not in boys, the pattern was reversed in infants whose mothers reported elevated symptoms during both the pre- and postnatal periods, with girls having lower levels of fear than did boys. In contrast, Braithwaite et al. (2020) found that the association between postnatal maternal depression and offspring's emotional problems was

stronger in 3.5-year-old boys (but not in girls) whose mothers also had prenatal depression. Additional research is needed to clarify the precise nature of the relation between parental depression and risk for psychopathology in male and female offspring.

Sleep. Sleep disturbances have long been associated with psychological disorders and, indeed, are diagnostic symptoms of depressive disorders. Maternal depression, both prenatally and postnatally, has been found to affect offspring's sleep behaviors and sleep quality. For example, in a large Finnish cohort, prenatal maternal symptoms of depression predicted more sleep problems and higher risk for developing a sleep disorder in 3.5-year-old offspring, an association that was mediated by concurrent maternal depressive symptoms (Toffol et al. 2019). Postnatal maternal depression symptoms have also been found to mediate the association of the goodness of fit between mother–child sleep at 8 months postpartum and mother–child attachment at 30 months postpartum (Newland et al. 2016). Finally, de Jong et al. (2016) found that maternal postnatal depressive symptoms were associated with increased variability in sleep onset time in 4.5-year-old offspring, although only for low- and not for high-socioeconomic-status dyads. Researchers have begun to focus explicitly on the temporal nature of the relation between sleep problems and depressive symptoms. While some research suggests that the association between these two constructs is bidirectional (Wang et al. 2020), other investigators have found disturbed sleep to precede and predict the development of depression (Lovato & Gradisar 2014), indicating that sleep difficulties may be a useful target for intervention in the study of children at familial risk for depression.

Dysregulated emotion. Another mechanism by which parental mood may contribute to maladaptive functioning in offspring is through dysregulated emotion and stress reactivity. We describe above how offspring of depressed parents have altered regulatory abilities; importantly, these difficulties have consistently been found to underlie behavioral differences associated with parental depression. For example, maternal prenatal depressive symptoms are related to lower levels of cortisol, which in turn are associated with more externalizing symptoms in 12-month-old children (Galbally et al. 2019). In a sample of adopted children, Laurent et al. (2013) found that child cortisol at 54 months of age moderated the association of parental depressive symptoms with child internalizing and externalizing symptoms. Maternal depression has been found to be associated with increased mother and child cortisol, which in turn predicted increased internalizing and externalizing symptoms in middle childhood (Ulmer-Yaniv et al. 2018). In 9- to 15-year-old participants, maternal depressive symptoms were associated with increased internalizing and externalizing symptoms among adolescents using less adaptive stress coping strategies (Vreeland et al. 2019); this association was not evident in children who used more adaptive coping strategies. Further, in 8- to 17-year-old children, emotion regulation difficulties and negative life events partially mediated the relation between parent depressive symptoms and child depressive symptoms (Loechner et al. 2020). Thus, alterations in stress reactivity and emotion regulation may serve as mediating paths of intergenerational risk but may also magnify the relation between parental depressive symptoms and difficulties in offspring.

Non-White Participants

The associations between maternal mood and child outcomes have generally been assessed in White participants, to the exclusion of Black and Hispanic people in particular; there are important reasons to attend to racial and ethnic differences in this area. For example, Liu et al. (2016) found that concerns about financial and relational stressors were associated with depressed mood among Black and Hispanic pregnant people, but not White pregnant people, indicating that depressive symptoms may emerge and manifest differently in different racial or ethnic groups. Indeed,

researchers have conducted studies in non-White populations to examine the generalizability of results found in White samples. Giurgescu et al. (2015) found that preterm birth was 16 times more likely and low birth weight 4 times more likely to occur in Black than in White women with depression. In a sample of low-income Black women, prenatal depression was associated with a more negative view of the child before, during, and after birth (Lee & Hans 2015). The relation between parental depression and offspring functioning has also been examined in countries other than the United States and in non-White participants. For example, newborns were more likely to be of lower birth weight in samples of Zambian and Nigerian women with prenatal depression (Sanchez et al. 2013, Wado et al. 2014). Similarly, Bangladeshi and Peruvian women with prenatal depression gave birth to infants of younger gestational age (Nasreen et al. 2013, Sanchez et al. 2013). Maternal postpartum depression in Pakistani women has been found to be associated with a greater likelihood of delayed emotional, language, and motor development (Ali et al. 2013). Finally, among indigenous people in Canada, having a parent or grandparent who was forced to attend an Indian Residential School was associated with increased risk of suicide ideation and attempts (McQuaid et al. 2017). In general, studying the intergenerational transmission of risk for depression in ethnic minorities, in people of color, and in non-Western samples is particularly important given the historical underfunding of and lack of access to health care in these communities and the potential for members of these communities to have experienced racism and discrimination (Hankerson et al. 2022). Indeed, to this end, the World Health Organization has designated depression as a target of the Mental Health Gap Action Programme, which aims to increase prevention and treatment initiatives in low- and middle-income countries (World Health Organ. 2019).

COVID-19

The global COVID-19 pandemic has led to increased rates of depression, anxiety, and other psychopathological disorders (Gotlib et al. 2021, Racine et al. 2021); researchers are just beginning to explore the effects of the pandemic on the intergenerational transmission of risk for psychopathology. The pandemic appears to have increased levels of depressive symptoms in pregnant women (King et al. 2020, Racine et al. 2021), which has implications for the exposure of the developing fetus to prenatal depression and its associated factors. Buthmann et al. (2022) found that pandemic-related stress was associated with both increased prenatal depressive symptoms and infant negative affect. Comparing women who became mothers before versus during the pandemic, Chang et al. (2023) found that the association between postpartum depression and maternal anxiety about the child was stronger during than before the pandemic; further, parental bonding mediated the relation between postpartum depression and infant negative affect across both samples. The COVID-19 pandemic may have also exacerbated associations between parent depression and child mental health. For example, caregivers who reported having experienced greater pandemic-related stress were found to have more symptoms of depression and infants with higher levels of negative affect (Buthmann et al. 2022). Parental depression during the pandemic, in turn, has been associated with feeling less prepared to educate children in the home during school shutdowns (Lee et al. 2021) and with poorer caregiving quality (Roos et al. 2021).

CONCLUSIONS AND DIRECTIONS FOR FUTURE RESEARCH

In this review we document that offspring of depressed parents are characterized not only by high rates of depression but also by alterations in diverse domains, including their processing of information; cognitive development; interpersonal relationships; neural function, structure, and connectivity; HPA-axis functioning; biological aging; inflammation; and sleep. The range of

functioning across which we now know that offspring of depressed parents differ from their peers without a family history of disorder attests to the progress we have made over the last two decades in elucidating the extent of the difficulties experienced and exhibited by these offspring. In particular, scientific advances during this period have permitted researchers to examine aspects of the functioning of offspring of depressed parents that were not previously possible or viable, allowing us to obtain a more complete characterization of these individuals. Nevertheless, there are important issues that are still unresolved. In this section we raise and discuss questions that we believe must be addressed if we are to continue to make significant progress in understanding the nature of the relation between parental depression and altered psychobiological functioning in their offspring.

Altered Characteristics and Clinical Outcomes in Offspring of Depressed Parents

One critical issue that has not yet been adequately examined concerns the nature of the relation between altered functional characteristics in offspring of depressed parents and the onset of depression or other forms of psychopathology in these individuals. Many studies in this area have assessed and reported altered psychobiological functioning in offspring with a family history of depression while they are not in a depressive episode. Thus, it seems reasonable to postulate that altered characteristics are not simply correlates of depression in the offspring but instead factors that might place offspring at risk for the development of disorder; however, it is still not clear whether and/or how these characteristics, individually or jointly, increase the likelihood that offspring will develop psychopathology. That is, although there are exceptions in which researchers have tested explicitly whether specific alterations predict subsequent depression in offspring of depressed parents (e.g., Foland-Ross et al. 2015), investigators have not demonstrated reliably that offspring who are characterized by various forms of altered psychobiological functioning are more likely to develop depression than are their at-risk peers who do not exhibit these anomalies. In this context, not all researchers have examined and documented alterations in only those offspring who have not yet experienced an episode of depression (e.g., Dearing & Gotlib 2009, Foland-Ross et al. 2015, Joormann et al. 2007), raising the possibility that reported alterations are a consequence of having been depressed; still fewer studies have tested whether, within the same sample of offspring of depressed parents, those with altered characteristics are more likely to develop depression than are those without alterations. Elucidating these temporal differences in functioning within samples of offspring of depressed parents will further illuminate the nature of the relation between maladaptive psychobiological functioning and depression in offspring of depressed parents.

Other important questions involve related aspects of the altered characteristics that have been documented in offspring of depressed parents. For example, are these characteristics transient, fading over time, or are they enduring aspects of these individuals' functioning? Are they time-locked in a meaningful way to the onset or offset of a depressive episode? Does the development of depression in offspring exacerbate these characteristics and worsen the prognosis of the disorder in depressed parents' offspring who have them? Are different altered characteristics (or their interactions) related reliably to different symptoms or symptom profiles in offspring of depressed parents? And is the presentation of depression in offspring with a family history different than the presentation of depression in individuals without such a history? That is, are specific symptoms of depression more prevalent in offspring of depressed parents than they are in same-age depressed individuals without a family history of the disorder? In general, despite the progress we have made thus far, we need much more information concerning the nature of the relation between altered functioning and subsequent disorder in offspring of depressed parents.

Issues of Specificity

In this review we describe alterations in a range of psychobiological and social domains in offspring of depressed parents; it is not clear, however, whether these difficulties and anomalies are specific to parental depression. Indeed, given the high comorbidity of depression with other psychiatric and physical disorders (ter Meulen et al. 2021), it is likely that offspring of parents with diagnoses and difficulties other than depression also exhibit at least some of these anomalous characteristics. Similarly, it is not clear that depression is the only (or even the most prevalent) psychiatric disorder diagnosed in offspring of depressed parents. Finally, frequent correlates of depression, such as poverty and environmental toxins, marital difficulties, and psychosocial stressors, both alone and in interaction with a parental diagnosis, are also likely to be important in understanding the intergenerational transmission of risk for psychopathology.

Specificity to parental depression. With respect to parental diagnosis, early studies of offspring at familial risk for psychopathology, most of which focused on children of schizophrenic parents, typically included control groups of children of parents with a psychiatric diagnosis other than schizophrenia. For example, in the Stony Brook High Risk project, Neale, Weintraub, and their colleagues compared the functioning of children of parents diagnosed with schizophrenia with that of offspring of parents diagnosed with unipolar or bipolar depression in addition to children with no family history of any psychiatric disorder. In a number of publications from this project, children of unipolar depressed parents obtained lower scores than did children of healthy parents on measures of attention and cognitive functioning (Winters et al. 1981) and on teacher (Weintraub et al. 1975) and peer (Weintraub et al. 1978) ratings of behavior; often, however, children of depressed parents were indistinguishable from children of parents with schizophrenia, raising questions early on about the specificity of these difficulties to offspring of depressed parents.

Other studies have included offspring of medically ill patients as controls in studies of offspring of depressed parents. For example, Hirsch et al. (1985) compared adolescent offspring of unipolar depressed parents with offspring of arthritic and healthy parents and found no difference between adolescents of depressed and of arthritic parents in the number of depressive symptoms they endorsed. Similarly, Hammen et al. (1987) compared children of unipolar depressed mothers with children of bipolar depressed, medically ill, and healthy mothers. There were few differences in symptoms among children in the three experimental groups, and most of those differences were attenuated after controlling for current psychosocial stressors. Gotlib & Lee (1989) compared the functioning of children of depressed psychiatric patients, nondepressed psychiatric patients, nondepressed medical patients, and healthy controls and also found few differences between children of depressed and nondepressed patients. Hirshfeld-Becker et al. (2012) assessed the functioning of offspring of parents diagnosed with depression and/or panic disorder, in addition to offspring of healthy controls, and found that both forms of parental psychopathology were associated with elevated rates of diagnosed psychopathology in the offspring. Rasic et al. (2014) conducted a meta-analysis examining diagnoses in offspring of parents with depression, bipolar disorder, schizophrenia, and anxiety disorders. They found that although all forms of parental psychopathology were associated with elevated rates of diagnosable disorder in offspring, there was little evidence of specificity of difficulties to parental depression. Finally, Kerr et al. (2018) reported that comorbidity of personality disorders with parental depression increased offspring's risk for psychopathology.

Specificity of depression as an outcome in offspring. It appears, therefore, that there is little specificity to parental depression as a cause of adverse outcomes in offspring; offspring of

depressed parents are often indistinguishable from offspring of nondepressed psychiatric and medical patients with respect to their clinical and psychosocial functioning. A related issue concerns the specificity of a diagnosis of depression in offspring as a consequence of depression in the parents. Most studies that have examined diagnostic consequences of parental depression for their offspring have focused on depression as the outcome. Importantly, studies that have included other diagnostic outcomes have reported that offspring of depressed parents have elevated rates not only of depression but also of anxiety disorders and substance dependence (Weissman et al. 2021), as well as higher rates of physical illness (Weissman et al. 2006). For example, Côté et al. (2018) found in a large sample of youth that individuals who were exposed to elevated symptoms of maternal depression during their first five years of life had higher rates of symptoms of major depression, generalized anxiety, and social phobia in adolescence. Similarly, Weissman et al. (2006, 2021) found that offspring of depressed parents had significantly higher rates of bipolar disorder, anxiety disorders, and substance use disorders than did offspring of nondepressed parents. Thus, it is clear from our review that not only are more offspring of depressed than of nondepressed parents diagnosed with a significant psychiatric disorder (including, but not limited to, depression) but also offspring of depressed parents exhibit alterations in a wide range of domains of function, extending well beyond a psychiatric diagnosis.

In the context of these findings concerning both the lack of specificity to parental depression of difficulties in offspring and the wide range of altered behavior and functioning in offspring of depressed parents, we should note that the National Institute of Mental Health has formulated Research Domain Criteria (RDoC), a research framework to serve as a foundation from which to investigate issues involving mental disorders (see Morris & Cuthbert 2022). The broad aim of RDoC is to understand the nature of mental health and illness not by psychiatric diagnosis but in terms of varying degrees of dysfunction in fundamental psychological and biological systems, similar in important ways to the alterations documented in offspring of depressed parents. Because behavioral and biological aspects of functioning change throughout childhood and adolescence and across the life span, RDoC entails an explicit focus on developmental research. In fact, one can begin to map the maladaptive functioning of these offspring (and, indeed, of their depressed parents) onto the domains and constructs of the RDoC framework, including anomalies in neural function, structure, and connectivity, inflammation, sleep, and behavioral and cognitive difficulties. RDoC provides guidelines on assessing these alterations at different levels of analysis (in RDoC terms, units of analysis). Investigators have begun to consider how to best integrate RDoC constructs across different units of analysis and use computational methods to identify psychobiological targets for treatment (Sanislow et al. 2019). We believe that this approach has considerable potential to elucidate the broad effects of parental depression on the functioning of their offspring.

Accompanying factors. The final aspect of the specificity of adverse consequences of parental depression to offspring concerns factors that often accompany depression, such as poverty and ethnic minority status (Smith & Mazure 2021), marital difficulties (Mead 2002), and psychosocial stressors (Gilman et al. 2013). These factors, both alone and in interaction with a parental diagnosis, almost certainly contribute to the intergenerational transmission of risk for psychopathology, leading to findings of adverse consequences for offspring that are not due directly to the parent's psychopathology. For example, poverty, marital distress, and psychosocial stress all have been found to adversely affect children's development (e.g., Knopp et al. 2017).

In this context, in one of the most comprehensive studies in this area conducted to date, Barker et al. (2012) analyzed data from the Avon Longitudinal Study of Parents and Children, a population-based study of the effects of factors influencing children's health and development.

They examined the effects of maternal depressive symptoms and of an index of cumulative risk, created by summing participants' scores on measures of socioeconomic status, living conditions, and family risk factors (e.g., being a single caretaker, experiencing partner cruelty, lack of affection and support), on children's psychiatric diagnoses. Importantly, Barker et al. (2012) found that maternal depression was associated with 10 of the 11 individual risk factors that they included in their study and, further, that both maternal depression and cumulative risk factors increased children's odds of experiencing internalizing and externalizing disorders. Thus, studies that do not assess and control for these factors are likely overestimating the impact of parents' depression on their offspring's functioning.

SUMMARY

In this article we review the current literature examining the intergenerational effects of parental depression, describing maladaptive characteristics of offspring of depressed parents in a diverse range of domains, including clinical, cognitive, and biological functioning. Researchers have drawn on scientific advances over the past two decades to present a broader and more comprehensive picture of the functioning of offspring of depressed parents. Nevertheless, there are unresolved questions that warrant further investigation. We discuss several of these issues and offer directions for future research that we believe will move the field forward in elucidating the nature of the relation between parental depression and altered psychobiological functioning in their offspring.

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