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Studying the Intergenerational Transmission of Risk for Depression: Current Status and Future Directions

Ian H. Gotlib[1], Sherryl H. Goodman[2], and Kathryn L. Humphreys[3]
[1]Department of Psychology, Stanford University; [2]Department of Psychology, Emory University; and [3]Department of Psychology and Human Development, Vanderbilt University

Abstract
Studying offspring of depressed mothers is a promising strategy for elucidating factors that contribute to depression onset, given that these offspring are 3 to 6 times more likely to develop depression than are their low-risk peers. In this article, we briefly describe representative findings from studies of younger and older offspring of depressed mothers and identify factors that have garnered the most consistent empirical support across development. We discuss what these studies can and cannot tell us about mechanisms that might underlie the intergenerational transmission of risk for depression regardless of the age of offspring being studied. Finally, in light of limitations of this literature, we offer recommendations for future research.

Keywords
depression, intergenerational transmission, risk, development

Major depressive disorder (MDD) is among the most prevalent and costly of all psychiatric disorders; nearly 17% of Americans will experience an episode of MDD in their lifetime (Kessler & Bromet, 2013). Depressive episodes are recurrent: In clinical samples, 75% of individuals with MDD will experience more than one episode (Mueller et al., 1999). Perhaps not surprisingly, MDD has the greatest disease burden worldwide in terms of years lost to disability (Prince et al., 2007). Given these alarming statistics, it is critical that we gain a better understanding of factors that contribute to the onset of this disorder.

Addressing this need, researchers have studied individuals who are at the highest risk for developing MDD: offspring of depressed parents. In fact, these offspring are 3 to 6 times more likely to develop depression than are their peers with no maternal MDD history (Lieb, Isensee, Höfler, Pfister, & Wittchen, 2002). Given that the prevalence of depression is twice as high in women as in men (Bromet et al., 2011), most investigators study offspring of depressed mothers and, in doing so, have identified a range of factors that may contribute to the intergenerational transmission of risk for depression.

Studies in this area have tended to examine either younger (i.e., infants and young children) or older (i.e., school-age children and adolescents) offspring. Certainly, some methods, such as self-report, can be reasonably used only with older offspring; moreover, it is easier to follow older than younger offspring into the period of highest risk for the first onset of a depressive episode (i.e., during adolescence and early adulthood). It is important to note, however, that offspring's first exposure to their mothers' depression may occur as early as gestation. Further, given the critical importance of the early environment, maternal depression early in life may set motions into effect that are foundational for subsequent development. Indeed, the alterations documented in high-risk school-age children and adolescents are likely to have had precursors earlier in development.

With the exception of a small number of longitudinal investigations, most studies of the intergenerational

Corresponding Author:
Ian H. Gotlib, Stanford University, Department of Psychology, Building 420, Jordan Hall, Stanford, CA 94305
E-mail: ian.gotlib@stanford.edu

transmission of risk for depression focus on offspring in a narrow age range. Despite this limitation, it is noteworthy that investigations of offspring of depressed mothers, conducted across development, have assessed conceptually similar risk factors and outcomes. The broad purpose of this review is to describe what we have learned from studies of the offspring of depressed mothers and advance suggestions for how we can best make progress in this area of study. We have three primary goals in this article. The first is to briefly describe representative findings from studies of older and of younger offspring of depressed mothers and to identify factors that have garnered the most consistent empirical support across development. The second goal is to explicate what these studies can and cannot tell us about mechanisms that might underlie the intergenerational transmission of risk for depression in younger and older offspring of depressed mothers. Our third goal is to offer recommendations for future research based on the limitations of this literature. We begin by describing findings from studies of older and then of younger offspring of depressed mothers.

School-Age Children and Adolescents

A large number of studies have demonstrated that school-age children and adolescents of depressed mothers not only have a higher prevalence of depression than do their peers with nondepressed mothers but also, and often in the absence of significant depressive symptoms, exhibit psychobiological anomalies similar to those that have been found to characterize depressed adults. Thus, compared with older offspring of mothers with no family history of depression, children and adolescents with a depressed parent have higher levels of irritability and fear (e.g., Rice, Eyre, Riglin, & Potter, 2017), more negative cognitive biases (e.g., Joormann, Talbot, & Gotlib, 2007), weaker reward responsivity (e.g., Forbes & Dahl, 2012), stronger stress reactivity and shorter telomeres (Gotlib et al., 2015), and anomalous brain function and structure (e.g., Chai et al., 2015). They also have more problematic interpersonal relationships (e.g., Hammen & Brennan, 2001) and experience more stressful environments (e.g., Hammen, 2006). Importantly, although not always in the context of high-risk samples, several of these characteristics have also been found to predict the onset of a depressive episode, including more problematic interpersonal relationships (e.g., Teo, Choi, & Valenstein, 2013), greater cortisol secretion (e.g., Colich, Kircanski, Foland-Ross, & Gotlib, 2015), greater cortical thinning (e.g., Foland-Ross, Gilbert, Joormann, & Gotlib, 2015), and blunted neural response to reward (e.g., Bress, Foti, Kotov, Klein, & Hajcak, 2013).

Infants and Young Children

Even with recent advances in assessment, diagnosable depression in infants and very young children is rare. Consequently, researchers typically examine the relations (a) between depression in mothers and caregiving behaviors and environments and (b) between maternal depression and aspects of young offspring's functioning that are known to be associated with the development of depression (Field, 2010; Lovejoy, Graczyk, O'Hare, & Neuman, 2000). These studies indicate that maternal depression is associated with lower quality parenting (i.e., less sensitive, responsive), problematic caregiving practices (e.g., less preventive health care), and other environmental stressors (e.g., lower family functioning) in these early years. Further, young offspring of depressed mothers, compared with low-risk comparison subjects, have been found to have more severe internalizing and externalizing symptoms, greater negative and lower positive affectivity, less social engagement, lower social competence, more dysregulated behavior, slower cognitive development, greater heart rate reactivity to external stimulation, greater hypothalamic-pituitary-adrenal axis dysregulation, greater frontal electroencephalogram asymmetry, alterations in neural circuitry, and higher rates of insecure or disorganized attachment (see Peltola et al., 2014; Posner et al., 2016; Stein et al., 2014). Paralleling findings in older children and adolescents, results of other studies have shown that these characteristics predict subsequent elevations in internalizing problems or psychiatric disorders (e.g., Dougherty, Klein, Durbin, Hayden, & Olino, 2010; Sayal, Heron, Maughan, Rowe, & Ramchandani, 2014), and in one prospective study, mothers' postnatal depression predicted children's onset of depression at age 16 years (Murray et al., 2011).

Offspring of Depressed Mothers: Summary

Taken together, these findings indicate that both older and younger offspring of depressed mothers have, as a group, anomalous psychobiological characteristics and differ from their low-risk peers in important aspects of their environments (e.g., caregiving quality, exposure to stressors). Given the markedly different ages of the offspring in these two literatures, it is not surprising that there are differences in investigators' focus on specific environmental contexts (e.g., the early caregiving relationship vs. peer and romantic relationships) and in how they measure specific offspring characteristics (e.g., observed behavior vs. self-report). Despite these differences, however, there are striking parallels in their findings across development. Perhaps most consistently, both younger and older offspring of depressed mothers

exhibit difficulties in interpersonal relationships, abnormalities in stress reactivity, and attenuated reward- or approach-related functioning. Further, at least in older children and adolescents, there is evidence that these characteristics also predict the onset of depression. It is also not surprising to note that these difficulties in the functioning of high-risk offspring have all been found to characterize currently depressed individuals. Indeed, blunted reward responsivity, or anhedonia, is a cardinal diagnostic symptom of MDD. Thus, these characteristics in high-risk offspring prior to the onset of a depressive episode appear to be early markers of risk for MDD and may serve as risk factors for depression, mediating the association between maternal depression and disorder in the offspring.

What We Can and Cannot Learn From These Studies

Our second goal is to explicate what we can and cannot conclude from the findings of these studies of younger and older offspring concerning mechanisms that might underlie the intergenerational transmission of risk for depression. Although researchers initially attributed the high rates of depression in offspring of depressed mothers as evidence of inherited risk (e.g., mothers passing on their "genes for depression"), subsequent findings of associations between maternal depression and environmental risk factors have led to more complex causal formulations. Collectively, these findings, combined with results of recent studies examining additional domains of functioning (e.g., Gotlib, Joormann, & Foland-Ross, 2014), yield valuable information concerning how and when vulnerabilities or early signs of depression, such as heightened stress reactivity and blunted reward processing, emerge; both the nature and the timing of the vulnerabilities provide important information for the design and implementation of intervention efforts.

Despite these strengths, we do not yet understand how environmental exposures associated with having a depressed mother are related to genetic risk. In particular, we cannot map specific characteristics of maternal depression to risk for MDD in offspring nor do we understand precisely how specific vulnerabilities in offspring may increase risk for the subsequent onset of depression.

Directions for Future Research

We have learned much about the psychobiological characteristics of offspring of depressed mothers; nevertheless, we have noted essential questions and issues that must be addressed if we are to make progress in this field. In this final section of the article, we recommend research that we believe will increase the ability both to elucidate causal associations between maternal depression and child outcomes and to identify when and how to intervene. Specifically, we call for studies that (a) are genetically informed, (b) take into consideration the significant relations among risk factors associated with maternal depression, (c) are multidomain and longitudinal with respect to both mothers' and offspring's functioning, (d) focus on protective factors and resilience, and (e) are based on intervention science. Together, addressing these five areas has the potential to build an evidence-based understanding of precisely how maternal depression is associated with vulnerabilities that contribute to offspring's risk of developing depression.

First, with respect to genetically informed studies, there are several points to briefly make. Although the broad field of psychiatric genetics has not yet yielded the gains that some investigators had hoped it would, data from the Children of Twins design do indicate that risk for depression in offspring is mediated, at least in part, environmentally (Singh et al., 2011). Moreover, although heritability estimates help to determine the degree to which genes predict depression, there is a growing understanding that genetic risk factors can be moderated by the environment (Tucker-Drob, Briley, & Harden, 2013). In particular, the environment, including stress, may alter the influence of inherited risk differentially across the spectrum of experiences and, importantly, across development (Lamb et al., 2010). In fact, in this context, Kendler, Gardner, and Lichtenstein (2008) documented patterns of both genetic innovation (previously inactive genes become active) and genetic attenuation (previously influential genes weaken) with respect to symptoms of depression at different developmental stages. Thus, future research might profitably examine when in development, how, and under what conditions the environment can influence or alter the negative effects of maternal depression, perhaps through the use of polygenic risk scores (Whalley et al., 2013), thereby directly informing intervention protocols.

Second, researchers must recognize that maternal depression is associated with a range of other risk factors, such as poverty, marital conflict, and other environmental stressors. Although some studies have controlled statistically for potential confounds or tested moderation effects, the "confounds" are often treated as nuisance variables rather than as factors that may provide essential information. One approach to address potential confounds in observational data is to use propensity-score matching to equate groups on preidentified aspects while allowing the matched groups to differ on the variable of interest (West et al., 2014).

Using this approach, researchers would equate mothers with and without depression on selected measures of environmental characteristics associated with depression, essentially matching these two groups on those variables, and then compare outcomes to test the causal effect of the primary risk factor—in this case, depression in mothers. It is important to note, however, that the correlated factors may not be nuisance variables; indeed, they may play a causal role in the onset of MDD. Thus, models of intergenerational transmission of risk for depression must reflect these interrelated factors and effects.

Third, there is a need for multimodal longitudinal studies of risk for depression across development that conceptualize and assess age-related differences in the presentation of depression symptoms (i.e., heterotypic continuity; see Tyrell, Yates, Widaman, Reynolds, & Fabricius, 2019). These studies permit both the simultaneous modeling of multiple risk factors and the execution of cross-lagged analyses to elucidate temporal patterns of associations among risk factors and outcomes. In addition, given growing evidence of common elements of psychopathology across disorders (Caspi & Moffitt, 2018), combined with the formulation that maternal depression may be a nonspecific risk factor for multiple negative outcomes (Cicchetti & Rogosch, 1996), researchers would benefit from assessing not only MDD but also general psychopathology and adaptive functioning in both mothers and offspring. Identifying which children will develop depression and which children will develop other frequently co-occurring disorders (e.g., anxiety disorders, eating disorders, personality pathology) is important in elucidating mechanisms that underlie vulnerability to psychopathology and delineating pathways to specific disorders.

It is also important for researchers to acknowledge the role of the environment in shaping development, recognizing that children are not merely passive recipients of their environment and that experiences occurring earlier in life may have cascading effects. Indeed, risk factors may have a differential influence on children based on the plasticity of the developing brain. In this context, multimodal longitudinal studies and intervention designs may provide the best opportunity for researchers to test the timing of factors implicated in risk for depression as well as the dependence (vs. interdependence) of multiple risk factors and potential causal mechanisms. These studies can also yield tests of mediation to examine whether, how, and when certain markers or characteristics increase risk for depression.

Fourth, it is important to recognize the diversity within families in which offspring are at risk for depression. Such families are heterogeneous with respect not only to race, ethnicity, socioeconomic status, and other personal and demographic characteristics but also to the clinical features of depression. Indeed, we know little about the differential impact of various symptoms or subtypes of maternal depression on the offspring's functioning. Further, half of the offspring of depressed parents will not develop depression (Weissman et al., 2016). In this context, therefore, it is important that researchers examine specific aspects of this heterogeneity at different points in development and identify factors that promote resilience in offspring (e.g., Fischer, Camacho, Ho, Whitfield-Gabrieli, & Gotlib, 2018).

Finally, we encourage researchers to conduct studies using an intervention-science framework, not only to evaluate new treatment approaches but also to test mechanisms that affect risk for depression. For example, physiology in dyadic contexts can be examined using laboratory-based manipulations of stress (Thorson, West, & Mendes, 2018). Similarly, targeting caregivers' negatively biased cognitions (Bugental & Johnston, 2000) may normalize their expectations and improve interactions with their offspring, potentially reducing offspring's risk for depression. Finally, interventions designed to target risk factors associated with maternal depression (Garber, 2006) or to enhance high-risk children's coping and emotion-regulation skills (Compas et al., 2009) offer direct tests of specific risk factors implicated in the intergenerational transmission of risk for depression.

In closing, decades of research have taught us much about the intergenerational transmission of risk for depression; we have learned a great deal about outcomes in offspring of depressed mothers, including the ultimate outcome of MDD in these offspring. In addition, despite the fact that studies in this area have each targeted offspring in a narrow age range, researchers have identified similar constructs across developmental periods, which likely reflect continuity in vulnerabilities to depression. At this point, we recommend that researchers design studies that target those vulnerabilities for intervention, examine when in development it is most effective to do so, and determine how we might disrupt the intergenerational transmission of risk.

Recommended Reading

Goodman, S. H., & Gotlib, I. H. (1999). Risk for psychopathology in the children of depressed mothers: A developmental model for understanding mechanisms of transmission. *Psychological Review, 106*, 458–490. The first comprehensive and integrative review of mechanisms contributing to risk in offspring of depressed mothers.

Goodman, S. H., Rouse, M. H., Connell, A. M., Broth, M. R., Hall, C. M., & Heyward, D. (2011). Maternal depression and child psychopathology: A meta-analytic review. *Clinical Child and Family Psychology Review, 14*, 1–27.

A comprehensive review and meta-analysis documenting the magnitude of the effect size between maternal depression and children's psychopathology.

Gotlib, I. H., Joormann, J., & Foland-Ross, L. C. (2014). (See References). Provides the historical framework for understanding the study of offspring of depressed parents.

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ORCID iDs

Ian H. Gotlib https://orcid.org/0000-0002-3622-3199
Kathryn L. Humphreys https://orcid.org/0000-0002-5715-6597

References

Bress, J. N., Foti, D., Kotov, R., Klein, D. N., & Hajcak, G. (2013). Blunted neural response to rewards prospectively predicts depression in adolescent girls. *Psychophysiology*, *50*, 74–81. doi:10.1111/j.1469-8986.2012.01485.x

Bromet, E., Andrade, L. H., Hwang, I., Sampson, N. A., Alonso, J., de Girolamo, G., . . . Kessler, R. C. (2011). Cross-national epidemiology of DSM-IV major depressive episode. *BMC Medicine, 9*, Article 90. doi:10.1186/1741-7015-9-90

Bugental, D. B., & Johnston, C. (2000). Parental and child cognitions in the context of the family. *Annual Review of Psychology*, *51*, 315–344. doi:10.1146/annurev.psych.51.1.315

Caspi, A., & Moffitt, T. E. (2018). All for one and one for all: Mental disorders in one dimension. *The American Journal of Psychiatry*, *175*, 831–844. doi:10.1176/appi.ajp.2018.17121383

Chai, X. J., Hirshfeld-Becker, D., Biederman, J., Uchida, M., Doehrmann, O., Leonard, J. A., . . . Gabrieli, J. D. E. (2015). Functional and structural brain correlates of risk for major depression in children with familial depression. *NeuroImage: Clinical*, *8*, 398–407. doi:10.1016/j.nicl.2015.05.004

Cicchetti, D., & Rogosch, F. A. (1996). Equifinality and multifinality in developmental psychopathology. *Development and Psychopathology*, *8*, 597–600. doi:10.1017/S0954579400007318

Colich, N. L., Kircanski, K., Foland-Ross, L. C., & Gotlib, I. H. (2015). HPA-axis reactivity interacts with stage of pubertal development to predict the onset of depression. *Psychoneuroendocrinology*, *55*, 94–101. doi:10.1016/j.psyneuen.2015.02.004

Compas, B. E., Forehand, R., Keller, G., Champion, J. E., Rakow, A., Reeslund, K. L., . . . Cole, D. A. (2009). Randomized controlled trial of a family cognitive-behavioral preventive intervention for children of depressed parents. *Journal of Consulting and Clinical Psychology*, *77*, 1007–1020. doi:10.1037/a0016930

Dougherty, L. R., Klein, D. N., Durbin, C. E., Hayden, E. P., & Olino, T. M. (2010). Temperamental positive and negative emotionality and children's depressive symptoms: A longitudinal prospective study from age three to age ten. *Journal of Social and Clinical Psychology*, *29*, 462–488. doi:10.1521/jscp.2010.29.4.462

Field, T. (2010). Postpartum depression effects on early interactions, parenting, and safety practices: A review. *Infant Behavior and Development*, *33*, 1–6. doi:10.1016/j.infbeh.2009.10.005

Fischer, A. S., Camacho, M. C., Ho, T. C., Whitfield-Gabrieli, S., & Gotlib, I. H. (2018). Neural markers of resilience in adolescent females at familial risk for major depressive disorder. *JAMA Psychiatry*, *75*, 493–502. doi:10.1001/jamapsychiatry.2017.4516

Foland-Ross, L. C., Gilbert, B. L., Joormann, J., & Gotlib, I. H. (2015). Neural markers of familial risk for depression: An investigation of cortical thickness abnormalities in healthy adolescent daughters of mothers with recurrent depression. *Journal of Abnormal Psychology*, *124*, 476–485. doi:10.1037/abn0000050

Forbes, E. E., & Dahl, R. E. (2012). Research review: Altered reward function in adolescent depression: What, when and how? *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *53*, 3–15. doi:10.1111/j.1469-7610.2011.02477.x

Garber, J. (2006). Depression in children and adolescents: Linking risk research and prevention. *American Journal of Preventive Medicine*, *31*(6, Suppl. 1), 104–125. doi:10.1016/j.amepre.2006.07.007

Gotlib, I. H., Joormann, J., & Foland-Ross, L. C. (2014). Understanding familial risk for depression: A 25-year perspective. *Perspectives on Psychological Science*, *9*, 94–108. doi:10.1177/1745691613513469

Gotlib, I. H., Lemoult, J., Colich, N. L., Foland-Ross, L. C., Hallmayer, J., Joormann, J., . . . Wolkowitz, O. M. (2015). Telomere length and cortisol reactivity in children of depressed mothers. *Molecular Psychiatry*, *20*, 615–620. doi:10.1038/mp.2014.119

Hammen, C. (2006). Stress generation in depression: Reflections on origins, research, and future directions. *Journal of Clinical Psychology*, *62*, 1065–1082. doi:10.1002/jclp.20293

Hammen, C., & Brennan, P. A. (2001). Depressed adolescents of depressed and nondepressed mothers: Tests of an interpersonal impairment hypothesis. *Journal of Consulting and Clinical Psychology*, *69*, 284–294. doi:10.1037/0022-006X.69.2.284

Joormann, J., Talbot, L., & Gotlib, I. H. (2007). Biased processing of emotional information in girls at risk for depression. *Journal of Abnormal Psychology*, *116*, 135–143. doi:10.1037/0021-843X.116.1.135

Kendler, K. S., Gardner, C. O., & Lichtenstein, P. (2008). A developmental twin study of symptoms of anxiety and depression: Evidence for genetic innovation and attenuation. *Psychological Medicine*, *38*, 1567–1575. doi:10.1017/S003329170800384X

Kessler, R. C., & Bromet, E. J. (2013). The epidemiology of depression across cultures. *Annual Review of Public Health*, *34*, 119–138. doi:10.1146/annurev-publhealth-031912-114409

Lamb, D. J., Middeldorp, C. M., van Beijsterveldt, C. E. M., Bartels, M., van der Aa, N., Polderman, T. J. C., & Boomsma, D. I. (2010). Heritability of anxious-depressive and withdrawn behavior: Age-related changes during adolescence. *Journal of the American Academy of Child and Adolescent Psychiatry*, *49*, 248–255. doi:10.1097/00004583-201003000-00008

Lieb, R., Isensee, B., Höfler, M., Pfister, H., & Wittchen, H. U. (2002). Parental major depression and the risk of depression and other mental disorders in offspring: A prospective-longitudinal community study. *Archives of General Psychiatry*, *59*, 365–374. doi:10.1001/archpsyc.59.4.365

Lovejoy, M. C., Graczyk, P. A., O'Hare, E., & Neuman, G. (2000). Maternal depression and parenting behavior: A meta-analytic review. *Clinical Psychology Review*, *20*, 561–592. doi:10.1016/S0272-7358(98)00100-7

Mueller, T. I., Leon, A. C., Keller, M. B., Solomon, D. A., Endicott, J., Coryell, W., . . . Maser, J. D. (1999). Regular articles: Recurrence after recovery from major depressive disorder during 15 years of observational follow-up. *The American Journal of Psychiatry*, *156*, 1000–1006.

Murray, L., Arteche, A., Fearon, P., Halligan, S., Goodyer, I., & Cooper, P. (2011). Maternal postnatal depression and the development of depression in offspring up to 16 years of age. *Journal of the American Academy of Child and Adolescent Psychiatry*, *50*, 460–470. doi:10.1016/j.jaac.2011.02.001

Peltola, M. J., Bakermans-Kranenburg, M. J., Alink, L. R. A., Huffmeijer, R., Biro, S., & van IJzendoorn, M. H. (2014). Resting frontal EEG asymmetry in children: Meta-analyses of the effects of psychosocial risk factors and associations with internalizing and externalizing behavior. *Developmental Psychobiology*, *56*, 1377–1389. doi:10.1002/dev.21223

Posner, J., Cha, J., Roy, A. K., Peterson, B. S., Bansal, R., Gustafsson, H. C., . . . Monk, C. (2016). Alterations in amygdala–prefrontal circuits in infants exposed to prenatal maternal depression. *Translational Psychiatry*, *6*, Article e935. doi:10.1038/tp.2016.146

Prince, M., Patel, V., Saxena, S., Maj, M., Maselko, J., Phillips, M. R., & Rahman, A. (2007). No health without mental health. *The Lancet*, *370*, 859–877. doi:10.1016/S0140-6736(07)61238-0

Rice, F., Eyre, O., Riglin, L., & Potter, R. (2017). Adolescent depression and the treatment gap. *The Lancet: Psychiatry*, *4*, 86–87. doi:10.1016/S2215-0366(17)30004-4

Sayal, K., Heron, J., Maughan, B., Rowe, R., & Ramchandani, P. (2014). Infant temperament and childhood psychiatric disorder: Longitudinal study. *Child: Care, Health and Development*, *40*, 292–297. doi:10.1111/cch.12054

Singh, A. L., D'Onofrio, B. M., Slutske, W. S., Turkheimer, E., Emery, R. E., Harden, K. P., . . . Martin, N. G. (2011). Parental depression and offspring psychopathology: A Children of Twins study. *Psychological Medicine*, *31*, 1485–1495. doi:10.1017/S0033291710002059

Stein, A., Pearson, R. M., Goodman, S. H., Rapa, E., Rahman, A., McCallum, M., . . . Pariante, C. M. (2014). Effects of perinatal mental disorders on the fetus and child. *The Lancet*, *384*, 1800–1819. doi:10.1016/S0140-6736(14)61277-0

Teo, A. R., Choi, H. J., & Valenstein, M. (2013). Social relationships and depression: Ten-year follow-up from a nationally representative study. *PLOS ONE*, *8*(4), Article e62396 doi:10.1371/journal.pone.0062396

Thorson, K. R., West, T. V., & Mendes, W. B. (2018). Measuring physiological influence in dyads: A guide to designing, implementing, and analyzing dyadic physiological studies. *Psychological Methods*, *23*, 595–616. doi:10.1037/met0000166

Tucker-Drob, E. M., Briley, D. A., & Harden, K. P. (2013). Genetic and environmental influences on cognition across development and context. *Current Directions in Psychological Science*, *22*, 349–355. doi:10.1177/0963721413485087

Tyrell, F. A., Yates, T. M., Widaman, K. F., Reynolds, C. A., & Fabricius, W. V. (2019). Data harmonization: Establishing measurement invariance across different assessments of the same construct across adolescence. *Journal of Clinical Child & Adolescent Psychology*, *48*, 555–567.

Weissman, M. M., Wickramaratne, P., Gameroff, M. J., Warner, V., Pilowsky, D., Kohad, R. G., . . . Talati, A. (2016). Offspring of depressed parents: 30 years later. *The American Journal of Psychiatry*, *173*, 1024–1032. doi:10.1176/appi.ajp.2016.15101327

West, S. G., Cham, H., Thoemmes, F., Renneberg, B., Schulze, J., & Weiler, M. (2014). Propensity scores as a basis for equating groups: Basic principles and application in clinical treatment outcome research. *Journal of Consulting and Clinical Psychology*, *82*, 906–919. doi:10.1037/a0036387

Whalley, H. C., Sprooten, E., Hackett, S., Hall, L., Blackwood, D. H., Glahn, D. C., . . . McIntosh, A. M. (2013). Polygenic risk and white matter integrity in individuals at high risk of mood disorder. *Biological Psychiatry*, *74*, 280–286. doi:10.1016/j.biopsych.2013.01.027