

THE DYNAMIC INTERPLAY BETWEEN PARENT DEPRESSIVE SYMPTOMS
AND CHILD BEHAVIOR PROBLEMS ON CHILD PSYCHIATRIC DISORDERS:
AN ADOPTION STUDY

by

CAMILLE C CIOFFI

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Student: Camille C Cioffi

Title: The Dynamic Interplay Between Parent Depressive Symptoms and Child Behavior Problems on Child Psychiatric Disorders: An Adoption Study

This dissertation has been accepted and approved in partial fulfillment of the requirements for the Doctor of Philosophy degree in the Department of Counseling Psychology and Human Services by:

Leslie Leve	Chairperson and Advisor
John Seeley	Core Member
Elizabeth Stormshak	Core Member
Dave DeGarmo	Core Member
Sanjay Srivastava	Institutional Representative

and

Kate Mondloch	Interim Vice Provost and Dean of the Graduate School
---------------	--

Original approval signatures are on file with the University of Oregon Graduate School.

Degree awarded June 2020

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DISSERTATION ABSTRACT

Camille C Cioffi

Doctor of Philosophy

Department of Counseling Psychology and Human Services

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Title: The Dynamic Interplay Between Parent Depressive Symptoms and Child Behavior Problems on Child Psychiatric Disorders: An Adoption Study

The dynamic interplay between parents' depressive symptoms and child behavior problems over time is not well understood. This dissertation used data from a prospective parent-offspring adoption design to estimate paths for birth parents' psychopathology, birth mother depressive symptoms during pregnancy, and adoptive parents' depressive symptoms across early and middle childhood on child behavior problems over time, including the contributions of infant negative emotionality and how these jointly contributed to subsequent child psychiatric disorders. Additionally, the bidirectional associations between parent depressive symptoms and child behavior problems were examined. Overall, results provided some support for general genetic risk for psychopathology and no evidence for prenatal depressive symptoms as a risk for child behavior problems or child psychiatric disorder. Additionally, there was little evidence for cross-lagged associations overtime between parent depressive symptoms and child behavior problems but there was evidence for overall associations between the two constructs overtime. Negative emotionality was a clear predictor of both child behavior problems and parent depressive symptoms. Taken together, these results suggest that

there is a genetic risk posed to offspring by birth mother psychopathology and that child behavior problems, influenced by adoptive parent depressive symptoms, predicate child psychiatric disorders. Adoptive mother's and father's depressive symptoms each influence a child's susceptibility to child behavior problems. Early intervention for parent depressive symptoms is thus warranted.

CURRICULUM VITAE

NAME OF AUTHOR: Camille C Cioffi

GRADUATE AND UNDERGRADUATE SCHOOLS ATTENDED:

University of Oregon, Eugene

DEGREES AWARDED:

Doctor of Philosophy, Prevention Science, 2020, University of Oregon
Bachelor of Science, Psychology and Political Science, 2012, University of Oregon

AREAS OF SPECIAL INTEREST:

Quantitative Methods

GRANTS, AWARDS, AND HONORS:

2019	Pilot Award from the Center for Parenting and Opioids under P50 DA048756
2019	College of Education Dissertation Fellowship
2019	Prevention Science, Doctoral Travel Award
2019	Society for Prevention Research Early Career Travel Award
2018	Prevention Science, Doctoral Travel Award
2018	Society for Prevention Research Early Career Travel Award
2018	Graduate Research Symposium, First Place, Three Minute Thesis
2018	Graduate Research Symposium, People's Choice Award, Three Minute Thesis
2016-2017	College of Education, First Year Fellowship
2012	International Congress of Infant Studies Undergraduate Travel Award
2012	Phi Beta Kappa Society
2012	Graduated magna cum laude, University of Oregon
2012	Graduated with Honors in Political Science, University of Oregon
2012	Graduated with Honors in Psychology, University of Oregon

2012 Psychology Excellence Award- Undergraduate Thesis, University of Oregon
2011–2012 Psi Chi, National Psychology Honor Society, University of Oregon
2009–2012 Dean’s List, University of Oregon

PUBLICATIONS:

Cioffi, C. C., Leve, L. D., & Seeley, J.R. (2019). Accelerating the impact of science: Improving parenting practices in individuals with Opioid Use Disorder. *Parenting*, 19, 244-266. doi: 10.1080/15295192.2019.1615801

Cioffi, C. C., Leve, L. D., Natsuaki, M. N., Shaw, D. S., Reiss, D., & Neiderhiser, J. N. (2020). Does parental warmth moderate longitudinal associations between infant attention control and children’s inhibitory control? *Infant and Child Development*, 29(1). doi: 10.1002/icd.2147

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For my family.

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CHAPTER I

I. BACKGROUND AND SIGNIFICANCE

One out of six children aged 2-8 has been diagnosed with a mental, behavior, or developmental disorder (Cree et al., 2018). Disorders such as depressive disorders (DD), anxiety disorders (AD), Oppositional Defiance Disorder (ODD), Conduct Disorder (CD), and Attention Deficit/Hyperactivity Disorder (ADHD) commonly occur (3.2%; 7.1%; 7.4%; 9.4% of all U.S. children respectively) and are often co-morbid (Danielson et al., 2018; Ghandour et al., 2019). Child psychiatric disorders are not only challenging for the developing child and their parents, but also place a significant burden on health care systems given that children with psychiatric disorders make up the majority of the rise in pediatric hospitalizations currently occurring in the United States (Zima et al., 2016). Therefore, it is important to understand the genetic and environmental precursors to the manifestation of psychiatric disorders, to better understanding their etiology, and ultimately, to help improve prevention and intervention services.

Parent depression and depressive symptoms have been consistently characterized as a salient environmental risk factor for a number of child behavior problems, including child behavior problems and psychiatric disorder in early childhood (Kerr et al., 2013; Laurent, Leve, Neiderhiser, Natsuaki, Shaw, Fisher, et al., 2013; Meadows et al., 2007; Netsi et al., 2018; Pemberton et al., 2010; Psychogiou et al., 2017; Ringoot et al., 2015; Wolford et al., 2017) and child behavior problems and psychiatric disorders in middle childhood (Harold et al., 2011; Lewis, Rice, Harold, Collishaw, & Thapar, 2011; Netsi et al., 2018; Rice et al., 2010; Weissman et al., 1987). Depressive symptoms in parents of

adolescents and young adults are related to a number of psychological disorders in offspring, including DD, AD, ODD/CD, and ADHD (Marmorstein et al., 2012; Tully et al., 2008). The transmission of risk conferred by parent depressive symptoms may occur because of both the heritability of depression (Flint & Kendler, 2014; Kendler, Gardner, & Lichtenstein, 2008; Frances Rice, Harold, & Thapar, 2002; Sullivan, Neale, & Kendler, 2000; Thapar, Collishaw, Pine, & Thapar, 2012), and because of postnatal environmental exposure (Natsuaki et al., 2014). Therefore, it is important to consider both genetic and environmental aspects of parent depressive symptoms. One method to disentangle the genetic and environmental transmission of parent depressive symptoms on child outcomes is to use an adoption design where children are placed with non-relative adoptive parents. The parent-offspring adoption design allows for the separation of genetic transmission of risk and postnatal environmental risks since children are reared by parents who are not genetically related to them. Studies using genetically sensitive designs such as adoption and twin studies have found exposure to parent depressive symptoms are related to a host of outcomes, including child and adolescent behavior problems and psychiatric disorders (Harold et al., 2011; Kendler et al., 2008; Kerr et al., 2013; Laurent et al., 2013; Marmorstein et al., 2012; Pemberton et al., 2010; Tully et al., 2008).

While genetically sensitive designs have validated maternal depressive symptoms as an environmental risk, additional work is needed to better understand the contributions of postnatal exposure to parent depressive symptoms on child behavior problems and child psychiatric disorders, and salient strategies for intervention. Questions that remain

include: 1) what are the separate contributions of genetic transmission, prenatal, and adoptive mother and father depressive symptoms on child psychiatric disorders?; 2) do child behavior problems serve as a precursor of risk for psychiatric disorders?; 3) how do adoptive parents' depressive symptoms and child behavior problems influence one another throughout early and middle childhood, and are there sensitive time periods where associations are more pronounced?; and 4) are there identifiable pathways from birth parent symptoms to adoptive parent symptoms via child negative emotionality, which would reflect evocative gene-environment correlations? This dissertation will address the risks conferred by genetic risk for psychopathology and prenatal and postnatal parent depressive symptoms on child behavior problems and child psychiatric disorders.

Genetic Risk for Psychopathology and Risks associated with Prenatal Depressive Symptoms for Child Behavior Problems and Child Psychiatric Disorders

Genetic Transmission of Psychopathology. Child behavior problems and psychiatric disorders are known to be genetically influenced. Research has provided support for the “*p* factor”- or a single factor that captures a person’s liability to a mental health disorder (Selzam et al., 2018). Research using the *p* factor has found that a higher aggregate score of externalizing, internalizing, and psychotic experiences (i.e., a higher score on the *p* factor), predicts a greater incidence of brain, child developmental, and adult impairments (Caspi et al., 2014). Moreover, twin studies have found that the liability for a variety of psychiatric disorders is influenced by the same underlying genetic factors (Kendler, 1996; Lichtenstein et al., 2009). However, much of the

behavioral research to date has focused on liability for child behavior problems and psychiatric disorder conferred by parent depression, rather than general parent psychopathology. These associations have found both homotypic continuity (i.e., depressive symptoms in birth parents predicts depressive symptoms in adopted offspring) and heterotypic continuity (i.e., depressive symptoms in birth parents predicts substance use disorder in adopted offspring) (Marmorstein et al., 2012). Given that psychiatric disorders are often comorbid (Anttila et al., 2018), it unclear whether outcomes resulting in homotypic parent-offspring associations occur because general risk confers general risk, in line with the *p* factor hypothesis, or if heterotypic disorders occur because there is unique genetic variance attributed to each disorder, which is then transmitted to offspring. Research seems to suggest the former may be more likely since parent psychopathology as a whole appears to be a more salient predictor of individual child disorders rather than individual parent disorders (McLaughlin et al., 2012). While there is strong evidence to support associations between parent general psychopathology and child behavior problems and psychiatric outcomes, much of the longitudinal research has been done without the use of genetically sensitive designs, thus confounding the rearing environment with genetic transmission. Moreover, research using genetically sensitive designs has typically only included depression/depressive symptoms, rather than a range of psychopathology, to infer genetic risk. Therefore, the variance explained by genetic risk is likely underestimated. Prior work using the current sample confirms the need to estimate general liability given that birth mothers' lifetime history of a major depressive disorder predicted the intercept of child externalizing behaviors at 18 months, whereas

birth mothers' antisocial behavior predicted the intercept of child internalizing behavior at age 18 months (Kerr et al., 2013).

Additional evidence for use of a general p factor comes from work on two components of child behavior problems: child internalizing and externalizing behaviors. While internalizing and externalizing behaviors represent different constructs, there is high genetic co-morbidity between the two, with 62% of the explained covariance due to common genetic variance and correlations between the two constructs ranging from .66 and .72 in children (Cosgrove et al., 2011; Lahey et al., 2004). Genetic research across childhood, adolescence, and adulthood has found support for general genes underlying both internalizing and externalizing disorders, while also finding that environmental exposures (e.g., type of parenting a child is exposed to) differentiate the manifestation of disorder types (Eley, 1997; Kendler et al., 2011; Lahey, Van Hulle, Singh, Waldman, & Rathouz, 2011; Rhee, Lahey, & Waldman, 2015). Additionally, across ages 5 to 7, comorbid internalizing and externalizing behavior problems demonstrate considerably high continuity (Willner et al., 2016). Individuals with only internalizing or externalizing problems also have high continuity in symptom expression, but symptom continuity is less pronounced than children with comorbid behavior problems (Willner et al., 2016). Similar results have been obtained across adolescence (Snyder et al., 2016), suggesting that comorbid symptoms may be more stable over time than internalizing and externalizing behaviors that do not co-occur, perhaps in part due to a common genetic etiology underlying both internalizing and externalizing problems and disorders.

Prenatal Exposure to Depressive Symptoms. Broadly speaking, prenatal exposures, including maternal depressive symptoms, are thought to influence the developing fetus by interfering with the organization of plastic biological systems, such as the central nervous system (among others), resulting in later vulnerability to behavior problems and psychiatric disorder (Lewis, Austin, Knapp, Vaiano, & Galbally, 2015). Thus, it is important to separate maternal depressive symptoms during pregnancy from general depressive symptoms across the lifespan. Depressive symptoms during the prenatal period may be a risk factor for child behavior problems, including internalizing and externalizing behaviors, and for child psychiatric disorders such as DD, AD, CD, ODD, ADHD (Allen et al., 1998; Field, 2011; Lahti et al., 2017; Luoma et al., 2001; Mclean et al., 2018; Monk et al., 2019; Wolford et al., 2017). However, these prior findings were identified among children who were reared by their biological parents, meaning that parent depressive symptoms could have influenced both the child's prenatal and postnatal environments, and contributed to the child's genetic liability for psychopathology. In one study using a sibling comparison design, prior to examining familial clustering, prenatal depressive symptoms were related to child behavior problems; however, after adjusting for familial confounding, no associations were found between prenatal depressive symptoms and child behavior problems (Gjerde et al., 2017). Similarly, using the current sample, one study found there were no significant associations between mother's prenatal internalizing symptoms and her child's internalizing or externalizing behaviors in toddlerhood, but rather, adoptive parent depressive symptoms and birth parent lifetime internalizing symptoms predicted toddler

behavior problems (Marceau et al., 2013). These results highlight the need for genetically sensitive designs to assess the influence of prenatal depressive symptoms on child behavior and psychiatric disorders. They also suggest that, when accounting for shared genetics and environmental influences, prenatal depressive symptoms may not be an independent risk factor for later child behavior problems.

While this dissertation will only focus on child exposure to prenatal depression and will not focus on all prenatal and perinatal exposures, there are, of course, other influences which are correlated with parent depressive symptoms and are also predict child behavior problems and psychiatric disorders, such as exposure to both licit and illicit substances (Ross et al., 2015), exposure to environmental toxins (Bellinger, 2013; Williams & Ross, 2007), and birth complications (McCormick et al., 2011). As such, it is important to consider these potential contributors to child behavior and psychiatric disorders and as covariates in the current analyses.

Pathways from Early Temperament to Child Behavior Problems to Later Psychiatric Disorders

Behavior problems during early life may serve as indicators of psychiatric risk and provide insight into the transmission of psychiatric disorders (Natsuaki et al., 2014). In infancy, negative emotionality has been suggested to be a heritable underlying feature common to both internalizing and externalizing disorders (Rhee et al., 2015). Furthermore, negative emotionality partially accounts for the covariance between internalizing and externalizing disorders (Mikolajewski et al., 2013). This research suggests that negative emotionality is an indicator of conferred genetic risk for

psychopathology. In early childhood, the Child Behavior Checklist (CBCL) has been proposed as a simple, cost-effective approach to identify children at high risk for psychiatric disorder (Petty et al., 2008). This is in line with research identifying relations between the CBCL internalizing and externalizing scales and psychiatric disorders. Specifically, externalizing problems at ages 5 to 6 have been associated with ADHD and CD 1.5 years later, and internalizing problems at ages 5 to 6 have been associated with DD and AD 1.5 years later (Kroes et al., 2002). Moreover, the CBCL has demonstrated general continuity in internalizing and externalizing problems from preschool into adolescence (Pihlakoski et al., 2006).

It has been suggested that emerging behavior problems differentially predict psychiatric disorders in children of depressed parents compared to non-depressed parents (Petty et al., 2008). For example, children whose mothers had a psychiatric disorder were more likely to experience multiple psychiatric disorders, even if early on, the child only displayed risk for one type of psychiatric disorder. In comparison, mothers who did not have a psychiatric disorder were more likely to have children that exhibited a homotypic trajectory from behavior problems to the expression of a specific psychiatric disorder (e.g., externalizing behavior early on predicted externalizing disorders, such as CD) (Jobs et al., 2019). Therefore, it is important to account for maternal depressive symptoms when understanding trajectories from early behavior problems to child psychiatric disorders, since the magnitude of the contributions of child behavior problems on child psychiatric disorders for children of parents with and without depressive symptoms may differ based on the specific psychiatric diagnosis. The separable contributions of maternal

depression for children with behavior problems on child psychiatric disorders has yet to be investigated using a genetically sensitive design.

Postnatal Associations between Parent Depressive Symptoms and Child Behavior Problems

Associations with Adoptive Parent Depressive Symptoms. Most research on the associations between parent depressive symptoms and child behavior problems has focused on parent depressive symptoms postnatally, in the child's rearing environment. This large body of research has demonstrated that adoptive parent depressive symptoms are related to child behavior and psychiatric disorders (Côté et al., 2018; Goodman et al., 2011; Pratt et al., 2017; Verbeek et al., 2012). Generally, one meta-analysis found small, yet consistent, effects of maternal depressive symptoms for child behavior problems, and on general child psychopathology (Goodman et al., 2011). More specifically, maternal depression during early childhood has been found to be most important for predicting adolescent internalizing problems (Côté et al., 2018) and maternal depression during the first year has been identified as an important risk factor for child behavior problems and psychopathology (Pratt et al., 2017; Verbeek et al., 2012).

Genetically sensitive designs also find associations between both maternal and paternal depression and depressive symptoms and child behavior problems and psychiatric disorders (Natsuaki et al., 2014). However, the pattern of associations between maternal depressive symptoms and child behavior problems seems to be more consistent than those between paternal depressive symptoms and child behavior problems. Most often, associations between parent depressive symptoms and child

behavior problems, when using genetically sensitive designs, emerge when maternal depressive symptoms are considered as a composite (both observed or latent) overtime (Grabow et al., 2017; Pemberton et al., 2010), rather than paths directly estimated from maternal depressive symptoms during infancy and toddlerhood to school-age behavior problems (Hails et al., 2018). This is consistent with findings in the extant literature that show that, compared to postnatal depressive symptoms that do not persist, persistent maternal depressive symptoms are associated with an increased risk for child behavior problems in early childhood and for depression in adolescence (Netsi et al., 2018). Therefore, it is important to consider maternal depressive symptoms at multiple time points across childhood to understand the roll of persistent of parent depressive symptoms. Additionally, controlling for persistent parent depressive symptoms allows for a more specific examination of sensitive developmental periods.

Although the majority of extant literature is on mothers, men who are fathers experience higher rates of depression compared to the overall male population (10% compared to approximately 5% in the general male population), and up to 25% of fathers report experiencing postpartum depression during the first year of fatherhood (Paulson & Bazemore, 2010). Moreover, extant research has identified that fathers' depressive symptoms also predict child behavior problems and psychiatric disorders. Studies that used the current sample found a direct association between adoptive fathers' depressive symptoms during infancy and behavior problems during toddlerhood (Pemberton et al., 2010) and child internalizing symptoms at age 6 (Hails et al., 2018). Father depression has also been associated with child psychiatric disorders seven years later, in particular,

ODD (Ramchandani et al., 2008). Moreover, fathers' depressive symptoms are typically related to maternal depressive symptoms (Paulson & Bazemore, 2010), and when mothers and fathers both experience heightened depressive symptoms, children are more likely to exhibit behavior problems (Volling et al., 2018). Thus, it is important to account for both parents' depressive symptoms when estimating associations between parent depressive symptoms on child behavior problems and child psychiatric disorders.

Reciprocal Relations Between Parents' Depressive Symptoms and Child Behavior Problems. Transactional models of development consider the bidirectional associations of the child and the environment and incorporate the idea that experiences provided by the environment are not independent of the child (Sameroff & Mackenzie, 2003). Using both genetically sensitive and non-genetically sensitive designs, child behavior problems have been associated with subsequent parent depressive symptoms. In toddlerhood, there is evidence for associations between child behavior problems and maternal depressive symptoms, and between child behavior improvements and reductions in maternal depressive symptoms, and vice versa (Chazan-Cohen et al., 2007; Hails et al., 2018; Roben et al., 2015). Throughout childhood and adolescence, similar outcomes emerge such that maternal depressive symptoms predict child internalizing behaviors and both internalizing and externalizing behaviors (18-24 months; 9-17 years) exacerbate maternal depressive symptoms (Gross et al., 2009; Sellers et al., 2016; Yan & Dix, 2014). Moreover, child psychiatric disorder predicts parent psychiatric disorder such that child depression in middle childhood (7-15 years) predicts parent depressive symptoms

approximately two years later; similarly, child ADHD in middle childhood predicts parent ADHD symptoms approximately two years later (Wesseldijk et al., 2018).

Bidirectional associations may occur as the result of evocative genotype-environment correlation (rGE), which suggests that genetically influenced child characteristics may evoke specific parent responses (Ge et al., 1996). Indeed, infant negative emotionality (e.g., fewer facial expressions, fewer vocalizations), due in part to parent depressive symptoms, have been found to evoke a depressed response from nondepressed adults (e.g., fewer vocalizations towards the infants, less responsiveness) (Field et al., 1988). This suggests that infants who are at-risk due to parent depressive symptoms possess characteristics that elicit specific non-adaptive responses from their caregiver. However, it is unclear if infant negative emotionality emerges as a result of genetic transmission of psychopathology, or as a result of the experience of being reared by a parent with depressive symptoms (or both). Therefore, there is a need to test paths from both genetic risk and adoptive parent depressive symptoms to infant negative emotionality, and in turn to adoptive parent depressive symptoms, to examine potential child effects on parent depressive symptoms that may or may not arise from genetic influences on child behavior problems.

Most of the studies examining transactional models of child behavior problems and parent depressive symptoms have used samples of genetically related individuals, thus, it is unclear whether associations occurred as a result of a biological predisposition, since parents' environmental contributions are confounded with their genetic contributions. Few models using a genetically sensitive design have modelled

transactional associations using a longitudinal latent variable design, and those that have used very few time points (Chazan-Cohen et al., 2007; Roben et al., 2015). Furthermore, the extant literature using genetically sensitive designs of child behavior problems on fathers is sparse to nonexistent, and thus warrants attention.

Summary

The existing literature points to critical gaps in examination of the interplay between parent depressive symptoms and child behavior problems. First, prior studies that examine the associations between parent depressive symptoms and children's behavior problems within a traditional family structure-- biological parents rearing their biological children--are unable to disentangle risk conferred by parent depressive symptoms genetically and those that occur as a result of the rearing environment. Second, models seeking to understand the complex interplay between parent depressive symptoms and child behavior problems have not routinely accounted for the individual differences in initial and chronic parent depressive symptoms and child behavior problems and how stability and change in a parent's depressive symptoms is related to a child's manifestation of behavior problems. Third, parent depressive symptoms on child behavior problems and the reciprocal effects of child behavior problems on parent depressive symptoms are rarely considered simultaneously to adequately model bi-directional associations. Fourth, fathers must be considered in order to better capture the rearing environment and assess how fathers may contribute unique variance to child behavior problems and child psychiatric disorders. An adoption design allows for an estimation of genetic transmission of psychopathology and the postnatal environmental

depressive symptoms. Understanding the unique contributions of each allows for the development of interventions that can be appropriately tailored to identify children at risk and to identify salient time periods for interventions for parents experiencing depressive symptoms.

CHAPTER II

II. RESEARCH QUESTIONS AND HYPOTHESES

1) What are the separate contributions of genetic risk for psychopathology and prenatal parent depressive symptoms on child behavioral problems across early and middle childhood, and on child psychiatric disorders (ADHD & ODD/CD; Depression/Anxiety) in middle childhood?

Hypotheses

Genetic risk for psychopathology: In line with the “*p*” factor hypothesis, child behavior problems and psychiatric disorders will be predicted by birth parent psychopathology.

Prenatal influences: In line with prior research using genetically sensitive designs, birth mother prenatal depressive symptoms will be unrelated to child behavioral problems and child psychiatric disorders after accounting for birth parent psychopathology and adoptive parent depressive symptoms.

2) Do child behavior problems serve as an early indicator of psychiatric disorder?

Hypothesis

Chronic child behavior problems from child age 18 months to 6 years will predict each psychiatric disorder.

3) How do adoptive parent depressive symptoms and child behavioral problems simultaneously influence one another throughout early and middle childhood, and are there sensitive periods where time-specific associations are present?

Hypotheses

Chronic adoptive father and mother depressive symptoms will be related to child behavior problems, and child psychiatric disorders.

I expect that adoptive mother and father depressive symptoms during very early childhood (when children are nine months of age) will be associated with child behavior problems at 18 months and that other cross-lagged associations between parent depressive symptoms and child behavioral problems which will likely not be significant.

I expect that child behavioral problems in later childhood, when children are six years of age, will demonstrate a time-specific association with concurrent adoptive parent depressive symptoms for both mothers and fathers compared to child behavioral problems at ages 18 months, 27 months, and 4.5 years.

4) Are there identifiable pathways from birth parent symptoms to adoptive parent symptoms via negative emotionality, which would reflect evocative gene-environment correlations?

Hypothesis

I expect that children with a genetic liability for temperamentally challenging behavior, as marked by the relation between birth parent psychopathology and heightened infant emotionality, will evoke a depressive response from their adoptive parents.

CHAPTER III

III. METHOD

Participants

The current study uses data drawn from the Early Growth and Development Study (EGDS; Leve et al., 2019, 2013). EGDS is a prospective, longitudinal study consisting of 561 linked sets of adoptive families (adopted child, adoptive parents, and birth parents). Data collection occurred with two cohorts: cohort I was 361 linked sets and cohort II was 200. Participants were recruited via 45 adoption agencies in 15 states across the United States (see Leve et al., 2019, 2013 for further description). Participants were eligible for participation if (1) the adoption was domestic, (2) the infant was placed with a non-relative adoptive family (3) and the infant was placed prior to 3 months of age ($M = 7.11$ days, $SD = 13.28$), (4) the infant had no known major medical conditions, and (5) the birth mother and adoptive parents could read or understand English at least at an eighth-grade level. Same-sex couples were included in the sample.

Procedures

Participants were recruited through adoption agencies located throughout the United States following the birth of a child. If birth mothers provided permission for EGDS to contact them, the EGDS birth parent recruiter contacted the birth mother. Once the birth mother returned a signed consent form via postage-paid mail and began the assessment, she was considered an active study participant. Next, the EGDS adoptive family recruiter attempted to recruit the adoptive family using contact information provided by the adoption agency. If the adoptive parents agreed to participate, they were

sent informed consent forms and additional study information. Like birth mothers, adoptive parents were considered recruited once they returned a signed informed consent form and began the first assessment. After the birth mother and adoptive parents were recruited, project staff attempted to recruit the birth father.

Assessments included questionnaires, in-person interviews, and standardized testing for birth parents, adoptive parents, and children; diagnostic interviews with adoptive parents (about themselves and about the adopted child) and birth parents and in-person assessments. In-person assessments for adoptive family occurred at ages 9 months, 18 months, 27 months, and age 4.5, 6, 7, and 8 years and for birth parents at 5- and 18-months postpartum and 4.5 years postpartum. The only exception was at 4.5 years, cohort II adoptive parent data were collected by mail or e-mail and cohort II birth parents did not participate. In-person visits lasted approximately 3–4 hrs. each and were usually conducted in the participant's home.

Separate birth parent and adoptive family recruiters and assessors were used to ensure that project staff did not transfer information between members of the adoption triad. At any point, if the birth mother or adoptive family declined participation or was unable to be contacted, recruitment efforts for that adoption triad ceased. However, once an individual had consented to participate, that individual continued as a participant. For additional recruitment and assessment details, please see Leve et al. (2019).

Measures

Birth parent psychopathology. A latent genetic risk variable for birth mothers and birth fathers was used to determine genetic liability for child behavior problems and

psychiatric disorders. An overall factor of birth mother and father psychopathology (p factor), previously computed for this EGDS (Marceau et al., 2019), was used to estimate genetic risk for child behavior problems and psychiatric disorders. The p factor is a higher-order factor based on factor scores for two lower order factors: internalizing problems and externalizing problems. Each of the lower order factors were computed using four indicators, symptom count, diagnosis count, age of onset, and first-degree relatives with a disorder. The indicators were derived from birth parents' self-report data at 18 and 54 months post-adoption. For the first three indicators, the Composite International Diagnostic Instrument (Kessler & Üstün, 2004) and the Diagnostic Interview Schedule (Robins et al., 1981) were used to create birth parents counts for (a) number of disorders on the diagnostic interview, (b) number of symptoms endorsed on the diagnostic interview, and (c) age of onset of each disorder. For internalizing disorders, the diagnostic categories assessed included major depression, brief recurrent depression, dysthymia, separation anxiety, adult separation anxiety, social phobia, agoraphobia (with and without panic), panic disorder, specific phobia, and generalized anxiety. For externalizing disorders, the diagnostic categories assessed included CD, antisocial personality disorder, alcohol abuse and dependence, drug abuse and dependence, and tobacco dependence. For both externalizing and internalizing scores, episodes of prenatal symptoms were excluded. The fourth indicator was the proportion of first-degree relatives with either internalizing or externalizing problems. This indicator was calculated as the maximum proportion of first-degree relatives [mother, father, and up to three siblings] the birth parent rated as having externalizing problems, internalizing

problems and/or substance use problems. Indicators were entered into a principal component analysis separately for each disorder and extracted factor scores were aggregated to create a composite score for internalizing symptoms and for externalizing symptoms and aggregated again to create the higher order, *p* factor. For additional information see Marceau et al. (2019).

Prenatal depressive symptoms. Birth mother prenatal depressive symptoms were collected using a subset of 5 continuous items from the Beck Depression Inventory (BDI; Beck et al., 1988; Cronbach's $\alpha = .93$). Using the BDI, participants were asked to choose one of four statements that range from positive to depressed feelings about life in the past week. For example, participants were asked to choose among the following statements regarding their feelings of sadness in the last week: 1) I do not feel sad, 2) I feel sad, 3) I am sad all the time and I can't snap out of it, 4) I am so sad or unhappy that I can't stand it.

Adoptive parent depressive symptoms. Adoptive mothers and fathers completed self-reports of their depressive symptoms when children were 9 months, 18 months, 27 months, 4.5 years, and 6 years of age. There was variation across the study as to which measure of depressive symptoms was used, however, either the BDI (Beck et al., 1988) or Center for Epidemiologic Studies- Depression Scale (CES-D; Lewinsohn et al., 1997) was used at each time point. Since there was variation as to which scale was used at each time point, the scores were converted to z-scores for ease of comparison. The BDI was administered when children were 9 months (Cronbach's $\alpha = .73, .73$; adoptive mother and adoptive father, respectively), 18 months (Cronbach's $\alpha = .79, .81$),

and 27 months (Cronbach's $\alpha = .83, .86$). The original BDI contains 21 items, but the version for adoptive parents contains only 20, as the item that asks about suicidal ideation (item 9) was dropped due to limited funds to provide referrals. Scores on the BDI range from 0-60 with higher scores indicating greater severity of depression. The CES-D is 20 items, each rated on a scale of 0 to 3 for a range of scores from 0 to 60, with higher scores indicating a greater severity of depression. A score of 16 or more is considered an indicator of depression. The CES-D was administered when children were 4.5 years (Cronbach's $\alpha = .86, .83$) and 6 years (Cronbach's $\alpha = .90, .85$). Adoptive mother depressive symptoms and adoptive father depressive symptoms were tested in separate models as latent variables, but in each model, the other was controlled for using an observed composite of depressive symptoms from 9 months to 6 years.

Infant negative emotionality. At 9 months, the Infant Behavior Questionnaire (IBQ; Rothbart, 1981) was used to assess infant negative emotionality with the 20-item Distress to Limitations scale (Cronbach's $\alpha = .83$ and $.83$, respectively) and the 20-item Fearfulness scale (Cronbach's $\alpha = .73$ and $.72$, respectively). When ratings from both adoptive mother and father report were available (87% of the sample), a mean score was computed ($r = .56$ and $.55, p < .05$).

Child behavior problems. Scores on the parent-reported version of the Child Behavior Checklist (CBCL) for total behavior problems were used to assess child behavior problems at the ages of 18 months, 27 months, 4.5 years, and 6 years. For all children in cohort I and children under age 6 in cohort II, the preschool version (CBCL/1½-5; Achenbach & Rescorla, 2000) was used. For children in cohort II who

were age 6, the school age version (CBCL/6-18; Achenbach & Rescorla, 2001) was administered. Raw scores of the CBCL total behavior problems were converted into z-scores to create comparable factors across the different versions of the CBCL, since T scores are normed for each version and thus are not directly comparable. When available, adoptive mother and father report were averaged (r 's: 18 months = .43, 27 months = .49, 4.5 years = .47, 6 years = .52) at each time point prior to z-scoring (Cronbach's α : 18 months = .91, .93; 27 months = .92, .93; 4.5 years = .94,.94; 6 years = .94, .94).

Child psychiatric disorders. When children were between 6 - 8 years of age, adoptive parents were interviewed in their homes and asked to report their child's psychiatric symptoms during the last three months, using the Preschool Age Psychiatric Assessment (PAPA). The PAPA is a structured interview lasting approximately 100 minutes and includes sections on a range of psychiatric symptoms (Egger et al., 2006). These sections also include behaviors regarded as typical child behaviors which provide a normative reference for determining when behaviors reach a clinically significant level. The PAPA was deemed the most suitable option for psychiatric assessment for children at this age by the PAPA developers because the Child and Adolescent Psychiatric Assessment, which uses child self-report of symptoms, is only reliable and valid for ages 9 through 18. Four variables were created from the diagnoses generated by the PAPA: DD, AD, ADHD, and CD/ODD. These were coded as presence/absence of disorder. DD exists as a dichotomous variable from the PAPA; a child was coded as having the presence of AD if they had the presence of separation anxiety disorder, general anxiety disorder, specific phobia or social phobia; ADHD presence/absence exists as a variable

from the PAPA; and the presence absence of ODD/CD was counted if a child had the presence of either of these diagnoses. In terms of prevalence of child psychiatric disorders, of the 396 children who had complete data from the PAPA, 21 met the criteria for a diagnosis of any DD, 228 met the criteria for a diagnosis for any AD, 55 children met the criteria for a diagnosis of CDD/ODD, and 59 met the criteria for a diagnosis of ADHD.

Covariates. *Prenatal and perinatal risk.* Along with prenatal exposure to maternal depressive symptoms, there are several other prenatal and perinatal risk factors to consider because they have been shown to be associated with child behavior problems and psychiatric disorders (Bellinger, 2013; McCormick et al., 2011; Ross et al., 2015; Williams & Ross, 2007). These include pregnancy complications, neonatal complications, substance use, and exposure to toxins. Composite scores for each of these risks were assessed using birth mother report of pregnancy complications, including medical risks, drug use, and chemical exposures, using a pregnancy screener and a pregnancy calendar method to enhance recall (Caspi et al., 1996) at the first birth mother time point of the study (child age 3-6 months). The birth mother was first asked to identify key events (e.g., birthdays, anniversaries, holidays) that occurred during her pregnancy on a one-page pregnancy calendar. She then recalled the occurrence of a series of any obstetric complications during her pregnancy. Scoring was derived from the McNeil-Sjostrom Scale for Obstetric Complications (McNeil et al., 1994). When medical records were more reliable, they were used in lieu of birth mother recall. Responses to each item were assigned a score from 1 (not harmful or relevant) to 6 (very great harm to

or deviation in offspring), indicating the level of risk indicated by each response.

Consistent with McNeil-Sjostrom scoring protocols, a prenatal risk score that reflected severity of risk was created by summing items with a score of 3 or greater. For more information about how these scales were determined, see Marceau, Araujo-Greecher, Miller, and Massey (2016).

Openness of adoption. Degree of openness in adoption was assessed using perceptions of the degree of contact between parties, as rated by the birth mother, adoptive mother, and adoptive father to control for similarities that may result from contact between birth and adoptive families. This was a composite score when children were 9 months of age. Each participant rated the degree and nature of contact and communication with their counterpart. The scale ranged from 1 very closed, to 7 very open (IRR = .66-.81, $ps < .001$) For additional information see Ge et al. (2008).

Child sex. Child sex was considered as a covariate given that girls typically experience more internalizing symptoms and disorders whereas boys typically experience more externalizing symptoms and disorders (Liu, 2004). Child sex was coded as 0 for male and 1 for female.

Analytic Plan

Missing Data. Missing data was evaluated using missing values analyses and standard attrition analyses. First, Little's test of missing data (Little, 1988) was used to identify if there were patterns in missing data. Little's test of missingness was significant, indicating that there were patterns of missingness in the data. Attrition analyses revealed that, compared to baseline characteristics, there were no differences between children,

adoptive mothers, and adoptive fathers who were retained in the study compared to those who did not complete the final assessment. There were also no differences on birth mother variables for birth fathers who did and did not participate. Thus, it is unclear where there was a mechanism for missingness. When data are missing at random, full information maximum likelihood is appropriate since it uses all available information from the observed data (Jeličić et al., 2009). When data are not missing at random, full information maximum likelihood may produce biased standard errors but using full information maximum likelihood with structural equation modelling accounts for item measurement error and typically performs similarly to multiple imputation (Little, Jorgensen, Lang, & Moore, 2013). Thus, full information maximum likelihood was used to account for missing data.

Analyses. The cross-lagged panel model (CLPM) has been previously used to study causal influences in developmental models. However, prior research has highlighted that the CLPM does not adequately control for the trait-like or time-invariant nature of each construct and thus leads to biased estimates of cross-lagged paths. The random intercept-cross lag panel model (RI-CLPM) has been proposed as an anecdote to this dilemma as it allows for accurate estimates of reciprocal processes over-time at the within-dyad level (Hamaker et al., 2015). Thus, the RI-CLPM provides an ideal means by which to model the dynamic interplay between parent depressive symptoms and child behavior problems (Mund & Nestler, 2018), as it allows for an examination of reciprocal relations between parent depressive symptoms and child behavior problems as they

unfold over time and delineates within- and between-person effects (Hamaker et al., 2015).

Within the RI-CLPM, it was also important to use a robust estimator given that maximum likelihood estimation, the most commonly used estimator for structural equation modelling (Kline, 2016), assumes that residuals are normally distributed. However, was this not the case as most of the variables of interest (i.e., depressive, behavior problems) were positively skewed. Thus, robust maximum likelihood was more appropriate for analyses. RI-CLPM was used to examine hypotheses 1-3. Specifically, the reciprocal nature of adoptive parent depressive symptoms and child behavior problems were modelled while controlling for the trait-like stability of each construct.

Simultaneously, birth parent psychopathology, prenatal depressive symptoms, and infant negative emotionality were modelled to predict the random intercept. Birth parent psychopathology, prenatal depressive symptoms, infant negative emotionality, adoptive parent depressive symptoms and child behavior problems were modelled to predict each child psychiatric disorder. The RI-CLPM was run twice: first, examining the reciprocal relations between adoptive mother depressive symptoms and child behavior problems, controlling for an observed composite of adoptive father depressive symptoms, and second, examining the reciprocal relations between adoptive father depressive symptoms and child behavior problems whilst controlling for an observed composite of adoptive mother depressive symptoms.

To test hypothesis 4, a path model was tested with birth mother psychopathology to infant negative emotionality at 9 months and from infant negative emotionality at 9

months to an average of adoptive mother and father depressive symptoms. Adoptive mother and father depressive symptoms at 9 months were not included in the average score so that associations between infant negative emotionality and parent depressive symptoms was not overestimated with respect to time-varying associations at 9 months. Within the path model, an indirect effect coefficient was computed that was a function of the compound pathway a (birth parent psychopathology to infant negative emotionality) and b (infant negative emotionality to adoptive parent depressive symptoms (MacKinnon, 2008) using lavaan (Rosseel, 2011).

CHAPTER IV

IV. RESULTS

Preliminary Analyses

Table 1 presents correlations among study variables. Birth mother psychopathology was correlated with birth father psychopathology and prenatal depressive symptoms, and with adoptive mother depressive symptoms at 27 months and child behavior problems at 27 months. There were no other correlations between birth parent psychopathology nor prenatal depressive symptoms and any other variables.

Infant negative emotionality at 9 months was associated with adoptive mother depressive symptoms at 9 months and 6 years and adoptive father depressive symptoms at 9 months, 18 months, 4.5 years, and 6 years. Infant negative emotionality was also associated with child behavior problems at each time point: 18 months, 27 months, 4.5 years, and 6 years, and with child ODD/CD but not with child DD, AD, or ADHD.

Adoptive mother depressive symptoms were related to child behavior problems at each time point. Adoptive father depressive symptoms were related to child behavior problems at most time points, but not all time points, as seen in table 1. Adoptive mother depressive symptoms at 4.5 years were associated with child ODD/CD; at 6 years were associated with child ADHD; at 18 months, 27 months, 4.5 years, and 6 years were associated with child DD; and at 9 months, 27 months, 4.5 years, and 6 years were associated with child AD. Adoptive father depressive symptoms at 18 months were associated with child AD.

Table 1 Correlations Among Main Study Variables

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	
1. BF <i>p</i>																						
2. BM <i>p</i>	.17**																					
3. Prenatal dep	.07	.18**																				
4. Negative emo 9m	-.00	-.05	.06																			
5. AM dep 9m	.05	.01	-.03	.12**																		
6. AM dep 18m	.02	.01	-.05	.04	.63**																	
7. AM dep 27m	.03	.09*	-.04	.09	.56**	.62**																
8. AM dep 4.5y	.01	-.03	-.07	.04	.42**	.37**	.49**															
9. AM dep 6y	-.05	-.05	-.07	.11*	.38**	.36**	.45**	.56**														
10. AF dep 9m	-.01	.05	.02	.14**	.10*	.11*	.10*	.02	.02													
11. AF dep 18m	.06	.07	.03	.11*	.10*	.17**	.19**	-.00	.12*	.65**												
12. AF dep 27m	.07	.04	.01	.03	.07	.11*	.22**	.04	.20**	.58**	.73**											
13. AF dep 4.5y	.07	.06	.03	.17**	.15**	.10	.13*	.11*	.14**	.51**	.54**	.45**										
14. AF dep 6y	.02	.04	.08	.17**	.18**	.13*	.13**	.10	.20**	.47**	.56**	.54**	.61**									
15. Child beh 18m	.03	.05	.00	.37**	.19**	.24**	.19**	.13**	.22**	.15**	.17**	.12**	.13*	.13**								
16. Child beh 27m	.08	.12**	.03	.28**	.20**	.24**	.27**	.11*	.22**	.19**	.19**	.19**	.10*	.14**	.74**							
17. Child beh 4.5y	.06	.05	.01	.21**	.17**	.13*	.13**	.14**	.20**	.15**	.15**	.10	.09	.07	.52**	.55**						
18. Child beh 6y	.09	.06	.05	.13**	.17**	.15**	.18**	.13*	.26**	.10*	.16**	.18**	.10	.17**	.48**	.56**	.73**					
19. Child ODD/CD	-.01	-.01	-.02	.13*	.07	.00	.02	.14**	.07	-.05	.01	.03	-.00	-.01	.26**	.23**	.33**	.32**				
20. Child ADHD	-.03	-.02	.03	.05	.04	.08	.09	.08	.11*	.04	.04	.09	-.02	-.03	.17**	.13*	.29**	.28**	.25**			
21. Child depression	-.06	.05	-.00	.01	.10	.15**	.16**	.12*	.19**	-.01	.08	.06	-.02	.01	.15**	.08	.22**	.22**	.23**	.22**		
22. Child anxiety	-.05	-.02	.03	.05	.12*	.09	.18**	.12*	.12*	.06	.11*	.08	.06	.07	.19**	.23**	.24**	.24**	.16**	.17**	.16**	

Note. BF = birth father, BM = birth mother, AM = adoptive mother, AF = adoptive father, *p* = psychopathology, dep = depressive symptoms, beh = behavior problems, ODD/CD = Oppositional Defiant Disorder/Conduct Disorder, ADHD = Attention Deficit/Hyperactivity Disorder. Correlations were estimated after accounting for covariates **p* < .05. ***p* < .01.

Adoptive mother depressive symptoms were correlated at all time points, as were adoptive father depressive symptoms. The only *non-significant* associations between adoptive mother and father depressive symptoms were between adoptive mother symptoms at 9 months and adoptive father symptoms at 27 months; adoptive mother symptoms at 18 months and adoptive father symptoms at 4.5 years; adoptive mother symptoms at 4.5 years and adoptive father symptoms at 9 months, 18 months, 27 months, and 6 years; and adoptive mother symptoms at 6 years and adoptive father symptoms at 9 months.

Child behavior problems were correlated at each time point, and each time point was correlated with each child psychiatric disorder, except that there was no association between child behavior problems at 27 months and child depression.

Hypothesis Testing

Two RI-CLPM models were fit to the data using structural equation modelling. The first model specified correlations and cross-lagged paths between adoptive mother depressive symptoms and child behavioral problems with random latent intercepts for each, while controlling for adoptive father depressive symptoms using an average of adoptive father depressive symptoms over time as an observed variable. Fit indices for the adoptive mother RI-CLPM indicated good fit to the data (Robust Comparative Fit Index (CFI) = .97; Standardized Root Mean Square Residual (SRMR) = .04; Robust Root Mean Square Error of Approximation (RMSEA) = .04) by standards reported in Hu and Bentler (1995). Indicators of adoptive mother depressive symptoms loaded onto a random intercept (std. coefs. = .59- .64) as did indicators of child behavior problems intercept

(std. coefs. = .63- .70). The second model specified correlations and cross-lagged paths between adoptive father depressive symptoms and child behavioral problems in the same manner as previously described for the adoptive mother RI-CLPM. Fit indices for the adoptive father RI-CLPM indicated good fit to the data (Robust CFI = .97; SRMR = .04; Robust RMSEA = .04). In the adoptive father model, indicators of adoptive father depressive symptoms loaded onto a random intercept (std. coefs. = .67- .76) as did indicators of child behavior problems intercept (std. coefs. = .63- .70). Results from both models are presented in parentheses throughout the text, with adoptive mother RI-CLPM results first and adoptive father RI-CLPM results second. Figure 1 presents the adoptive mother RI-CLPM and Figure 2 presents the adoptive father RI-CLPM. Estimates for predictors of child psychiatric disorder are presented in Table 2.

Table 2 Results from RI-CLPMs for Predictors of Child Psychaitric Disorders

	Std. coef.		Unstd. coef,		SE		z		p	
ODD/CD										
BM psychopathology	-0.05	-0.05	-0.04	-0.03	0.03	0.03	-1.08	-1.03	.280	.303
BF psychopathology	-0.04	-0.03	-0.02	-0.02	0.04	0.04	-0.61	-0.58	.545	.561
Prenatal depressive symptoms	-0.03	-0.02	-0.01	-0.01	0.01	0.01	-0.64	-0.52	.524	.602
AM/AF depressive symptoms (latent)	-0.09	-0.13	-0.11	-0.16	0.12	0.09	-0.96	-1.81	.340	.071
AF/AM depressive symptoms (observed)	-0.10	-0.06	-0.09	-0.06	0.07	0.07	-1.33	-0.93	.185	.353
Child negative emotionality at 9 months	-0.02	-0.02	-0.03	-0.02	0.07	0.07	-0.40	-0.36	.692	.723
Child behavior problems	0.50	0.51	0.64	0.64	0.15	0.14	4.38	4.54	.001	.001
ADHD										
BM psychopathology	-0.05	-0.05	-0.03	-0.03	0.03	0.03	-1.00	-1.00	.316	.318
BF psychopathology	-0.05	-0.05	-0.03	-0.03	0.03	0.03	-1.10	-1.10	.273	.270
Prenatal depressive symptoms	0.04	0.03	0.01	0.01	0.01	0.01	0.61	0.59	.541	.553
AM/AF depressive symptoms (latent)	0.02	-0.03	0.03	-0.03	0.11	0.09	0.28	-0.37	.778	.715
AF/AM depressive symptoms (observed)	-0.02	0.00	-0.02	0.00	0.07	0.06	-0.24	0.04	.811	.969
Child negative emotionality at 9 months	-0.06	-0.06	-0.06	-0.06	0.06	0.06	-0.99	-1.01	.322	.312
Child behavior problems	0.31	0.33	0.41	0.43	0.13	0.13	3.12	3.35	.002	.001

Table 2 Results from RI-CLPMs for Predictors of Child Psychiatric Disorders

	Std. coef.		Unstd. Coef.		SE		z		p	
Depression										
BM psychopathology	0.04	0.04	0.02	0.02	0.03	0.03	0.71	0.69	.479	.489
BF psychopathology	-0.08	-0.08	-0.04	-0.04	0.02	0.02	-2.47	-2.49	.013	.013
Prenatal depressive symptoms	0.00	0.00	0.00	0.00	0.01	0.01	0.01	-0.10	.989	.921
AM/AF depressive symptoms (latent)	0.18	-0.03	0.17	-0.03	0.10	0.07	1.74	-0.38	.082	.701
AF/AM depressive symptoms (observed)	-0.03	0.13	-0.02	0.10	0.06	0.06	-0.38	1.75	.705	.081
Child negative emotionality at 9 months	-0.08	-0.08	-0.06	-0.06	0.05	0.05	-1.27	-1.25	.205	.211
Child behavior problems	0.21	0.25	0.19	0.22	0.09	0.09	2.20	2.61	.028	.009
Anxiety										
BM psychopathology	-0.06	-0.06	-0.05	-0.05	0.05	0.05	-1.11	-1.09	.269	.278
BF psychopathology	-0.07	-0.07	-0.07	-0.07	0.04	0.04	-1.53	-1.52	.127	.128
Prenatal depressive symptoms	0.04	0.04	0.01	0.01	0.02	0.02	0.82	0.75	.413	.451
AM/AF depressive symptoms (latent)	0.08	0.00	0.15	-0.01	0.14	0.10	1.08	-0.05	.281	.962
AF/AM depressive symptoms (observed)	0.00	0.06	0.00	0.08	0.07	0.08	0.04	1.01	.967	.311
Child negative emotionality at 9 months	-0.08	-0.08	-0.11	-0.11	0.08	0.08	-1.39	-1.33	.165	.183
Child behavior problems	0.33	0.34	0.56	0.57	0.14	0.14	4.00	4.10	.001	.001

Note. Estimates for the adoptive mother RI-CLPM are listed in columns on the left and adoptive father RI-CLPM are on the right. BF = birth father, BM = birth mother, AM = adoptive mother, AF = adoptive father, ODD/CD = Oppositional Defiant Disorder/Conduct Disorder, ADHD = Attention Deficit/Hyperactivity Disorder.

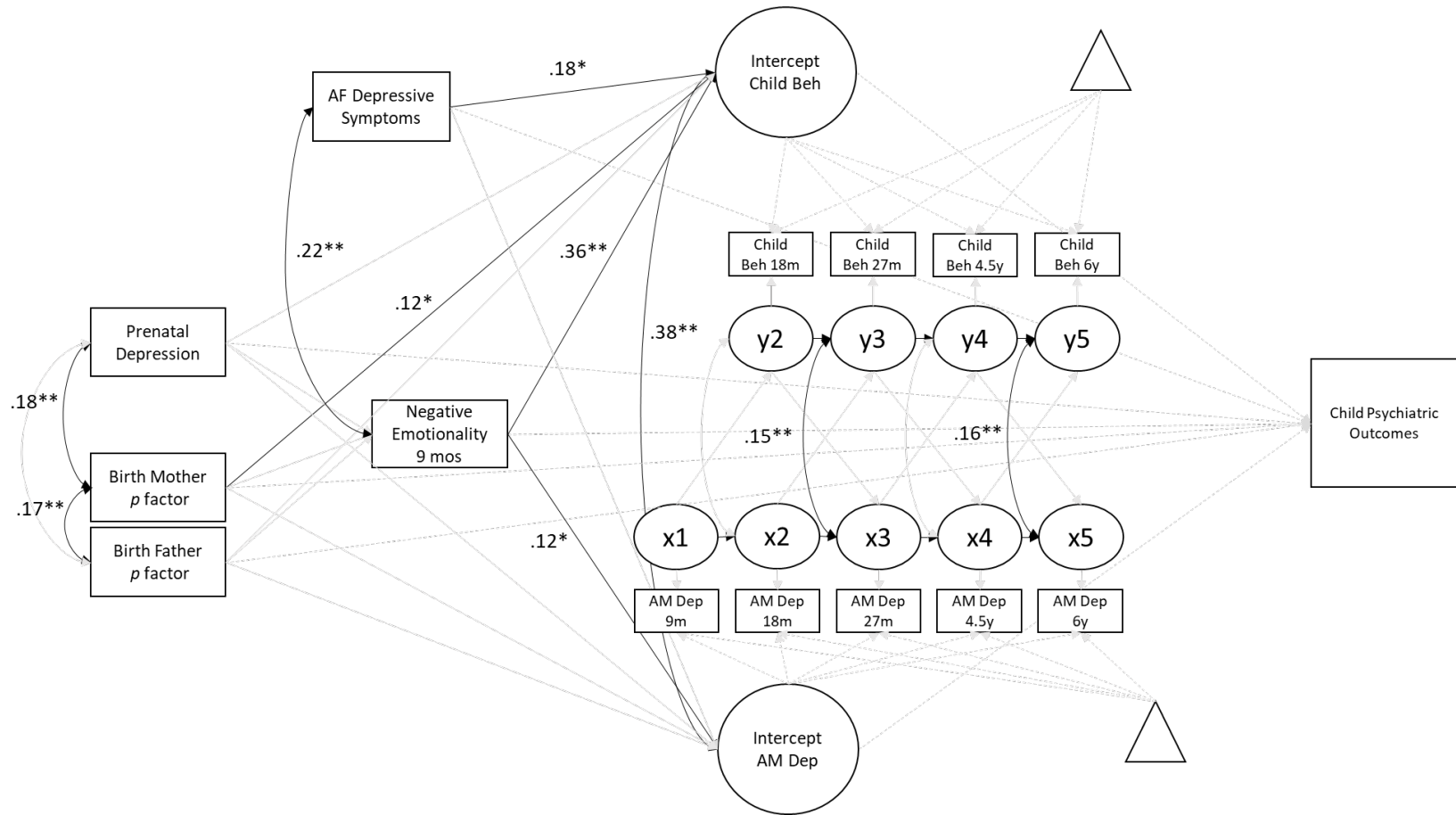


Figure 1. Random Intercept Cross-Lagged Panel Model between adoptive mother depressive symptoms and child behavior problems. Significant paths are depicted in black. Dotted lines represented paths that were tested within the model but estimates are not provided for visual clarity. Triangles represent mean structure. AM = adoptive mother, AF = adoptive father, Beh = behavior problems, Dep = depressiv symptoms.
 * = $p < .05$; ** = $p < .01$.

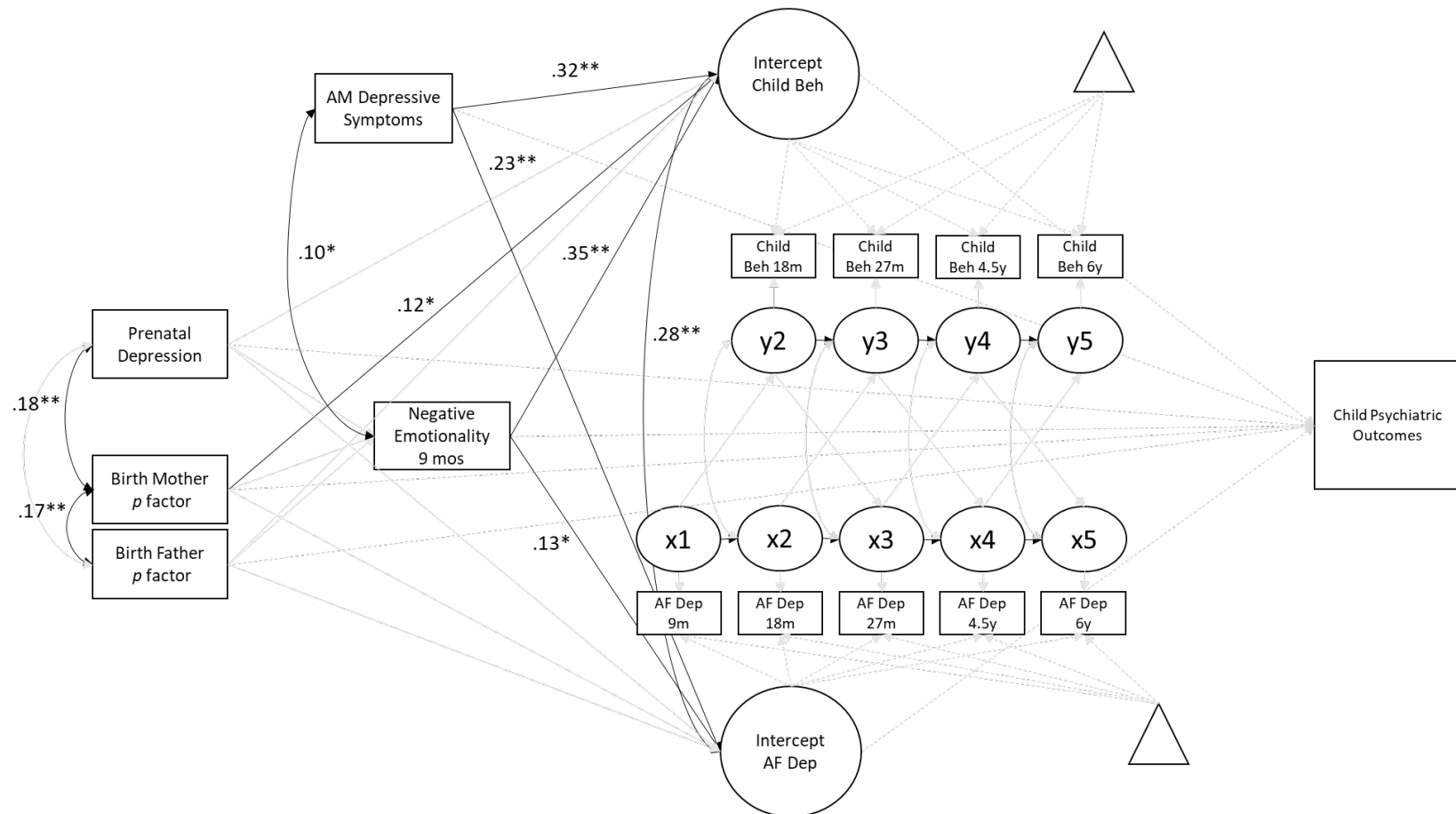


Figure 2. Random Intercept Cross-Lagged Panel Model between adoptive father depressive symptoms and child behavior problems. Significant paths are depicted in black. Triangles represent mean structure. Dotted lines represented paths that were tested within the model but estimates are not provided for visual clarity. AF = adoptive father, AM = adoptive mother, Beh = behavior problems, Dep = depressiv symptoms.
 * = $p < .05$; ** = $p < .01$.

Question 1. Question 1 asked, what are the separate contributions of genetic risk for psychopathology and prenatal depressive symptoms on child behavioral problems across early and middle childhood, and on child psychiatric disorders (DD, AD, ADHD & ODD/CD) in middle childhood? The first hypothesis was, in line with the “*p*” factor hypothesis, that child behavior problems and behavioral disorders would be predicted by birth parent psychopathology. Results from the RI-CLPM indicated that child behavior problems were predicted by birth mother (std. coef. = .12, .12; $p = .03, .03$), but not birth father, psychopathology (std. coef. = .06, .06; $p = .22, .20$). The only relation between birth parent psychopathology and child psychiatric outcomes was between birth father psychopathology and child DD (std. coef. = -.08, -.05; $p = .01, .01$).

Second, it was hypothesized that birth mother prenatal depressive symptoms would be unrelated to child behavioral problems and child psychiatric disorders after accounting for birth parent depressive symptoms or parent psychopathology and adoptive parent depressive symptoms. Prenatal depressive symptoms were unrelated to child behavioral problems and child psychiatric outcomes. Findings were consistent with the hypothesis. Birth mother prenatal depressive symptoms was unrelated to child behavior problems (std. coef. = -.03, -.07; $p = .59, .88$), child AD (std. coef. = .04, .04; $p = .41, .45$), child DD (std. coef. = .01, -.01; $p = .99, .92$), child ADHD (std. coef. = .04, .03; $p = .54, .55$), and child CD/ODD (std. coef. = -.03, -.02; $p = .52, .60$).

Question 2. Question 2 asked, do child behavior problems serve as an early indicator of psychiatric disorder? It was hypothesized that chronic child behavior problems from child age 18 months to 6 years would predict each psychiatric disorder.

Results fully supported this hypothesis, such that higher scores on child behavior problems predicted child AD (std. coef. = .33, .34; $p < .01$), DD (std. coef. = .21, .25; $p = .03, .01$), ADHD (std. coef. = .31, .33; $p < .01$), and CD/ODD (std. coef. = .51, .55; $p < .01$). Additionally, infant negative emotionality at 9 months of age predicted child behavior problems (std. coef. = .36, .36; $p < .01$) but not child psychiatric disorders.

Question 3. The third question was, how do adoptive parent depressive symptoms and child behavioral problems simultaneously influence one another throughout early and middle childhood, and are there sensitive periods where time-specific associations are present? First, the hypothesis was that chronic adoptive father and mother depressive symptoms would be related to child behavior problems and child psychiatric disorders. In line with this hypothesis, chronic adoptive mother and father depressive symptoms were related to chronic behavior problems (std. coef. = .38, .28; p 's $< .01$), but did not predict child psychiatric disorders: AD (std. coef. = .08, -.01; $p = .28, .96$), DD (std. coef. = .18, -.03; $p = .08, .70$), ADHD (std. coef. = .02, -.03; $p = .78, .71$), and CD/ODD (std. coef. = -.08, -.13 $p = .34, .07$).

Second, it was hypothesized that adoptive parent depressive symptoms during very early childhood (when children were nine months of age) would demonstrate a cross-lagged association with child behavioral problems at 18 months for both father and mother depressive symptoms, compared to other cross-lagged associations between parent depressive symptoms and child behavioral problems, which would not be significant. This hypothesis was not supported, as results indicated that none of the cross-

lagged associations from adoptive mother and father depressive symptoms to child behavior problems were significant.

Third, it was expected that child behavioral problems in later childhood, when children were six years of age, would be associated with concurrent adoptive parent depressive symptoms for both mothers and fathers compared to child behavioral problems at ages 18 months, 27 months, and 4.5 years. There was partial support for this hypothesis for maternal depressive symptoms but not for father depressive symptoms. Maternal depressive symptoms and child behavior problems were only significant at ages 27 months (std. coef. = .15, $p < .01$) and 6 years (std. coef. = .16, $p < .01$). These two associations were comparable in magnitude. There were no associations between adoptive father depressive symptoms and child behavior problems at any time point.

Question 4. The final question asked, are there identifiable pathways from birth parent psychopathology to adoptive parent depressive symptoms via infant negative emotionality, which would reflect evocative gene-environment correlations of children's genetic risk for psychopathology influencing adoptive parents' symptoms? It was hypothesized that children with a genetic liability for behavioral problems, as marked by the association between birth parent psychopathology and heightened infant emotionality, would evoke a depressive response from their adoptive parents. This hypothesis was tested using the product of indirect effects from birth mother psychopathology to infant negative emotionality to adoptive parents' depressive symptoms. Figure 3 provides path estimates for the model. Fit indices indicated good fit to the data (Robust CFI = .99; SRMR = .02; Robust RMSEA = .02). The model indicated that the indirect effects from

birth mother psychopathology to infant negative emotionality to adoptive mother depressive symptoms (std. coef. = $-.01$, $p = .28$) and adoptive father depressive symptoms (std. coef. = $-.01$, $p = .23$) were non-significant. Thus, this hypothesis was not supported.

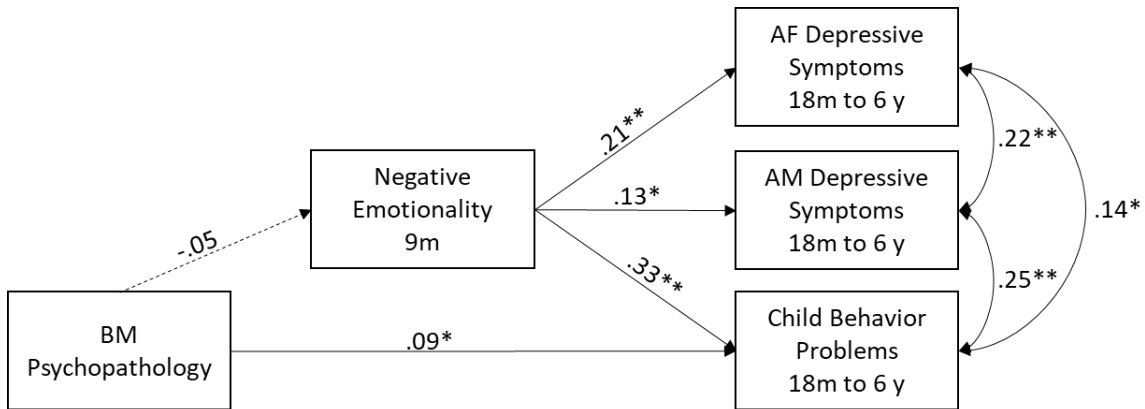


Figure 3. Indirect path from birth mother psychopathology to adoptive mother and father depressive symptoms. $*$ = $p < .05$; $**$ = $p < .01$.

CHAPTER V

V. DISCUSSION

I used an adoption design to examine the separate contributions of birth parent psychopathology, prenatal depressive symptoms, and adoptive parent depressive symptoms on child behavior problems and psychiatric disorders. Using an adoption design allowed for separate estimates for genetic risk for psychopathology and postnatal environmental exposure, since children did not share a rearing environment with their biological parent. Moreover, the use of RI-CLPMs controlled for the trait-like stability of child behavior problems and parent depressive symptoms, which allowed for unbiased estimates of the cross-lagged paths between parent depressive symptoms and child behavior problems. The use of two separate models to consider mothering and fathering separately, yet accounting for the other, allowed for a broader picture of the rearing environment to understand how mothers and fathers contributed uniquely to child behavior problems and psychiatric disorders.

Overall, results provided some support for general genetic risk for psychopathology and no evidence for prenatal depressive symptoms as a risk for child behavior problems or child psychiatric disorder. Additionally, there was no evidence for cross-lagged associations between parent depressive symptoms and child behavior problems but there was evidence for overall associations between the two constructs overtime. Negative emotionality was a clear predictor of both child behavior problems and parent depressive symptoms.

Genetic Transmission of Psychopathology to Child Behavior Problems

This study provides some support for a common “*p*” factor that captures a person’s liability to a mental health disorder (Selzam et al., 2018). Specifically, higher birth mother psychopathology was related to more child behavior problems. However, birth father psychopathology was unrelated to child behavior problems. The majority of birth father data was imputed when a general *p* factor was created (see methods section) because there was a reduced number of birth fathers who participated in the study. Thus, it is possible that variance explained by birth mother psychopathology explained most of the variance associated with birth father psychopathology for child behavior problems. Additionally, there were no differences on levels of birth mother psychopathology for those who had birth father data versus those who did not. Further, birth mother and father psychopathology were correlated, indicative of assortative mating. Correlations between birth mother and father psychopathology provides evidence that birth mother psychopathology potentially captured variance associated with birth father psychopathology for child behavior problems. Alternatively, there may be unique variance attributed to birth mother psychopathology for child behavior problems that is actually an unmeasured prenatal variable. Specifically, our data did not capture the full range of psychological experiences a woman may have experienced during pregnancy, thus, other unmeasured birth mother psychopathology experienced prenatally may have directly impacted the fetus in utero.

There was only one association of note between birth parent psychopathology and child psychiatric outcomes. Specifically, birth father psychopathology was associated

with child depression. However, this finding was not in the expected direction. Specifically, lower scores on birth father psychopathology were associated with a greater likelihood of a child having depression. Moreover, a significant association was not found when correlating birth father psychopathology and child depression but was only observed when the path was modelled with the structural equation model. The association in the model was small in magnitude and similar to other null effects for birth parent psychopathology on child psychiatric outcomes. Nevertheless, this suggests that independent variance in birth father psychopathology (i.e., variance separate from other variables in the model such as birth mother psychopathology), is negatively associated with child DD. It was also surprising that there were no other significant associations between birth parent psychopathology and child psychiatric outcomes. In order to identify clinically meaningful data, child psychiatric symptoms were coded as presence/absence of disorder rather than as symptoms of disorder in the current study. Using dichotomous rather than continuous variables for child psychiatric outcomes reduced power to detect an association (Altman & Royston, 2006). Additionally, since birth parent psychopathology puts children at-risk for general child behavior problems, problematic environmental exposures such as early life stress (Klengel & Binder, 2015), may be part of what increase a child's likelihood of experiencing the manifestation of any given psychiatric disorder.

Absence of Risk Conferred by Prenatal Depressive Symptoms

As hypothesized, birth mother prenatal depressive symptoms were unrelated to child behavior problems, child, child depression, child ADHD, and child CD/ODD. No

significant correlations were detected, prior to accounting for adoptive parent depressive symptoms, nor were there associations in the RI-CLPMs. Prior research has detected associations between prenatal depressive symptoms and child behavior problems and psychiatric outcomes, but only for children who were biologically related to their rearing parent (Allen et al., 1998; Lahti et al., 2017; Luoma et al., 2001; Mclean et al., 2018; Wolford et al., 2017). In instances where children were related to their rearing parent but familial confounding was controlled for, significant associations were no longer detected (Gjerde et al., 2017). Thus, this study provides additional evidence that prenatal depressive symptoms alone may not be a salient risk factor for later child behavior problems and psychiatric disorders. Rather, in studies with children who are biologically related to their rearing parent, measures of prenatal depressive symptoms may be capturing variance from genetic influences (Marceau et al., 2019) or other environmental influences such as father depressive symptoms, which may predict both prenatal depressive symptoms and child behavior problems and child psychiatric disorder (Chang et al., 2007; Letourneau et al., 2019). However, limitations in the study's ability to capture prenatal depressive symptoms may have also contributed to a lack of association between prenatal depressive symptoms and child behavior problems and psychiatric outcomes. Namely, prenatal depressive symptoms were reported by biological mothers retrospectively. Moreover, only a short form of the BDI was administered to biological mothers rather than the full version. The full BDI may have captured aspects of prenatal depressive symptoms that were not reflected by the short version of the measure. Additionally, other work using this same adoption study has found that prenatal exposure

to biological mother depressive symptoms is indirectly related to child internalizing behaviors, mediated by child reduced cortisol (Laurent, Leve, Neiderhiser, Natsuaki, Shaw, Harold, et al., 2013). This suggests that prenatal depressive symptoms may be specific to child internalizing behavior, which was outside of the scope of the current study.

Infant Negative Emotionality and Child Behavior Problems as Indicators of Risk

I hypothesized that chronic child behavior problems from child age 18 months to 6 years would predict each psychiatric disorder. Results fully supported this hypothesis, such that higher scores on child behavior problems predicted each child psychiatric disorder. The association between child behavior problems and child DD was the lowest (approximately .23), with larger associations for child ADHD (approximately .32), child anxiety (approximately .33), and child ODD/CD (approximately .53). Thus, chronic behavior problems, as measured by the CBCL, were a good indicator general risk for each child psychiatric disorder. Additionally, infant negative emotionality at 9 months of age predicted child behavior problems but not child psychiatric disorders. This was also the case when examining bivariate correlations, except for a correlation between infant negative emotionality and CD/ODD that was small in magnitude, which suggests that a lack of association between infant negative emotionality and child psychiatric disorders is not because of the association between child behavior problems and child psychiatric disorders. These results suggest that it is unlikely that infant negative emotionality is directly related to child psychiatric disorders. However, measuring child psychiatric disorders as presence/absence rather than as continuous symptoms counts limited the

amount of variance that could be explained. Moreover, the lack of association between negative emotionality and child psychiatric disorders does not discount the possibility of negative emotionality as an underlying indicator of p (Tackett et al., 2013). Rather, it may be that negative emotionality explains general liability for any disorder, rather than the presence or absence of a single disorder.

Postnatal Depressive Symptoms as an Environmental Risk

In line with hypotheses, chronic adoptive mother and father depressive symptoms covaried with chronic behavior problems; the standardized association was medium in magnitude. These findings echo findings from previous work. Specifically, mother depressive symptoms, considered as a composite, are typically associated with child behavior problems (Grabow et al., 2017; Netsi et al., 2018; Pemberton et al., 2010), suggesting that mothers with chronic rather than acute symptoms should be prioritized for intervention. Additionally, this study finds a similar pattern of results for adoptive father depressive symptoms. While the association between father depressive symptoms and child behavior problems is smaller in magnitude, father depressive symptoms were still significantly associated with child behavior problems, even when controlling for the effect of adoptive mother depressive symptoms. There is little extant research examining father depressive symptoms on child behavior problems using genetically sensitive designs (Natsuaki et al., 2014). The findings from this study provide evidence that father's depressive symptoms are associated with children's behavior problems. However, mother's depressive symptoms predicted both child behavior problems and adoptive father depressive symptoms, whereas adoptive father depressive symptoms only

predicted child behavior problems. Recent findings have suggested that, while father's depressive symptoms are related to child behavior problems, the absence of father's depressive symptoms alone cannot compensate for the presence of maternal depressive symptoms (Pietikäinen et al., 2019). The findings from this study suggest that maternal depressive symptoms are likely a driver of both father's depressive symptoms and child behavior problems.

Although chronic adoptive mother and father depressive symptoms were related to child behavior problems, they did not predict child psychiatric disorders. These findings were surprising given prior work that has established associations between mother and father depressive symptoms and child psychopathology (Goodman et al., 2011; Harold et al., 2011). However, the current study is one of few studies to specifically examine associations between adoptive mother and father depressive symptoms and child clinical outcomes using an adoption design aside from Tully et al. (2008). Tully et al. examined associations between parent depressive symptoms and adolescent psychiatric outcomes, rather than child psychiatric outcomes. It may be that parent depressive symptoms measured as a continuous variable are unrelated to clinical cut-offs for child psychiatric outcomes, but that child psychiatric symptoms measured continuously may be associated with parent depressive symptoms. Additionally, it may be that behavior problems are somewhat stable over the course of early childhood, thus, by adding child behavior problems as a predictor of child psychiatric disorders, the variance that may have been attributed to parent depressive symptoms was already accounted for. Of note, prior to estimating the full model, there were several correlations

between maternal depressive symptoms across early childhood and child depression and anxiety, suggesting the possibility of direct environmental transmission of internalizing behavior.

I next examined time specific associations between parent depressive symptoms and child behavior problems. It was hypothesized that there would be a significant cross-lagged association between parent depressive symptoms at nine months to child behavioral problems at 18 months for both father and mother depressive symptoms, whereas other cross-lagged associations between parent depressive symptoms and child behavioral problems would be non-significant. Results indicated that all cross-lagged associations from adoptive mother and father depressive symptoms to child behavior problems were non-significant. It was surprising that there were no cross-lagged associations from parent depressive symptoms in infancy to child behavior problems in toddlerhood since prior research has found that early depressive symptoms from mothers and fathers are most often related to child behavior problems (Pratt et al., 2017; Verbeek et al., 2012). However, toddlerhood is marked by escalations in child behavior problems which tend to plateau over the course of the preschool years (Braungart-Rieker et al., 2010; Hernández et al., 2018; Murphy et al., 1999; Sallquist et al., 2009). It may be that maternal depressive symptoms in infancy are not related to child behavior problems in toddlerhood, but instead are related to behavior problems after toddlerhood, specifically during the preschool years and at school age when behavior problems are more stable. Additionally, 9 months may be too late for the examination of the role of early parent depressive symptoms. At the time of birth, infants begin to understand the nature of their

caregiving environment, by approximately 2 months of age, they begin smiling, and by 4 months of age they are able to time their smiles to elicit a social response from their mother (Ruvolo et al., 2015). It has been demonstrated that mothers experiencing heightened depressive symptoms are more likely to display blunted sensitivity to infant cues (Muzik et al., 2017; Newland et al., 2016). Experimentally, differential patterns of interactions emerge between infants whose mothers are depressed and other adults as early as 3 months of age (Field et al., 1988). Thus, an examination of parent depression prior to 9 months using a genetically sensitive design and similar modelling approach is warranted.

While there were no cross-lagged associations, there were within-time correlations at 27 months and 6 years between adoptive mother depressive symptoms and child behavior problems. These within-dyad associations indicate that deviations from the person-specific mean in maternal depressive symptoms are accompanied by deviations in the person-specific mean in child behavior problems at 27 months and 6 years. Associations at 6 years were expected as there is research to suggest that child behavior problems in later development (childhood and adolescence) are likely to be accompanied by maternal depressive symptoms (Gjerde et al., 2017; Sellers et al., 2016). However, the association between 27 months maternal depressive symptoms and child behavior problems was notable. As previously discussed, behavior problems tend to peak between 2 to 3 years of age (Braungart-Rieker et al., 2010; deRegnier, 2017; Hernández et al., 2018; Murphy et al., 1999; Sallquist et al., 2009). During this time period, children are developing autonomy and learning how to leverage their growing capacity to express

language in a goal-directed manor (Andreadakis et al., 2019). In absence of their ability to express language, behavior problems are magnified (Putnam et al., 2006; Roberts et al., 2018). Thus, mother depressive symptoms may be heightened due to limitations in child language and greater problem behavior (Gueron-Sela et al., 2018). Additionally, maternal depressive symptoms may limit a child's ability to express language, leading to increased behavior problems in toddlerhood (Quevedo et al., 2012).

There were no time varying associations between adoptive father depressive symptoms and child behavior problems, suggesting there may be less variance among fathers in depressive symptoms over time compared to mothers. Indeed, prior work has identified that in infancy, father depressive symptoms may peak and return to baseline levels after the early postpartum period then remain somewhat stable (Volling et al., 2018), whereas maternal depressive symptoms exhibit a variety of trajectories over the course of a child's development such as low-stable, moderate-stable, moderate-increasing, and high-decreasing (Ahmed et al., 2019).

Finally, the possibility of an evocative effect of infant negative affect was examined since prior research and prior findings from this study suggested that dyadic interactions during infancy may be responsible for relations between parent depressive symptoms and child behavior problems over time (Leadbeater et al., 1996; Roben et al., 2015). It was hypothesized that there would be an indirect path from birth parent psychopathology through negative emotionality to adoptive parent depressive symptoms (i.e., an evocative effect). There was no evidence for genetically influenced evocative effects since birth mother psychopathology was unrelated to infant negative emotionality.

However, infant negative emotionality did predict adoptive mother and father depressive symptoms. This suggests early infant temperament may confer parent risk for depressive symptoms, but that negative emotionality is associated with early environmental influences rather than genetic influences. However, the indicator of birth parent psychopathology may not have adequately captured genetic liability for infant negative emotionality. Thus, studies using a genetically sensitive design are needed to examine predictors of infant negative emotionality.

Limitations and Future Directions

Limitations include measurement limitations and design limitations. Measurement limitations that exist include the timing of data collection and reporter bias. Specifically, the studies that exist examining parent depressive symptoms on child behavior problems typically measure depressive symptoms between three to six months postnatal (Gutierrez-Galve et al., 2019; Netsi et al., 2018). Typically, postnatal depression seems to be most predictive of child outcomes when measured earlier, however, this is using non-genetically sensitive designs. We were not able to measure the early postpartum period given the first time point of adoptive parent depressive symptoms was collected when children were nine months of age. Another limitation is that adoptive parents reported on their own depressive symptoms and their child's behavior problems. Use of the same reporter and the same method tends to inflate associations (i.e., shared method variance; LaGrange & A Cole, 2008). This was accounted for by using both parent's estimates of child behavior problems, but some bias may still exist since parents' depressive symptoms also influence each other (Volling et al., 2018). Additionally, there were

multiple tests conducted. Although all analyses were hypothesis driven, it could have been advantageous to correct for multiple testing to reduce the chance of type I error.

Some limitations exist within the adoption sample, itself. Birth parents may be at higher risk than the population norms, given that the majority had very low income and low educational attainment at each study period (Leve et al., 2013, 2019). Moreover, birth fathers were underrepresented in our sample, making genetic contributions from fathers difficult to estimate as there may not have been adequate power to detect significant associations. Additionally, adoptive parents typically had higher income and educational attainment than the general population at each study period, which may reduce their child's risk for behavior problems. Both higher income and higher parent educational attainment are related to improved outcomes for children (Hodgkinson et al., 2017; Huaqing Qi & Kaiser, 2003; Larson et al., 2015). Adoptive parents in our study also had lower prevalence of a major depressive episode (17% for adoptive mothers, 6% for adoptive fathers) compared to birth parents (37% for birth mothers, 26% for birth fathers) and compared to the general population (25% for the general female population, 18% for the general male population; *National Comorbidity Survey*, 2007), which makes it difficult to generalize results to clinical populations.

An additional limitation of this study is that only adoptive parent depressive symptoms were included. Of course, postnatal exposure to other parent psychopathology symptoms, such as adoptive parent anxiety and antisocial symptoms, are associated with child negative emotionality as well as behavior problems (Brooker et al., 2015; Kerr et al., 2013). Future work using genetically sensitive designs should examine cross-lag

associations between other aspects of postnatal exposure to other types of parent psychopathology.

Limitations notwithstanding, there are several key contributions from this study. Namely, this study was able to separate the influences of birth parent psychopathology, prenatal depressive symptoms, and environmental exposure to parent depressive symptoms. The trait-like stability in parent depressive symptoms and child behavior problems was accounted for and influences of both mothers and fathers were included in analyses. Findings indicated the presence of genetic transmission of child behavior problems, but not specific child psychiatric disorders. Additionally, parent depressive symptoms predicted child behavior problems but not psychiatric outcomes, which were consistently preceded by child behavior problems. Adoptive mother depressive symptoms appeared to be more associated with child behavior problems than the influences of adoptive father depressive symptoms. However, father depressive symptoms were also significantly associated with child behavior problems. Moreover, results suggested that child behavior problems and parent depressive symptoms are somewhat stable following infancy, and that the within-dyad variability of these constructs was limited to correlations between maternal depressive symptoms and child behavior problems when children were 27 months and 6 years. Early intervention to reduce the onset and chronicity of depressive symptoms is warranted for both mothers and fathers, in order to reduce the child's risk for developing behavior problems.

Interventions seeking to reduce the prevalence of behavioral problems and onset of psychiatric disorder must consider that there are genetically influenced pathways to

child behavior problems. A comprehensive assessment for children of biological risk for psychopathology may help to identify children who may need early intervention. Additionally, while this work cannot completely rule out the contributions of risk conferred by prenatal depressive symptoms, results suggests that less emphasis should be placed on prenatal symptoms as a risk factor than it should be placed on genetic risk and postnatal depressive symptoms. Instead, prenatal symptoms may be part of a broader picture of general risk conferred by parent psychopathology for child behavior problems and psychiatric disorder. The presence of infant negative emotionality at 9 months is an indicator that dyadic intervention is needed. Since infant negative emotionality was not predicated by birth parent psychopathology, it may be that early parenting puts increases infant risk of exhibiting heightened negative emotionality. Interventions that teach effective parenting strategies or improve parent mood may reduce the likelihood of infant expression of negative emotionality. Certainly, parenting programs exist that improve parenting skills among infants with negative emotionality (e.g., Bagner, Rodríguez, Blake, & Rosa-Olivares, 2013), however, there are currently no programs that exist which have specifically aimed to *prevent* infant negative emotionality. Existing prevention programs seeking to improve long term behavioral and psychological outcomes for children should measure infant negative emotionality to see if programs reduce the early development of heightened infant negative emotionality.

The chronicity of both mother and father depressive symptoms is related to chronic child behavior problems. Parenting interventions designed to improve parenting practices have been shown to improve both parent depressive symptoms and child

behavior problems (Beach et al., 2008; Shaw et al., 2009). Given the lack of clarity from this paper and a lack of clarity from the existing body of research on the directionality of the association between parent depressive symptoms and child behavior problems, preventative interventions should seek to ameliorate both challenges simultaneously in order to improve overall family outcomes. Moreover, it appears unlikely that intervening only on parents' depressive symptoms, at least after 9 months of age, would reduce the incidence of child psychiatric disorders. Rather, children must be screened for behavioral problems and provided with adequate supports, which may include parenting training, child skills training, and child cognitive behavioral therapy (Waddell et al., 2007).

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