PARENTAL DEPRESSIVE SYMPTOMS OVER TIME AND TRANSMISSION OF RISK TO ADOPTED TODDLERS’ EMOTION REGULATION

A Dissertation in
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by
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ABSTRACT

There are likely biological and environmental factors that account for the association between parental depression and emotion regulation difficulties of children. However, there are very few studies that have examined the mutual and distinct influences of genetic and rearing factors together. The current study sought to answer three questions: 1) what are the patterns of genetic and environmental risk conferred by parental depressive symptoms using timing, chronicity, and severity of birth and adoptive parents’ depressive symptoms, 2) how do genetic risk and environmental risk operate together to influence emotional exchanges in the parent-toddler interaction, and 3) are genetic and environmental risk to emotion regulation moderated by the nature of parent-child dyadic interaction? This study used toddler anger expression during a challenging task as an index of toddler emotion regulation. Data were from the prospective adoption study of 361 families, the Early Growth and Development Study, at child ages 9, 18, and 27 months. Findings indicated that both timing and chronicity of adoptive mothers’ depressive symptoms influenced toddler anger expression. Adoptive fathers’ symptoms did not influence children directly, but instead influenced mothers’ symptoms over time. These findings were in the presence of genetic risk, which operated through a perinatal risk factor, symptoms of preeclampsia. Moreover, these environmental and genetic patterns of risk were moderated by adoptive parent-toddler interaction, such that children’s anger expression was influenced by genetic and environmental risks, but only when the parent-toddler interaction had a negative quality. Taken together, these results offer new evidence on the intergenerational transmission of risk from parental depression, through a developmental psychopathology framework.
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Chapter 1. INTRODUCTION

Emotion regulation, conceptualized as the processes responsible for monitoring, evaluating, and modifying emotional reactions (Thompson, 1994), is a key component of socio-emotional competence and mental health (e.g., DelCarmen-Wiggins, 2008; Eisenberg & Fabes, 1992; Shonkoff & Phillips, 2000; Southam-Gerow & Kendall, 2000). The examination of the specific features of children’s emotion regulation is crucial for understanding child psychopathology, as evidence suggests that dysregulated emotional reactivity, both in response to negative events and as a temperamental trait, is linked to present or future psychopathology in children (Cole & Hall, 2008; Cole, Zahn-Waxler, Fox, Usher, & Welsh, 1996; Luby et al., 2006). Difficulties with regulating emotions are associated with both internalizing and externalizing symptoms in young children (Cole, Luby, & Sullivan, 2008; Eisenberg et al., 2001; Garber, Braafladt, & Weiss, 1995; Hill, Degnan, Calkins, & Keane, 2006; Martin, Boekamp, McConville, & Wheeler, 2010; Silk, Steinberg, & Morris, 2003). Research has examined multiple risk factors for compromised emotion regulation skills and development, among them parental depressive symptoms (e.g., Maughan, Cicchetti, Toth, & Rogosch, 2007; Silk, Shaw, Skuban, Oland, & Kovacs, 2006). Although typically developing children’s abilities to regulate emotions steadily progress throughout childhood, one study found a significantly flatter trajectory of growth in regulatory skills for children with mothers with high levels of depressive symptoms (Blandon, Calkins, Keane, & O’Brien, 2008). Examination of the intergenerational transmission of risk to emotion regulation during early childhood may be critical to understanding the pathways of influence from parental depressive symptoms. This study examined both potential genetic and environmental pathways of risk using an adoption design.
The toddler years are characterized by marked changes across multiple domains of development and by wide individual differences in the timing of when skills in these domains emerge (Brownell & Kopp, 2007). Emotion regulation changes significantly from infancy through toddlerhood, with the onset of self-initiation of regulatory strategies and attention control between the 2nd and 3rd birthdays (Calkins, Gill, Johnson, & Smith, 2001; Cole et al., 2011; Kopp, 1982; Rueda, Acosta, & Santonja, 2007; Ruff & Rothbart, 1996). Early childhood research highlights the distinctive importance of self-regulation and its prominent effects on development (e.g., Caspi, Henry, McGee, & Moffitt, 1995; Mischel, Shoda, & Peake, 1998), and similarly, research finds that difficulties with emotion regulation in toddlerhood and preschool lead to less optimal outcomes such as more aggression, less self-control, less peer acceptance, and more externalizing problems in kindergarten and first grade (Blandon, Calkins, & Keane, 2010; Gilliom, Shaw, Beck, Schonberg, & Lukon, 2002; Zahn-Waxler, Ianotti, Cummings, & Denham, 1990). Early deficits in emotion regulation may have lasting effects on later mental health and it may be especially important to study early predictors of toddler emotion regulation skills, when these skills are still developing and undergoing rapid and marked change.

In order to identify risk and intervene early in life, it is crucial to study early predictors of risk (Cicchetti & Cohen, 2006; Tolan & Dodge, 2005), including factors such as parental depressive symptoms that may contribute to child emotion regulation difficulties as early as toddlerhood. Depression, a serious and widespread mental disorder (Üstün, Ayuso-Mateos, Chatterji, Mathers, & Murray, 2004), may be uniquely challenging for parents of young children, given that caregivers of toddlers are likely to face challenges each day that require interest and energy and are aided by feeling a sense of efficacy and positive emotions (e.g., Dix, 1991; Teti & Gelfand, 1991). Interactions with toddlers, who are in a developmental period marked by high
emotional negativity (Bridges, 1932; Campbell, Spieler, Burchinal, & Poe, 2006; Tremblay & Nagin, 2005), may be especially challenging to mothers struggling to regulate their own negative emotions. Adult depression has been conceptualized in terms of deficits in emotion regulation (Davidson, Pizzagalli, & Nitschke, 2002; Gross & Muñoz, 1995; Joormann, Siemer, & Gotlib; Teasdale, 1988). Mothers who are depressed, and who thus may have deficits in emotion regulation, could feel more stressed by the demands in caring for their 15-month-old infants than mothers who had never been depressed (Cornish et al., 2006), and research finds that depression may interfere with the ability to sensitively parent (Cicchetti, Rogosch, & Toth, 1998; Goodman & Gotlib, 1999).

Associations between parental depression and child emotion regulation persist beyond toddlerhood as well. Preschool-age children of mothers with more depressive symptoms have dysregulated emotion patterns (Luby & Belden, 2006; Maughan et al., 2007) and may be less likely to engage in active distraction as a regulatory strategy in a frustrating task (Silk et al., 2006). One longitudinal study from ages 4 to 7 suggests that children of mothers with more depressive symptoms have a significantly flatter trajectory of growth in emotion regulation skills (Blandon et al., 2008), while another finds that postnatal depressive symptoms are related to more problems with anger and violence at age 11 (Hay, Pawlby, Angold, Harold, & Sharp, 2003). Moreover, the ability to generate positive emotion in the face of potential frustration may moderate the effects of maternal depression on child internalizing disorders (Silk, Shaw, Forbes, Lane, & Kovacs, 2006). These longitudinal studies highlight the importance of examining how the relations between parental depressive symptoms and child emotion regulation unfold over time; however, they do not focus on toddlerhood, when emotion regulation skills are rapidly developing and children are dependent on their parents for support and guidance. The
identification of risk in toddlerhood, which would provide more targeted sources of intervention, could be critical for preventing worsening problems for children as they develop.

Fathers also play a significant role in the development of psychopathology of offspring (Phares & Compas, 1992); significant correlations between paternal depression and child emotional and behavioral problems were found in 14 out of 19 identified studies (Phares, Duhig, & Watkins, 2002). Given the higher prevalence rates of depression in women, it is understandable that the majority of studies conducted with parents with depressive symptoms have examined depression in mothers only. Many studies including paternal depression are correlational in nature, although a meta-analysis of these studies suggests significant associations between paternal depression and many childhood psychopathologies and behavior problems (Kane & Garber, 2004). Multiple studies have shown that children with two depressed parents have an increased risk of behavior problems when compared to children with only one depressed parent (e.g., Brennan et al., 2002; Goodman, Brogan, Lynch, & Fielding, 1993; Weissman et al., 1984). Mother and father depressive symptoms are correlated (e.g., Horwitz, Roben, Neiderhiser, et al., in preparation; Cummings, Keller, & Davies, 2005), and the experience of coping with a partner’s depression can increase an individual’s own stress (Coyne, 1976), which may not be due to assortative mating alone (Joiner, 1994). While recent research highlights the importance of paternal depressive symptoms for both mother and child mental health and the importance of including fathers in research, little is known about the reasons for increased risk. One reason may be that a child who has two parents with depression may be at a greater risk for a genetic disposition to depression (Goodman & Gotlib, 1999). In addition, the childhood environment may increase risk such that a depressed father confers risk through parenting or through exacerbation of the mother’s depression. In support of this idea, one study found that high
paternal depressive symptoms exacerbated maternal depressive symptoms in infancy, but only in the presence of high child involvement (Mezulis, Hyde, & Clark, 2004). These findings highlight the need to study both mothers and fathers using methodologies that allow for examination of both genetic and environmental effects.

Intergenerational transmission of risk from parental depressive symptoms could occur through multiple pathways, through the interplay of genes, parenting, and environmental context that shift and change across development. Research has progressed in identifying specific parenting differences between parents with depression and those without, such as being less engaged and responsive and speaking less to their children (e.g., Bettes, 1988; Feng, Shaw, Skuban, & Lane, 2007; Rowe, Pan, & Ayoub, 2005; Shaw et al., 2006), yet is impossible to fully understand the environmental impact of these differences in studies that include only biologically-related families and in those that do not include both mothers and fathers. Using an adoption design, this dissertation is a first step toward identifying the relative influences of genetic and environmental effects of parental depressive symptoms in toddlerhood.

**Toddlerhood as Time of Risk**

This proposal focuses on a specific period in development that is hypothesized to be crucial in the development of emotion regulation: late infancy to toddlerhood. The toddler years are a time of neurodevelopmental integration of different domains of functioning (Johnson & Munakata, 2005), characterized by important developments in neurobiological, social, emotional, and cognitive domains that serve as a basis for future growth (Gunnar & Quevedo, 2007; Perez & Gauvain, 2007). It is also a period of increased autonomy seeking and parent-child negativity in typically developing children (Belsky, Woodworth, & Crnic, 1996; Crockenberg, 1985). Furthermore, it is a time of rapid change in emotion regulation development; for example,
there are improvements in toddlers’ abilities to forestall anger, express anger for shorter durations, and use self-initiated distractions during a challenge from 18 to 48 months (Cole et al., 2011). Due to the rapid developments in toddlerhood, it may be a period that is more sensitive to environmental risk. Therefore, it is essential to include the toddler period in studies of risk transmission. Because this period involves rapid neurodevelopmental change, it may also be an ideal period for intervention. Maladaptive connections between domains such as emotion and cognition during these years appear to lead to misattributions that underlie later aggression and socio-emotional problems (Crick & Dodge 1994; Izard, Fine, Mostow, Trentacosta, & Campbell, 2002; Rogosch, Cicchetti, & Aber, 1995). Most research on offspring of depressed parents focuses on infancy (e.g., Cohn & Tronick, 1987; Field et al., 1989), preschool-age or older (e.g. Feng, Shaw, Kovacs, Lane, O’Rourke, & Alarcon, 2008), or adolescence (e.g., Garber & Martin, 2002). It is only through developmental research that we can truly come to understand the transmission of risk (Goodman & Gotlib, 1999) and this dissertation will focus on development from 9 to 27 months.

Parental Depressive Symptoms and Child Psychopathology

    Research has established that a parent’s depressive symptoms are associated with problems in offspring. Offspring of depressed parents are at an increased risk for developing psychiatric disorders (Cummings & Davies, 1994; Goodman & Gotlib, 1999; Orvaschel, Walsh-Allis, & Ye, 1988; Radke-Yarrow, 1998; Weissman et al., 1987), even up to 10 and 20 years later (Nomura, Wickramaratne, Warner, Mufson, & Weissman, 2002; Pilowsky, Wickramaratne, Nomura, & Weissman, 2006). There is a lack of specificity in childhood disorders associated with maternal depression; in addition to depression it also predicts externalizing disorders, internalizing disorders other than depression, and reports of suicidal thoughts and behaviors
(Brennan, Hammen, Katz, & Le Brocque, 2002; Klimes-Dougan et al., 1999). For example, compared to a non-risk group, adolescent offspring of depressed parents had higher rates of current or past diagnoses of affective disorders (38% vs. 2%) and conduct disorder (15% vs. 0%; Beardslee, Schultz, & Selman, 1987). The lack of specificity in diagnosis outcome suggests that it is important to study the proximal effects of parental depression on developmental processes that later present as disorder. The early development of emotion regulation is such a candidate process, given its role in both internalizing disorders such as depression and anxiety and externalizing disorders such as conduct disorder (e.g., Cicchetti, Ackerman, & Izard, 1995; Chaplin & Cole, 2005; Gross & Muñoz, 1995; Keenan, 2006).

In addition to the lack of specificity in outcomes of children with depressed parents, not all children of depressed parents develop problems. This is another reason why it may be important to examine proximal influences on the developmental processes that underlie the development of psychiatric disorders. In one longitudinal study of offspring of depressed parents (therefore exposed to both genetic and environmental risk), 10% of the children did not present with internalizing or externalizing problems from age 2 1/2 to age 15 (Radke-Yarrow & Klimes-Dougan, 2002). Identifying the proximal influences, which may explain why some children whose parents are depressed are adversely affected but others appear not to be, should have valuable implications for preventive interventions. The goal of this dissertation is to study the effects of proximal aspects of parental depressive symptoms, such as emotion regulation, during early childhood.

**Child Emotion Regulation and Parental Depressive Symptoms**

It is established that emotion regulation is an essential aspect of a child’s overall well-being, social competence, and mental health (e.g., Cole, Michel, & Teti, 1994; Eisenberg &
Fabes, 1992; Shonkoff & Phillips, 2000). Regulatory strategies that influence emotional expression develop from infancy to preschool (Kopp, 1982). However, infant strategies mainly involve gaze aversion, self-soothing, and vocalizing (Rothbart, Ziaie, & O’Boyle, 1992), appear to be reactive or reflexive (Kopp, 1982), and are limited in their effectiveness (Buss & Goldsmith, 1998). During the third year, the age of outcome in this study, most children begin to self-initiate regulatory strategies without adult intervention (Kopp, 1989). That is, they engage in verbal support-seeking or distraction, strategies that are at least modestly effective (Calkins et al., 2001; Grolnick, Bridges, & Connell, 1996; Kopp, 1989; Mangelsdorf, Shapiro, & Marzolf, 1995; Rothbart, Ziaie, & O’Boyle, 1992). Recent research suggests that use of these strategies is associated with a decreased likelihood of becoming angry when goals are blocked, but on average, not until the fourth year (Cole et al., 2011; Gilliom et al., 2002).

At any age, children of depressed parents appear to have difficulties with emotion regulation. For example, infant researchers point to the early origins of emotion regulation in mother-infant interactions (e.g., Field, 1978, 1985, 1991), and interactions with a depressed parent in infancy can lead to behavioral and physiological disorganization through less attuned and synchronous interactions (see Field, 1994). Later in toddlerhood, as typically developing children begin to show more autonomy in initiating regulatory strategies that forestall or reduce angry reactions (Cole et al., 2011; Kopp, 1989), toddlers of depressed mothers (children between ages two and three years) show poorer regulation of sadness, more aggressiveness, less persistence during a challenge, lower levels of positive emotion, and lower levels of pride when compared to children in control conditions (Durbin et al., 2005; Feng, Shaw, Kovacs, Lane, O’Rourke, & Alarcon, 2008; Jennings & Abrew, 2004; Malik et al., 2007).
Externalizing symptoms, such as anger expression, show more continuity with later problem behavior than internalizing problem behavior from the toddler to school-age periods (Feng, Shaw, & Silk, 2008; Shaw, Gilliom, Ingoldsby, & Nagin, 2003). Therefore, in examining the joint and separate influences of genetic and environmental depressive risk on the emotion regulation capacity of adopted children, this study focused on how parental depressive symptoms influenced anger expression in 27-month-old children. Specifically, anger will be quantified using temporal variables that measure emotion regulation in a way that is often written about (Thompson, 1990, 1994), but rarely studied. It quantifies the temporal (latency, duration, frequency) aspects of emotions in addition to intensity. These temporal aspects of emotion have been written about as qualities of reactivity (e.g., Rothbart, Derryberry, & Hershey, 2000), yet Thompson (1990) has also described the temporal qualities as indices of regulation. Although emotion regulation strategies are also frequently utilized in the study of emotion regulation, this study analyzed only the temporal quality of anger because of physical restraints on the child in the anger-eliciting task that limited their ability to use a wide range of regulatory strategies.

Parent Emotions and Child Emotion Regulation

The literature describes several ways through which maternal depression could influence parenting. Depressive symptoms are associated with mothers being withdrawn and disengaged with their children and being intrusive and irritable (Cohn & Tronick, 1989, Goodman & Gotlib, 2002). Being withdrawn is more associated with displaying sad and flat emotion, whereas being intrusive is more associated with displaying anger towards their infants. Notably, there are also mothers with depression who regulate their emotions and are able to display positive emotions towards their infant (e.g., Feng et al., 2007). Infants as young as 3 months can detect these emotions in their parents (Weinberg & Tronick, 1998). The emotional difficulties of parents with
depression are important to understand when focusing on proximal influences that disrupt the development of children’s self-regulation of emotion because parental emotions organize parenting behavior (Dix, 1991). Some research finds that depressed mothers are generally more emotionally negative than non-depressed mothers and this has been observed in their interactions with their young children (Campbell, Cohn, Flanagan, Popper, & Meyers, 1992; Cohn, Matias, Tronick, Connell, & Lyons-Ruth, 1986; Cohn & Tronick, 1987; Downey & Coyne, 1990; Radke-Yarrow, Nottelmann, Belmont, & Welsh, 1993). Compared to non-depressed mothers, they can appear more irritable and angry (Cummings, Waxler, Radke-Yarrow, 1984; NICHD Early Child Care Research Network, 1999), and less happy or joyful (Cohn & Tronick, 1987; Hops et al., 1987). Stresses that co-occur with depression, such as family-related stress (Billings & Moos, 1983; Fendrich, Warner, & Weissman, 1990; Hammen, Burge, & Adrian, 1991) and marital conflict (Fergusson, Horwood, & Lynskey, 1995), also influence maternal emotion. These facts logically lead to the importance of examining maternal emotion during interactions with young children as a proximal factor in the transmission of risk to offspring at risk for depression.

Research suggests that children are influenced by maternal negative emotion across development. For example, in infancy maternal negative emotion diminishes infant emotion and interest in communicating (Field, 1995). Maternal negative emotion predicts dysregulated emotion patterns in early childhood, which negatively impact children’s social acceptance by peers (Maughan, Cicchetti, Toth, & Rogosch, 2007). Moreover, contingent negative emotion to preschool-age children’s frustration appears to exacerbate their level of externalizing behavior problems (Cole, Teti, & Zahn-Waxler, 2003), and increases 7- to 12-year-old children’s impulsive behavior (Elgar, Waschbusch, McGrath, Stewart, & Curtis, 2004). The negative emotions expressed during parent-child interaction in early childhood are a logical place to study
the proximal influences of depression on children’s emerging self-regulation of anger. Therefore, the present study includes observations of parent-toddler interaction to estimate the degree to which negative emotion in those interactions moderates the association between parental depression and toddler anger reactivity during a frustrating situation.

**Parent-Child Dyadic Interactions**

Caregivers play a critical role in their children’s emotional development, entering into countless dyadic emotional exchanges with their children (Cole, Martin, & Dennis, 1994; Kopp, 1989). Infants have limited innate capacities to regulate their own emotions and are dependent on caregivers to effectively repair distress (Tronick, 1989). Indeed, infants are highly attuned and responsive to their parents’ emotions and use parental emotional signals to guide their behavior (Klinnert, Campos, Sorce, Emde, & Svejda, 1983; Lelwica & Haviland, 1987; Malatesta & Izard, 1984). Emotion communication between infants and their parents allows for mutually coordinated interactions that are bidirectional (e.g., Tronick, 1989).

Researchers hypothesize that mutual interactions between parents and infants may consolidate infant emotion regulation skills (Panksepp, 2001), and research does find links between putative biological rhythms indicative of regulation in 3-month-olds and mother-infant synchrony (Feldman, 2006; Moore & Calkins, 2004). Moreover, affect synchrony and contingent responsiveness in infancy predict better self-regulation in toddlerhood (Feldman & Eidelman, 2004; Feldman & Greenbaum, 1997; Shaw et al., 1994, 1998). As children grow older and as they develop other forms of communication such as language, they communicate their needs to their parents through both verbal and nonverbal expressions, and still need parental support in the service of developing emotion regulation skills (Cole, Armstrong, & Pemberton, 2010; Kopp, 1989).
Research finds parent-child dyadic interactions between depressed parents and their children to be less optimal than in families without depression. Dyads with a depressed mother have more mutual negative emotion than dyads without a depressed mother (Field et al., 1990) and have more trouble maintaining joint attention (Goldsmith & Rogoff, 1997; Henderson & Jennings, 2003; Jameson, Gelfand, Kulcsar, & Teti, 1997). Depressed mothers are less accurate at interpreting their babies’ facial expressions, and this accuracy is associated with interaction quality (Broth, Goodman, Hall, & Raynor, 2004). This finding may explain why depressed mothers’ responses are less contingent, attuned, and synchronized with infant signals (Cohn & Tronick, 1989; Field et al., 1989; Gelfand & Teti, 1990). Depression is also associated with less infant-directed speech and less talk to children in general (Bettes, 1988; Rowe, Pan, & Ayoub, 2005). It is possible that depressive symptoms alter perceptions of infant cues, and altered perception could then explain some of the parenting differences between depressed and non-depressed parents. In fact, one study found that as mothers’ levels of depression increased, mothers rated infant cries as less arousing, urgent, aversive, and sick sounding (Schuetze & Zeskind, 2001). Such perception differences could explain some of the mechanisms between parental depressive symptoms and impaired parenting skills.

Parenting of older children is influenced by parental depression as well. In preschool-age children, mothers with a current depressive diagnosis were less responsive to children’s positive emotions (Feng, Shaw, Skuban, & Lane, 2007), and were less responsive to their children’s emotional states generally (Shaw et al., 2006). In one study, mothers with depression were less able to sustain social interaction with their children and were more likely to disengage and ask fewer questions of their children (Cox, Puckering, Pound, & Mills, 1987), an interaction style that mediated the relation between maternal depression and child behavior problems. Together,
these findings suggest that parents with more depressive symptoms, who are likely to have more negative emotions, are also more likely to have more problematic dyadic interactions with their infants and young children, and it may be within parent-child interactions that risk is transmitted.

Children who lack parental help in responding to emotional situations could lack opportunities for learning to regulate emotions on their own. Children may further contribute to the problematic interaction by being unresponsive or defiant to the parent, leading to further negative emotion in the interaction. For example, one recent study found that there were stronger relations between parent depressive symptoms and preschool-age child frequency of crying in families reporting higher global levels of parent-child conflict (Slatcher & Trentacosta, 2011).

Child risk factors may also contribute to the interaction (Cole, 2003). However, it is difficult to understand the child’s contributions to this interaction in studies of biologically-related families. In biologically related families, it is impossible to tell whether children’s patterns of responses are due to shared genes with the parent, or due to risk from the environment. Parental emotion is correlated with child emotion but the relative genetic and environmental contributions to this association are unclear (Radke-Yarrow, Nottelmann, Belmont, & Welsh, 1993). In one genetically-informed study with families in middle childhood no effects of passive gene-environment correlation were found, and instead effects consistent with bi-directional socialization between parent and child were evident (Deater-Deckard & Petrill, 2004).

Genetically-informed studies, such as adoption designs, can aid in understanding the child-driven contributions to the interaction and those contributions more likely associated with the early rearing environment.

Similar to other approaches introduced for the intensive study of parent-child interactions (e.g., Dumas, LaFreniere, & Serketich, 1995; Kochanska, 1997), we utilized microanalytic
coding instead of more global measures of parent-child conflict. Global measures would be unsuited to teasing apart parent and child contributions to the interaction. For example, if a child expressed negative affect from the start to the end of the task, a global measure would be likely to rate the entire interaction as negative. The method used in the current study coded each second of the interaction, and determined to what degree one partner’s behaviors were related to the other’s and accounted for within-subject continuity. In the example above, because the child’s behavior can be fully accounted for by the child’s own behavior in the previous second, our measure would not indicate that the parent had caused the child to be more negative. Thus, a child factor (being more generally fussy) would not be mistaken as a parent-child dyadic factor. We hypothesized that the microanalytic dyadic measures would moderate the level of risk transmitted to children through both environmental and genetic pathways.

**Timing of Depressive Symptoms**

Goodman and Gotlib (1999) assert that the earlier a child is exposed to parental depression, the more detrimental that depression will be to the child. The timing, chronicity, and severity of parental depression must be considered when measuring the effects of parental depression on child outcomes. There is some evidence suggesting that chronicity leads to poorer outcomes than single episodes (Sameroff, Seifer, Zax, & Barocas, 1987) and that severity and chronicity are both important factors contributing to maladaptive outcomes for children (Brennan et al., 2000; Hammen & Brennan, 2003); however, there is limited research on how the trajectory of timing and severity of parental depression over time influences child outcomes.

Current empirical research is mixed as to whether contemporaneous or earlier depression influences children most. For example, previous work has found differential effects of early and remitting parental depression versus current-only depression (Alpern & Lyons-Ruth, 2003).
However, a meta-analysis found that current depression is associated with the most negative child outcomes (Lovejoy, Graczyk, O’Hare, & Neuman, 2000), while some studies find no effect of timing on child psychopathology (Hammen & Brennan, 2003). Goodman and Gotlib (1999) assert that the earlier a child is exposed to parental depression, the more serious the effects of depression on the child will be. There is crucial development of inhibitory systems and the HPA system during infancy (Dawson, 1994; Porges et al., 1994; Stansbury & Gunnar, 1994) and earlier detriments will have a greater effect on a child’s developmental trajectory (Goodman & Gotlib, 1999; Hay, 1997). There is also evidence that earlier onset of depression is associated with more severe and more frequent episodes over time (Harrington, Fudge, Rutter, Pickles & Hill, 1990; Kasen et al., 2001; Pine, Cohen, Cohen, & Brook, 1999; Wickramaratne & Weissman, 1998). Given clear evidence of the heritability of depression (e.g. Kendler et al, 1994; Sullivan, Neale, & Kendler, 2000) and of the importance of the child-rearing environment on children’s emotional development (e.g., Davis & Windle, 1997; Eisenberg, Cumberland, & Spinrad, 1998; Rutter, 2000), it is essential to understand these influences in terms of the timing, chronicity, and severity of parental depression in a child’s development. This complex appreciation of transmission of risk is essential for advancing our understanding of how risk is conferred. The present study attempts to address this need by using longitudinal data and information from both birth and adoptive parents to examine how risk may be transmitted to the child over the developmental period ranging from child age 9 to 27 months of age.

Given that depression is often chronic (Weissman et al., 1999), longitudinal studies that account for both prior and current depressive symptoms are necessary for examining the timing of depression. One method of accounting for the chronicity of depression uses latent growth curve modeling to demonstrate that that chronic parental depressive symptoms are more
problematic than decreasing or mild depression (Ashman, Dawson, & Panagiotides, 2008; Gross, Shaw, Burwell, & Nagin, 2009); however, this method does not measure the relative importance of depressive symptoms at each observation. Therefore, the present study examined the both the timing and chronicity of depressive symptoms in parents of children from infancy to early toddlerhood, a period of rapid change and continuing dependence on parental responsivity. In a previous study using the current data set, after accounting for across-time continuity and a significant genetic effect, paternal depressive symptoms at 9 months were associated with child behavior problems at 27 months (Pemberton et al., 2010). However, maternal depressive symptoms were associated with child behavior problems regardless of the timing, suggesting that the timing of risk from parental depressive symptoms to behavior problems may differ between parents when considering multiple sources of risk. These findings emphasize how it is important to go beyond the question of whether parental depressive symptoms influence children, to more specific questions such as when and how depressive symptoms could influence child outcomes over time. This dissertation will focus on understanding how early (9 and 18 months) and concurrent risk (27 months) factors influence emotion regulation at 27 months, an age when children’s skills are rapidly changing and are important for later development. In addition to increasing the specificity of our knowledge about transmission of risk, identifying ages of young children’s vulnerability to parental depressive symptoms can contribute to targeting appropriate age groups for preventive interventions that may mitigate the presence of risk for young children.

In the case of being an adoptive parent, there may be experiences specific to the adoption process that factor in understanding parental depression and risk to the adopted child. Although there is only a small body of relevant research, it suggests that post-adoption depression in parents is relatively common (McKay, Ross, & Goldberg, 2010). However, there is some
evidence that becoming an adoptive parent may be relatively less stressful than the adjustment to biological parenthood (Ceballo, Lansford, Abbey, & Steward, 2004). Nonetheless, there are unique challenges to becoming an adoptive parent, such as the perception of how others judge the fact that one has adopted a child, uncertainty about the timing of bringing a child into the family, and often the fact that parents had experienced the stresses of infertility (Brodzinsky & Huffman, 1988; Ceballo et al., 2004). If infertility was a contributing reason for adoption, it is possible that adoptive parents experienced depression prior to adoption (Berg & Wilson, 1991; Brodzinsky & Huffman, 1988). Another consideration is that the success of an adoption could reduce the stresses that had been associated with infertility, because having a child leads to more feelings of well-being among adoptive parents who had infertility problems compared to those who had not (Abbey, Andrews, & Halman, 1994). Also, adoptive parents may have fewer adjustment problems if they have a greater appreciation of their parenting status and satisfaction with their parental role after an often arduous adoption process (Levy-Shiff, Goldshmidt, & Har-Even, 1991). In addition, a reason for improved well-being may result from the fact that adoptive parents are often older than biological parents (Brodzinsky & Huffman, 1988). It is not known whether or how any of these conditions relate to the predictors or time course of depression in adoptive parents as their children age, although these could have important implications for family adjustment and welfare as well as the child’s development.

**Genetic Risk to the Offspring of Depressed Parents**

Children placed for adoption at birth or soon after share only genes (and prenatal environment for birth mothers) with their birth parents. Thus, associations between adopted child and birth parent characteristics can be used to estimate genetic contributions to the child’s characteristics. Adoptive parents are often genetically unrelated to the adopted child, and thus
associations between most adoptive parents and their child cannot be the result of shared genetic influences between parent and child. Associations could be the result of evocative genotype-environment correlation (rGE), because individual differences in children evoke different responses from their environments. For example, a fussy infant who is typically fussy may evoke a type of parenting that differs from that of an infant who is genetically predisposed to be calm. Associations could occur as a direct result of the environment (E), for example, when a parent’s over reactive parenting style causes a child to develop fussiness.

Depression is heritable (Lesch, 2004); a meta-analysis using rigorously designed twin studies estimated the genetic effect on major depression to account for 36% of the variance in offspring’s depression (Sullivan et al., 2000). However, as previously noted, offspring of depressed parents are also at risk for other disorders and many children do not develop psychopathology. Given the heterogeneity of these outcomes associated with exposure to parental depression, it is reasonable to hypothesize that the indicators of genetic risk appear in more proximal phenotypic behavior, such as poor emotion regulation skills, and negative parent-child dyadic exchanges.

Genetically-informed research has also found evidence for genetic influences on constructs typically thought of as environmental, like parenting (Feinberg, Neiderhiser, Howe, & Hetherington, 2001; Neiderhiser et al., 2004; O’Connor, Deater-Deckard, Fulker, Rutter, & Plomin, 1998). This has been described as evidence of genotype-environment correlation (rGE). A few studies went further by distinguishing passive rGE – the result of parents and children sharing genes and environment – from evocative rGE – the result of children evoking responses from their environment (Narusyte et al., 2008; Neiderhiser et al., 2004; Neiderhiser et al., 2007). An adoption design is ideal for specifying evocative rGE in the case of transmission of risk from
a depressed parent to the toddler learning to regulate anger and child effects in the parent-child dyadic interaction because the rearing environment is provided by adoptive parents who are genetically unrelated to the adopted child. The approach takes into account genetic influences on the child’s anger reactivity and permits assessment of whether the more angry toddler elicits more negative parenting from the adoptive parent or whether the adoptive parent’s negativity (due to depression, for example) has a direct influence on the child’s ability to regulate anger.

Among many effects, genes influence children’s emotional expression (Van Hulle et al., 2007). Moreover, parental responses often depend on child emotion, as research suggests evidence of both parent-to-child and child-to-parent effects (Cole, Teti, & Zahn-Waxler, 2003; Dumas et al., 1995; Forbes et al., 2008; Hammen, Burge, & Stansbury, 1990). Of particular relevance to the current study, we find evidence of child contributions to maternal negative emotions in early childhood. Maternal negative emotion can be contingent on child negative emotion, and specifically contingent on child anger rather than disruptive behaviors (Cole, LeDonne, & Tan, under review; Lorber & Slep, 2005). The contribution of the child’s genetic risk (i.e., greater propensity to react angrily) to the parent-child interaction (i.e., more negative exchanges elicited by child anger reactivity) would be evidence of evocative gene-environment correlation (rGE). In addition to accounting for heritable characteristics of anger expression in children, the present study’s design also permitted tests of the contribution of parental behavior to the interaction. The observational methods available in the dataset used for the present study did not allow specific evaluation of parent emotion. However, because parental emotion organizes parental behavior (Dix, 1991) and because maternal self-reported emotions influence their parenting sensitivity (Martin, Clements, & Crnic, 2002), it is assumed that parental behaviors capture an aspect of parental emotion, albeit indirectly. The current study analyzed
parent and child behaviors in the parent-child interaction, motivated by the research that emphasizes the contributions of emotion to that interaction.

Several studies using genetically-informed designs have demonstrated both genetic and environmental influences of parental depression on child functioning. For example, family conflict is a stronger predictor of youth depression in those individuals who had an elevated genetic risk for depression (Rice, Harold, & Thaper, 2002). Few adoption studies have examined the genetic and environmental influence of parental depression on child outcomes. One exception is a study of adoptees that used retrospective reports and records of a history of treatment for depression and/or mania (Cadoret, O’Gorman, Heywood, & Troughton, 1985). The authors found a positive trend of an influence of birth parent depression on young adulthood depression but, as one meta-analysis pointed out (Sullivan et al., 2000), this study was underpowered and split the results by gender of the adoptee. When Sullivan and colleagues combined the adoptees into one analytic sample, they found a significant genetic effect. In another study, adoptive mother, but not adoptive father, depression elevated adolescent risk of psychopathology (Tully, Iacono, & McGue, 2008). This study found no role of genetics in the transmission of risk from parent symptoms to child psychopathology. No studies to date have used such a design with children in early childhood. The adoption design is also well suited for examining gene-environment interaction (GxE). Using data from the current study, findings have indicated that adoptive parents’ anxious and depressive symptoms play a critical role in amplifying genetic risk for child problems (Leve et al., 2010; Natsuaki, 2010). The current study also examined GxE, but instead of using parental depression as the moderating factor, the parent-child interaction was examined as a mechanism of risk; the quality of the interaction could have potential buffering or exacerbating effects on both genetic and environmental risk.
Previous work with these data has also used the adoption design and intensive measurement of the prenatal environment to estimate influences of prenatal risk factors. One study found that birth mother depressive symptoms did not predict parent-reported child externalizing symptoms at 27 months directly, but did have an indirect effect on child externalizing symptoms through pregnancy risk factors such as frequency of prenatal care (Pemberton et al., 2010). Another study similarly found an indirect effect of birth mother depressive symptoms on parent-reported behavior problems through symptoms of preeclampsia (Hajal, Marceau, Neiderhiser, & Reiss, 2011). It is possible that the current study’s estimates of genetic influences could operate indirectly through prenatal risk factors as well.

While the literature suggests that the timing, severity, and chronicity of parental depressive symptoms are important (e.g. Ashman et al., 2008; Gross et al., 2008; Goodman & Gotlib, 1999), few studies examine these multiple aspects using longitudinal data, and none do so with a genetically-informed design in toddlerhood. The current study includes longitudinal data and a design with which we can understand gene-environment interplay, which could increase the understanding of pathways of risk. It is important to examine predictors of risk during these stages of early and rapid development in order to provide optimal opportunities for intervention (Cicchetti & Cohen, 2006; Tolan & Dodge, 2005). This study will examine the role of the dyadic parent-child interaction for the timing, pathways, and relative influences of adoptive and birth parent depressive symptoms on child emotion regulation at 27 months of age.

**Proposed Investigation**

Participants are from a longitudinal, prospective adoption study (Early Growth and Development Study, EGDS; Leve et al., 2007; Leve et al., 2008). This current study adds to examined effects of adoptive parental depressive symptoms across time, when the child was 9,
18, and 27 months of age, on 27-month-old child emotion regulation. We also included multiple 
assessments of biological parent depression and observed indicators of the parent-child 
interaction at 18 months and child emotion regulation at 27 months. EGDS has the advantage of 
both detailed behavioral observations and the ability to examine the gene-environment interplay 
longitudinally.

The specific aims examined in this study (see Figure 1) were:

1. what are the patterns of genetic and environmental risk conferred by parental depressive 
symptoms using timing, chronicity, and severity of birth and adoptive parents’ depressive 
symptoms,
2. how do genetic risk and environmental risk operate together to influence negative 
emotion in parent-toddler interaction, and
3. does the quality of the adoptive parent-child dyadic interaction moderate risk associated 
with either the presence of a depressed adoptive parent (ExE or ExrGE), or of a 
biological parent (GxE or Gx rGE), on child emotion regulation?

*Figure 1. Model of transmission of risk to child emotion regulation.*
**Aim 1.** There is evidence that both early and concurrent parental depressive symptoms influence child outcomes, and that chronicity and severity of those symptoms predict child outcomes (Ashman et al., 2008; see Goodman & Gotlib, 2002; Hammen & Brennan, 2003). Moreover, preliminary findings from EGDS find that even after accounting for across-time continuity of depressive symptoms, the timing of parental depressive symptoms predicted toddler problem behaviors and was different for mothers and fathers (Pemberton et al., 2010). Therefore, it is predicted that the timing of parental depressive symptoms will be an important predictor of toddler emotion regulation. It is expected that chronicity of both birth and adoptive parents’ depressive symptoms will be associated with poorer anger regulation at child age 27 months. Further, it expected that fathers’ depressive symptoms will influence toddler emotion regulation, but the effects will not be as great as mothers’ depressive symptoms.

Specifically, continuous ratings of parental depressive symptoms measured when the child is 9, 18, and 27 months of age will be used to assess multiple models of depressive symptoms as predictors of toddler emotion regulation at 27 months. Emotion regulation will be measured using temporal variables that measure emotion regulation in a way that is often written about (Thompson, 1994), but rarely studied. It quantifies the temporal (latency, duration, frequency) aspects of emotions in addition to intensity. Emotion regulation will therefore be indexed by a latent variable including the duration, latency, and intensity of anger expressed during an anger-eliciting task, the arm restraint.

**Aim 2.** A transactional approach to studying social interactions is regarded as the best way to conceptualize behavioral processes that contribute to the development of psychopathology (Olson & Sameroff, 2009). At present, studies of dyadic processes in child development are limited; those studies that examine dyadic exchanges find that mutual positive
emotion and contingent responsiveness in infancy are associated with better compliance and self-regulation, better social-emotional adjustment, and fewer behavior problems in toddlers (Feldman & Greenbaum, 1997; Feldman & Eidelman, 2004; Shaw et al., 1994, 1998). Maternal depression, however, is associated with reciprocal negative emotion in parent-young child dyads (Field et al., 1990). Moreover, children with a genetic susceptibility to emotion regulation problems (i.e., those with at least one depressed birth parent) may be less likely to respond to their adoptive parents in a transactional manner.

Therefore, it is expected that parental depressive symptoms will predict the responsive qualities of the parent-child dyadic interaction. It is predicted that both genetic risk (biological parents’ depression) and environmental risk (adoptive parents’ depression) will predict the quality of the parent-child dyadic interaction. If genetic risk does predict the quality of the parent-child interaction (rGE), then the third aim can detect the influence of the interaction on environmental risk (Ex rGE) and on genetic risk (Gx rGE). If, on the other hand, there is no evidence of evocative rGE, the parent-child dyadic interactions can be described as environmental in nature.

Specifically, continuous measures of both adoptive and birth parent depressive symptoms will be tested as predictors of responsive qualities of the parent-child dyadic interaction during a teaching task when the child is 18 months old.

**Aim 3.** The last aim will examine pathways of risk influenced by the dyadic interaction. Research suggests that the dyadic interaction is important for child self-regulation in toddlerhood (Feldman & Eidelman, 2004; Feldman & Greenbaum, 1997; Shaw et al., 1994, 1998). The design of EGDS is a strength of this study because with this design both GxE or Gx rGE and Ex rGE or ExE hypotheses can be examined. Furthermore, the role of evocative genotype-
environment correlation (rGE; parental response to genetically influenced characteristics in the child) in the parent-child dyadic interactions can be considered, thus specifying whether moderation paths include interactions with environmental risk alone, or with environmental risk in response to the child’s genetics. It is expected that the parent-child dyadic interaction will moderate the effects of genetic risk on later emotion regulation, such that emotion regulation of children with less optimal parent-child interactions will be more negatively influenced by a genetic vulnerability from birth mother depressive symptoms. Similarly, it is expected that the emotion regulation of children with less optimal parent-child interactions will be more negatively influenced by living in an environment with adoptive parents who are higher in depressive symptoms. The examination of moderating transactional processes within a behavioral genetic framework can identify what is important for children’s emotion regulation development, and for whom it is important.

Specifically, responsive qualities of the parent-child interaction in the teaching task will be tested as moderators of the relationship between adoptive parent depressive symptoms and toddler emotion regulation and between birth parent depressive symptoms and toddler emotion regulation. As in the first aim, toddler emotion regulation will be measured by a latent variable including the duration, latency, and intensity of anger expression.
Participants

Participants are from the longitudinal, multisite Early Growth and Development Study. The sample consists of 351 adopted children, each linked to adoptive parents and birth parents. Most (N = 343, 95%) of the parent couples were mothers and fathers; 18 (5%) were same-sex couples. Prior to testing this study’s predictions, differences between opposite- and same-sex couples in depressive symptoms, parent behaviors in the parent-child interaction, toddler emotion regulation, and the relations between depressive symptoms, parent-child factors, and toddler emotion regulation were systematically examined. No significant differences were found; therefore same-sex couples were included in the analyses. Throughout the dissertation, parents identified as Parent 1 will be referred to as mothers, and parents identified as Parent 2 will be referred to as fathers.

Recruitment took place between 2003 and 2006, starting with the recruitment of adoption agencies (N = 33 agencies in 10 states located in the Northwest, Mid-Atlantic, and Southwest regions of the United States). Agencies were selected to reflect the full range of adoption agencies in the United States: public, private, religious, secular, those favoring closed adoptions, and those favoring open adoptions. An agency-appointed liaison assisted with recruitment and identified participants who completed an adoption plan through their agency and met the following eligibility criteria: 1) the adoption placement was domestic, 2) the baby was placed within 3 months postpartum, 3) the baby was placed with a nonrelative adoptive family, 4) the baby had no known major medical conditions such as extreme prematurity or extensive surgeries, and 5) the birth and adoptive parents were able to read or understand English at the eighth-grade level. The liaison used the contact information provided by the agency to invite the
birth mother and adoptive parents to participate. The liaison then recruited the birth father or he was recruited through the birth mother. The study participants were representative of the adoptive and birth parent populations that completed adoption plans at the participating agencies during the same time period. For further details about the study design and sample description, please refer to papers by Leve and colleagues (Leve et al., 2007; Leve et al., 2008).

The data used to test hypotheses are drawn from the first three waves of assessment. The children were assessed in-person at ages 9, 18, and 27 months. Approximately half (43%) were female. The mean child age at placement was 3 days (SD = 13 days). The birth parents typically had high school or trade school education levels and household incomes under $25,000, and adoptive parents were typically college-educated, middle-class families. Adoptive parents had been married an average of 11.8 years (SD = 5.1 years; Range: 1.2 to 27.1 years). Table 1 provides additional demographic information.
Table 1

**Demographics for Birth Parents and Adoptive Parents**

<table>
<thead>
<tr>
<th>Variable</th>
<th>BM</th>
<th>BF</th>
<th>AM</th>
<th>AF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age (years)</td>
<td>23.83 +/- 6.12</td>
<td>25.31 +/- 7.42</td>
<td>36.96 +/- 5.55</td>
<td>37.89 +/- 5.93</td>
</tr>
<tr>
<td>Race (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>78</td>
<td>63</td>
<td>93</td>
<td>92</td>
</tr>
<tr>
<td>African-American</td>
<td>11</td>
<td>20</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Hispanic/Latino</td>
<td>4</td>
<td>8</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Multiethnic</td>
<td>5</td>
<td>5</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Other*</td>
<td>2</td>
<td>4</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Mean education level</td>
<td>5</td>
<td>5</td>
<td>9</td>
<td>9</td>
</tr>
<tr>
<td>Median annual house income</td>
<td>$14,000</td>
<td>$21,000</td>
<td>$100,000</td>
<td></td>
</tr>
<tr>
<td>Mean number of individuals in home</td>
<td>3.6</td>
<td>3.5</td>
<td>3.7</td>
<td></td>
</tr>
</tbody>
</table>

*Note. BM = birth mother; BF = birth father; AM = adoptive mother; AF = adoptive father.*

Education scores were as follows: 1 (*< 8th grade*), 2 (*completed 8th grade*), 3 (*completed 12th grade*), 4 (*some trade school*), 5 (*completed trade school*), 6 (*some junior college*), 7 (*completed junior college*), 8 (*some college*), 9 (*completed college*), 10 (*some graduate/professional school*), and 11 (*completed graduate/professional school*).

*a*Includes Asian, Native American/Pacific Islander, American Indian/Alaskan Native, and unknown.
Study Design

Birth parent and adoptive family in-person assessments were conducted by interviewers who completed a minimum of 40 hours of training, including a 2-day group session, pilot interviews, and videotaped feedback prior to administering interviews with study participants. All birth parent assessments were conducted in a location convenient to the participant, most often at home. The first birth parent interview occurred 3-6 months (average of 4 months) postpartum and the second was at 18 months. Adoptive family assessments occurred at 9, 18, and 27 months postpartum. All in-person assessments lasted 2-3 hours and included a collection of mailed self-report booklets, computer-assisted personal interview questions and interviewer impressions. The first birth parent assessment also included a pregnancy history calendar about the birth mother’s drug use mental health and stress during pregnancy and each adoptive family assessment included a series of videotaped interaction and standardized tasks. See Table 2 for the timing of primary study variables. For brevity, BM will refer to birth mother, BF to birth father, AM to adoptive mother, and AF to adoptive father.

Table 2
Timing of primary study variables

<table>
<thead>
<tr>
<th></th>
<th>4mo</th>
<th>9mo</th>
<th>18mo</th>
<th>27mo</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Birth Mother</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depressive symptoms</td>
<td>Retrospective report of prenatal depression &amp; current BDI</td>
<td>BDI &amp; CIDI</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Adoptive Parents</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depressive symptoms</td>
<td></td>
<td>BDI</td>
<td>BDI</td>
<td>BDI</td>
</tr>
<tr>
<td><strong>Adoptive Parents and Children</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Observational Tasks</td>
<td></td>
<td></td>
<td>Teaching Task (Parent-Child Interaction)</td>
<td>Arm Restraint (Toddler Emotion Regulation)</td>
</tr>
</tbody>
</table>
Measures

**Parent depressive symptoms.** Depressive symptoms were measured for birth parents at the first two measurement occasions and adoptive parent depressive symptoms were assessed all three measurement occasions using the Beck Depression Inventory (BDI; Beck & Steer, 1993). At approximately 4 months postpartum, birth mothers were also asked over the phone about prenatal depression using selected items from the BDI in a pregnancy history calendar. For consistency, only the subset of items from 4 months and 18 months were also used for birth mothers. Preliminary analyses also examined full BDIs and found comparable associations across time. The BDI is a commonly used instrument that assesses the presence of 21 symptoms of depression. In the present study, 20 of the 21 items were administered; the suicidal ideation item was removed to minimize situations where clinical follow-up would be needed (Leve et al., 2010). The BDI is widely used (Goodman & Gotlib, 2002) and significantly correlated with the Center for Epidemiological Studies Depression Scale (CES-D; Radloff, 1977) and with diagnostic interviews (Wilcox, Field, Proromidis, & Scafidi, 1996), although using the BDI may yield false positives when classifying depression versus diagnostic interviews (Field, 2002).

Depressive symptoms were calculated as the sum of the 20 BDI items (BM $\alpha = .92, .90$; BF $\alpha = .89, .87$; AM $\alpha = .71, .79, .84$; AF $\alpha = .75, .81, .84$); 0-13 is considered minimal range, 14-19 is considered mild, 20-28 is considered moderate, and 29-63 is severe. The scores for adoptive mothers ranged from 0 – 30 and adoptive fathers from 0 – 27.

Birth parent diagnoses of Major Depressive Disorder were assessed with the Composite International Diagnostic Interview (CIDI; Andrews & Peters, 1998; Kessler & Üstün, 2004) at the 18-month assessment. The CIDI is a comprehensive, fully standardized interview used to assess 17 major diagnostic areas according to the definitions and criteria of the tenth revision of
the International Classification of Diseases (World Health Organization, 1992) and the fourth edition of the American Psychiatric Association’s Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 1994).

Adoptive parent and child interaction. Data from the Parent-Child Teaching task at 18 months was used to examine the parent-child interaction. Observational studies frequently use teaching tasks in which parents are instructed to show their children toys and to encourage parent-child engagement (e.g., Forman & Kochanska, 2001; NICHD Early Child Care Research Network, 2006; Page, Wilhelm, Gamble, & Card, 2010). In most studies, this procedure is used to code parent’s support for children’s engagement, sensitivity, positive regard, intrusiveness, and supportive presence (Egeland & Heister, 1993).

Interactions between adoptive parent and child were observed separately using a 3-5 minute Teaching task. This task was coded using an adaptation of the Monadic Phases coding system (Cohn & Tronick, 1988; Moore & Calkins, 2004; Moore, Cohn, & Campbell, 2001) using a powerful behavior observational coding and analysis software package (Interact with p.a.t.t.e.r.n. analysis module; Mangold International) to facilitate coding and provide sophisticated data management and analysis functionality.

Coders were naïve to hypotheses of the study and were trained by the author. Behaviors were coded for parents and children that reflect the main dimensions of child and parent positivity/negativity, engagement, and responsiveness. For more detailed descriptions of the codes used in the current study, see Table 3. The coding of behaviors was done on a second-by-second basis. To establish inter-rater reliability of codes, subsets of the cases were double-coded. For child behaviors, 18.9% of cases were double-coded (κ = .74, excluding one code that had
Table 3

*Codes used in the Teaching Task*

<table>
<thead>
<tr>
<th>Code</th>
<th>Brief Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Parent Positive</strong></td>
<td></td>
</tr>
<tr>
<td>Scaffold</td>
<td>Parent structures task materials to facilitate child’s engagement with toys</td>
</tr>
<tr>
<td>Social</td>
<td>Parent attempts to engage child with toy play</td>
</tr>
<tr>
<td>Positive Touch</td>
<td>Parent touches child in a concerned, affectionate, and/or warm manner</td>
</tr>
<tr>
<td><strong>Parent Negative</strong></td>
<td></td>
</tr>
<tr>
<td>Disengaged</td>
<td>Parent seems “zoned out” or disinterested in task or child</td>
</tr>
<tr>
<td>Take away</td>
<td>Parent removes toy from child’s grasp</td>
</tr>
<tr>
<td>Negative Touch</td>
<td>Parent moves or touches child in a rough manner</td>
</tr>
<tr>
<td>Explore</td>
<td>Parent engages with toy him/herself, without intention of demonstrating toy for child’s benefit</td>
</tr>
<tr>
<td>Off Task</td>
<td>Parent engages in activity that does not pertain to playing with child (e.g., talking on cell phone)</td>
</tr>
<tr>
<td>Gaze Away</td>
<td>Parent is not looking at the child or at the toy</td>
</tr>
<tr>
<td><strong>Child Positive</strong></td>
<td></td>
</tr>
<tr>
<td>Play</td>
<td>Child plays with toy appropriately</td>
</tr>
<tr>
<td>Approach</td>
<td>Child takes action to move toy closer</td>
</tr>
<tr>
<td>Social</td>
<td>Action to engage parent with toy</td>
</tr>
<tr>
<td>Gaze Partner</td>
<td>Child looks at parent</td>
</tr>
<tr>
<td><strong>Child Negative</strong></td>
<td></td>
</tr>
<tr>
<td>Resist</td>
<td>Child actively resists action of parent or tries to escape</td>
</tr>
<tr>
<td>Proximity Outside</td>
<td>Child is outside of parent’s reach</td>
</tr>
<tr>
<td>Away</td>
<td>Child moves toy away from him/herself</td>
</tr>
</tbody>
</table>

poor reliability and was not used in these analyses). For mother behaviors, 22.2% were double-coded ($\kappa = .79$), and for father behaviors 18.5% ($\kappa = .82$).

There were some missing data. In 11 families, no child-father task was recorded due to the father being away from home during the visit. Additional videos were not created because of missing disks ($N = 5, 1.4\%$) and families not completing the Wave 2 visit ($N = 13, 3.7\%$).
Finally, 16 (4.6%) other mother-child videos and 14 father-child videos (4%) were recorded but not coded due to video problems during coding.

Upon coding completion, a selection of parent and child codes were combined to create scores for data analysis. For the parent, these combined variables were: 1) positive focus on child (engaging the child in the toy, scaffolding the child’s use of the toy, and using positive touch), and 2) parent disengagement or negative behavior (using negative touch, taking the toy away from the child, exploring the toy without engaging the child, looking away from the child and the toy, being disengaged). For the child, the combined variables were: 1) positive child engagement (looking at parent, playing appropriately with the toy, approaching the toy, socially engaging parent with the toy), and 2) negative child behavior (moving outside of reach of parent, looking away from the toy or parent, engaging in tasks not related to the presented toy, resisting parent physically). For brevity, the composite parent and child variables are referred to as Parent Positive, Parent Negative, Child Positive, and Child Negative.

Time domain time-series methods of coding parent-child interaction were used to compute an index of contingent responsiveness for both adoptive parents and their children. Contingent responsiveness was measured using dynamic factor models (Molenaar, 1985). Using LISREL 8.80, models were run for each dyad, in which contemporaneous and lagged relationships within and between partners were computed. Lagged relationships (1 second) between partners, which accounted for the autocorrelation in each person’s time series of behaviors, were extracted as indices of contingent responsiveness. Contingent responsiveness variables were created that represented 1) the degree to which Parent Positive behaviors predicted Child Positive behaviors and vice-versa, and 2) the degree to which Parent Negative
behaviors predicted Child Negative behaviors and vice-versa. The details on calculating these variables are presented in the Results section.

**Child emotion regulation.** Child emotion regulation was assessed from the Arm Restraint task at 27 months. The physical restraint of young children is an unavoidable and frequent parenting task (e.g., diaper-changing, dressing). As part of the Laboratory Temperament Assessment Battery (Lab-TAB, Buss & Goldsmith, 1998), the gentle arm restraint has been used widely in studies of children’s emotions (e.g., Camras et al., 1998; Potegal et al., 2007; Stifter & Spinrad, 2002), and has been used to capture self-regulation strategies such as self-soothing (e.g., Stifter & Bruangart, 1995; Stifter & Spinrad, 2002).

In the first 30 seconds of the task, children were placed in a high chair and given an attractive toy. Mothers were instructed to remove the toy from the child’s grasp and gently hold down the child’s arms for 30 seconds. Once the mother released her hold, children could play with the toy for 30 seconds and then the mother repeated a 30 second gentle hold. Finally, the toy was returned to the child and the experimenter and mother talked and played with the child until a neutral or happy state was reached. Mothers were instructed to refrain from interacting with the child as much as possible; however, mothers did not always comply with this instruction. Mother talking to the child and physical comfort (e.g., rubbing the child’s arms in a soothing manner while holding them) were coded as mother noncompliance, which was included as a control variable in all analyses using the Arm Restraint data.

The data were coded again using *Interact's p.a.t.e.r.n.* analysis module (Mangold International). Emotions were coded second-by-second using a microanalytic coding system using facial and vocal affect expressions to measure anger, sadness, happiness, and anxiety. We utilized descriptions of facial and vocal cues of emotion based on AFFEX (Izard, Dougherty, &
Hembree, 1983), a widely used system for coding infant affect using facial and vocal affect expressions, and incorporated relevant postural and gestural behaviors similar to those used by Weinberg and Tronick (1994). If two (or more) discrete emotions were observed within the same second, all were coded. If blends of affect expressions were observed, either a Mixed Negative code or Mixed Positive/Negative code was assigned. Coders were naïve to hypotheses of the study and were trained by the author. Eighteen percent of cases were double-coded to assess inter-rater reliability over the course of the coding (κ = .75). Temporal variables were created using MATLAB. A bout of anger was defined as one or more seconds of anger surrounded by 2 seconds of non-anger. That is, if there was only one second of non-anger between seconds of anger, the bout was treated as continuous. Emotion expression variables to be used will be latency to first bout of anger, average duration of anger, average intensity of anger, and peak intensity of anger during the 30 seconds when mothers first held down children’s arms in the arm restraint.

Because gentle arm restraint was developed to serve as an anger-eliciting task, for the purposes of the present study anger expression variables served as indices of emotion regulation. Specifically, a latent variable composed of the latency, duration, average intensity, and peak intensity of anger expression was created. Behaviors during the arm restraint were also coded using a subset of procedures from the Emotion Regulation Strategies coding system (Cole, 2008), and then categorized into purported strategies (e.g., distraction, self-soothing). As mentioned in the Introduction, analyses will focus on emotion expression, not strategies, as an index of emotion regulation in this task. From the possible sample of 351 cases, 252 coded cases were used for the arm restraint task at 27 months. A number of cases were excluded because of video error (N = 8, 2.3%), administration error (e.g., the toy was not removed from the child, N =
19, 5.4%), the mother and experimenter decided not to begin the task because of the child’s mood or the mother’s discomfort (N = 10, 2.8%), or because the child was too upset to stay seated in the chair and the task was discontinued (N = 14, 4%). Some videos were not created because of missing disks (N = 18, 5.1%), broken cameras or damaged disks (N = 7, 2%), or the families did not complete the Wave 3 assessment (N = 24, 6.8%).

Additional covariates. Child gender (coded as 0 for males and 1 for females) was a covariate. In addition, several other factors that could confound or result in misinterpretation of the findings were entered as covariates.

Openness in adoption. In open adoptions, birth and adoptive parents are able to have contact with each other. To take into account the potential confounding of genetic and environmental influences resulting from such contact, it is necessary to control for the level of openness in the adoption. The perceived openness of APs and BPs, rated on a 7-point scale, were aggregated to create an openness variable. There was a high rate of convergence among reporters and the openness measure represents an aggregation across reporters (Ge et al., 2008).

Parent age. Age could relate to level of depressive symptoms (Kessler, Foster, Webster, & House, 1992) and parenting quality (e.g., Bornstein, Putnick, Suwalksy, & Gini, 2006). BM, AM, and AF age were entered as control variables.

Pre- and neo-natal complications. To disentangle pre- and/or neo-natal complications from genetic influences, a score was created that was adapted from The McNeil-Sjöström Scale for Obstetric Complications (Kotelchuck, 1994; McNeil & Sjöström, 1994). The items that comprised the score were taken from five categories of complications: 1) Toxin exposure (exposure to radiation, x-ray, lead, and chemicals), 2) Drug and alcohol use (cigarettes, second-hand cigarette smoke, alcohol, marijuana, cocaine, amphetamines, heroin, prescription pain...
killers, inhalants, sedatives, and tranquilizers), and 3) Pregnancy risk (circulatory problems, infections, maternal age, prenatal care, weight gain and loss, nausea, bleeding, and fetal movement), 4) Preeclampsia risk (e.g., high blood pressure), and 5) Neonatal risk factors (e.g., low birth weight). The scale generated a score for each birth mother based on retrospective self-report of pregnancy and neonatal events. Events meeting a minimum threshold of being at least potentially harmful or relevant to infant outcome were summed to form a total score. The McNeil-Sjostrom Scale has been shown to have predictive validity (Nicodemus et al., 2008) and to be more sensitive than other common measures of obstetric complications (McNeil, Cantor-Graae, & Sjostrom, 1994).

**Infant Fussiness.** At 9 months, AMs and AFs completed an abridged version of the 24-month Infant Characteristics Questionnaire (ICQ; Bates, Freeland, & Lounsbury, 1979), which consists of 24 items rated on a seven-point scale with values of 7 indicating a very difficult temperament. The fussiness scale, which sums 6 out of the 24 items, was used in the present analyses (AM $\alpha = .81$; AF $\alpha = .82$). The highest fussiness score was used to represent child fussiness, regardless of whether taken from mother or father. AM and AF reports of child fussiness scores were correlated at .67 ($p < .001$).
Chapter 3. RESULTS

Data Analysis Overview

Aim 1: To specify patterns of genetic and environmental risk conferred by parental depressive symptoms using timing, chronicity, and severity of birth and adoptive parents’ depression.

Structural equation modeling using LISREL 8.80 (Jöreskog & Sörbom, 2008) was used to examine across-time stability of parental depressive symptoms and their influence on child emotion regulation at 27 months (see Figure 2). First, depressive symptoms were modeled as observed variables, and anger regulation as a latent factor composed of anger expression variables (latency to first bout of anger, average intensity of anger bout, average duration of anger bouts, peak intensity of anger bouts). Second, to understand how change in parental depressive symptoms over time could influence anger expression, linear growth of adoptive parent depressive symptoms was modeled using LISREL 8.80. Last, latent class growth modeling using MPlus 5.21 (Muthén & Muthén, 2004) was used to examine the severity and chronicity of parental depressive symptoms over time.
Figure 2. Model for examining the timing effects of adoptive and birth parent depressive symptoms on toddler emotion regulation.

Note. BM = birth mother; AP = adoptive parent; Prenatal = Retrospective reporting of prenatal depressive symptoms through a pregnancy history calendar; 4 mo & 18 mo = BM depressive symptoms at 4 & 18 months child age; Lifetime Dx = Birth mother lifetime diagnosis of MDD

Aim 2. To examine how genetic risk and environmental risk operate together to influence qualities in parent-toddler interaction.

Structural equation modeling was then used to test the degree to which birth mother depressive symptoms and adoptive parent depressive symptoms predicted the frequency of specific patterns of contingent relations between toddler and parent during the teaching task interaction. The AM and AF depressive symptom assessment at 27 months was not used to test the parent-child interaction at 18 months; only parent data from child ages 9 and 18 months will be used for this aim.
Aim 3. To examine whether the quality of adoptive parent-child dyadic interaction moderated risk associated with either the presence of a depressed adoptive parent (ExE or ExrGE), or of a biological parent (GxE or Gx rGE), on child emotion regulation.

Moderation models were planned using LISREL 8.80. First, if there were direct effects of adoptive parent depressive symptoms on the quality of child anger regulation (Aim 1), then we planned to test whether the frequency with which adoptive parent and toddler engaged in contingent negative or positive interactions moderated the relation. Second, if there were direct effects of the birth parent depressive symptoms on the quality of child anger regulation (Aim 1), then we planned to test the moderation of this path by the frequency with which adoptive parent and toddler engaged in contingent negative or positive interactions.

Due to the lower number of BF participants, BMs only were used in our structural equation models to account for genetic effects.

**Power Analyses**

Structural equation modeling theory is based on the premise that confidence can be expressed in the distribution of the obtained test statistics and standard errors as long as the total sample size is large (Bollen, 1989). Rough guidelines on the ratio of subjects to estimated model parameters have been suggested as a minimum of 5:1 for normal theory methods (Bentler, 1989). The power analyses used in the present studied went beyond this simple guideline, with the sample size of N=360 exceeding Bentler’s recommendation.

**Genetic and environmental main effects.** It is not possible to hypothesize a priori exact effect sizes for genetic and environmental influences. However, it is possible to model several alternative values based on commonly observed genetic and environmental effects. Given the commonly reported genetic effects for the constructs under investigation (range = .10–.50) and
findings for the effects of birth parent characteristics on adolescent adoptees (b = .31 and .42; Ge et al., 1996; O'Connor et al., 1998), a sample size of 360 provided power above .90.

**GxE interaction effects.** Based on previous results reported by Cadoret et al. (1995), power analyses indicated that the minimum sample sizes needed for detecting significant GxE interaction effect at the level of power .80 in multiple regression samples ranged from 19 to 220. Therefore the sample in the present study was more than adequate to detect interaction effects. In the SEM framework, power analyses indicate that, for differences between Fisher z transformations for two groups at .30 or .40 levels (Cohen, 1988), as hypothesized, a sample size of \( N_1 = N_2 = 177 \) (z differences = .30) or \( N_1 = N_2 = 101 \) (z difference = .40) are adequate for a power of .80, both of which were within the range for the sample size of the present study. A study by Bates et al. (1998) on Temperament x Parenting found that with split samples of \( N_1 = N_2 = 200 \), the resulting power for detecting the interaction effects in this study range from .50 to .98, indicating a good to excellent power in the current sample.

**Power for Testing Model Fit in SEM.** A critical issue in estimating SEM is related to the minimum sample size necessary to detect model fit. Power analytical procedures have been proposed by defining effect size in terms of a null and alternative value of root-mean-square error (RMSEA) fit index (MacCallum et al., 1996). MacCallum & Hong (1997) have shown that the RMSEA is a more preferable and accurate index for power analysis than traditional goodness-of-fit and adjusted goodness-of-fit indices. A sample size of 200 will fall short of achieving adequate power (.80) in accurate evaluation of model fit in SEM when \( df \)s range 30–50. With a sample size of 350, models with \( df \)s of 30–50 will have adequate power. The 360 triads will provide adequate power to detect genetic and environmental main effects, GxE interaction effects, and SEM model fit.
Aim 1: To specify patterns of genetic and environmental risk conferred by parental depressive symptoms using timing, chronicity, and severity of birth and adoptive parents’ depression.

Overview of Timing Analyses

It was expected that a) higher levels of birth parent depressive symptoms would be associated with quicker, longer, more intense anger expressions (i.e. poorer anger regulation) in toddlers during gentle arm restraint and b) both earlier and concurrent adoptive mother and father depressive symptoms would contribute to this association. The first step in these analyses was to examine the timing of parental depressive symptoms, expecting that both early and concurrent parental depressive symptoms would matter for child anger expression during the toddler period.

Before completing a full model including adoptive parents, we first examined whether depressive symptoms in BMs should be included as multiple assessments in one latent factor or as independent predictors over time. One possibility was that depressive symptoms during pregnancy – a prenatal timing effect – would predict the quality of toddler anger expression. It was also possible that the diagnosis of Major Depressive Disorder (MDD) alone would predict toddler anger expression, rather than just the level of symptoms, suggesting that the consolidation of a disorder would be more potent an influence than a mere symptom count. We first combined all parental depression assessment time points to create a latent factor of BM depressive symptoms. This approach was supported by preliminary SEM analyses suggesting that birth parent depressive symptoms were relatively stable over time and significantly loaded onto a latent factor with BM diagnoses of MDD. In addition, when tested as individual observed variables, assessments at each time did not differ in predicting quality of toddler anger expression or BM risk factors in pregnancy. Finally, BM depressive symptoms were not limited
to a prenatal effect or an effect of diagnoses-only. Thus all assessments were combined to form a latent factor of BM depressive symptoms that was used for all analyses.

Before testing the full model including BMs, AMs, and AFs, we first examined the timing effects in AMs and AFs separately in the presence of a genetic effect to guide the construction of the full model. First, we tested a model in which all three measurements of depressive symptoms predicted quality of toddler anger expression at 27 months in addition to the genetic prediction. The full model predicted quality of toddler anger expression at 27 months by a latent factor of BM depressive symptoms, as well as the determined timing effects of both adoptive parents (APs).

Each structural equation model included the following covariates: highest score from mothers or fathers from the fussiness subscale of the IBQ at 9 months, average score of openness of the adoption process, BM age, AM age, AF age, child gender, pregnancy risk, prenatal exposure to toxins, prenatal exposure to drugs, preeclampsia risk, and neonatal complications. The models also controlled for the number of times the adoptive mother intervened in the arm restraint task (maternal noncompliance), as mothers varied in their compliance with instructions to refrain from intervening to soothe the child.

Structural equation models were executed with covariance matrices of observed variables, using THEIL (Molenaar, 1996), a FORTRAN program for robust covariance matrix estimation. THEIL improves the condition of a covariance matrix while leaving its underlying structure intact. Descriptives, correlations, and data preparation were calculated in SPSS 18.0. Modeling was performed using LISREL 8.80 (Jöreskog & Sörbom, 1996). Model fit was assessed using the root mean square error of approximation (RMSEA), the comparative fit index (CFI), the non-normed fit index (NNFI), and the Normal Theory Weighted Least Squares Chi-
Sqaure ($\chi^2$). The RMSEA index includes adjustments for model complexity and is less influenced by the number of parameters in the model than other indicators of fit. The CFI compares the covariance matrices predicted by the model to the observed covariance matrix, and compares the null model with the observed covariance matrix. Like RMSEA, CFI is among the measures least affected by sample size (Fan, Thompson, & Wang, 1999). The NNFI corrects for model complexity, i.e., favors more parsimonious models (Kline, 2004), and by convention, if the NNFI is below .90 it is not an acceptable fit and above .95 is judged to be an excellent fit. For RMSEA, a value less than .05 is considered a good fit, and below .08 an adequate fit (Kline, 2004). By convention, the CFI should be equal or greater than .90 to accept the model. Chi-square is influenced by sample size and indicates good fit when $p > .05$. AIC was used to compare models; a lower AIC indicates a better fit. Only standardized betas are reported.

The means, standard deviations, ranges, minimums, and maximums for variables of interest are presented in Table 4. Log transformations and square root transformations were used to improve skewed distributions on all adoptive mother (AM), adoptive father (AF), and birth mother (BM) depressive symptoms data. An N of 351 was used for all analyses. Models were also tested with an N of 252 (the N subjects with data in the arm restraint), and the pattern of results remained the same.
Table 4

<table>
<thead>
<tr>
<th>Descriptive Statistics</th>
<th>Mean</th>
<th>SD</th>
<th>Min</th>
<th>Max</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Birth Mother Depressive Symptoms</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pregnancy History Calendar (reduced items, N=346)</td>
<td>9.16</td>
<td>4.60</td>
<td>5</td>
<td>40</td>
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<tr>
<td>4 months (N=344)</td>
<td>11.18</td>
<td>9.78</td>
<td>0</td>
<td>54</td>
</tr>
<tr>
<td>18 months (N=312)</td>
<td>11.23</td>
<td>9.37</td>
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<td>43</td>
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<tr>
<td>% diagnosed with MDD in lifetime = 30.8%</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Adoptive Mother Depressive Symptoms</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9 months (N=346)</td>
<td>3.59</td>
<td>3.15</td>
<td>0</td>
<td>15</td>
</tr>
<tr>
<td>18 months (N=336)</td>
<td>3.75</td>
<td>3.85</td>
<td>0</td>
<td>25</td>
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<td>27 months (N=328)</td>
<td>3.87</td>
<td>4.30</td>
<td>0</td>
<td>30</td>
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<tr>
<td><strong>Adoptive Father Depressive Symptoms</strong></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>9 months (N=335)</td>
<td>2.87</td>
<td>3.38</td>
<td>0</td>
<td>27</td>
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<tr>
<td>18 months (N=310)</td>
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<td>27 months (N=312)</td>
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<tr>
<td><strong>Parent-Child Dyadic Variables at 18 months</strong></td>
<td></td>
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<tr>
<td>Positive AM → Child (N=317)</td>
<td>-0.03</td>
<td>0.10</td>
<td>-0.33</td>
<td>0.25</td>
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<tr>
<td>Positive Child → AM (N=317)</td>
<td>-0.03</td>
<td>0.09</td>
<td>-0.33</td>
<td>0.19</td>
</tr>
<tr>
<td>Negative AM → Child (N=298)</td>
<td>0.01</td>
<td>0.07</td>
<td>-0.13</td>
<td>0.38</td>
</tr>
<tr>
<td>Negative Child → AM (N=298)</td>
<td>0.01</td>
<td>0.08</td>
<td>-0.16</td>
<td>0.32</td>
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<td>Positive AF → Child (N=308)</td>
<td>-0.00</td>
<td>0.09</td>
<td>-0.22</td>
<td>0.28</td>
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<td>Positive Child → AF (N=308)</td>
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<tr>
<td><strong>Child Anger Expression at 27 months</strong></td>
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<tr>
<td>Average Duration of Anger (in seconds, N=252)</td>
<td>11.26</td>
<td>10.38</td>
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<tr>
<td>Latency to First Bout of Anger (in seconds, N=252)</td>
<td>13.73</td>
<td>10.96</td>
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<tr>
<td>Average Intensity of Anger (N=252)</td>
<td>1.08</td>
<td>0.76</td>
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<td>3</td>
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<tr>
<td>Peak Intensity of Anger (N=252)</td>
<td>1.54</td>
<td>1.13</td>
<td>0</td>
<td>3</td>
</tr>
</tbody>
</table>

*Note. MDD = Major Depressive Disorder; Pregnancy History Calendar items are not directly comparable to other Birth Mother or Adoptive Parent depressive symptoms due to differences in how the questions were asked (e.g., with screener questions).*

**Timing of adoptive mother depressive symptoms.** In this model, both AM and BM depressive symptoms were examined as predictors of toddler anger expression. We first tested a model that included all hypothesized paths of interest and covariates. These included paths from
AM symptoms at 9, 18, and 27 months and from the latent BM depressive symptoms variable to 27 month toddler anger expression (Figure 2). In this model, several covariates, child gender, preeclampsia symptoms, and maternal noncompliance in the arm restraint task were significantly associated with the quality of toddler anger expression. Infant fussiness at 9 months was not associated with quality of toddler anger expression at 27 months, but was associated with adoptive mother depressive symptoms. In addition, significant intercorrelations among birth mother age, openness, and prenatal risk factors were included in the model. As expected, AM depressive symptoms were moderately stable over time (β’s = .57, .37, .31). In this full model with covariates and all hypothesized paths, only two hypothesized paths to toddler anger expression were significant: AM depressive symptoms at 9 months (β = .16, p < .05) and AM depressive symptoms at 27 months (β = -.19, p < .05). The model was adequate fit by most measures (AIC = 410.68, RMSEA = .038; CFI = .96; NNFI = .97; \( \chi^2 \) (190) = 284.68, p < .05).

In order to understand the AM timing effects to be entered into the full model, covariates that did not reach significance in predicting toddler anger expression were removed, as were hypothesized paths that did not achieve significance. Also in this model, we tested a path in which birth mother depressive symptoms predicted preeclampsia symptoms, which in turn predicted the quality of toddler anger expression.

While controlling for child gender, fussiness at 9 months, and maternal compliance in the arm restraint task, this model also revealed that higher levels of AM depressive symptoms at 9 months predicted greater toddler anger (β = .15, p < .05), whereas higher levels of AM depressive symptoms at 27 months predicted less toddler anger (β = -.19, p < .05). Also, higher levels of BM depressive symptoms predicted higher levels of preeclampsia symptoms during pregnancy (β = .30, p < .05), which in turn, predicted higher levels of toddler anger expression (β
By the AIC criterion, this model was an improvement over the previous model, though it was not a significant improvement with a $\chi^2$ test (Figure 3; AIC = 393.02, RMSEA = .035; CFI = .96; NNFI = .95; $\chi^2(199) = 285.02, p > .05$).

**Figure 3.** Adoptive mother timing model.

*Note.* $* = p < .05$. For clarity, associations between BM depressive symptoms and age, openness, and other prenatal risk factors are not pictured.

**Timing of adoptive father depressive symptoms.** In this model, both AF and BM depressive symptoms were examined as predictors of toddler anger expression. We first tested a model that included all hypothesized paths of interest and covariates. This model was built identically to the AM model, substituting AF symptoms for AM symptoms. In this model, AF symptoms were moderately stable over time ($\beta$’s = .52, .43, .36), but no AF assessment of
depressive symptoms predicted toddler anger expression when tested together or alone. All BM and preeclampsia findings remained the same as in the AM model.

**Full model with adoptive mother and father symptoms.** The next model test included BM, AM, and AF predictors. We selected timing effects based on the prior models: BM was tested as a latent factor, and AM 9 month and 27 month predictors were included. In addition, we included within time correlations between AM and AF symptoms, as well as cross-lagged associations between AM and AF depressive symptoms over time. AM and AF symptoms were not associated at 9 months or 18 months, but were significantly correlated at 27 months. Only one cross-lagged association was significant: AF depressive symptoms at 9 months predicted AM depressive symptoms at 18 months ($\beta = .12, p < .05$). This full model was an adequate fit by most measures (Figure 4; $\text{AIC} = 518$, RMSEA = .032; CFI = .96; NNFI = .96; $\chi^2 (285) = 386, p < .05$).
Figure 4. Adoptive mother and father timing model.

Note. Stability lines for AMs and AFs are in gray in order to highlight the associations added in this model between adoptive parents. * = \( p < .05 \). For clarity, associations between BM depressive symptoms and age, openness, and other prenatal risk factors are not pictured.

Overview of Chronicity Analyses

In order to understand how change in parental depressive symptoms over time could influence anger expression, linear growth of adoptive parent depressive symptoms were also modeled using LISREL 8.80. The approach to including covariates and understanding model fit replicates that in the timing analyses described above. Instead of modeling each assessment of depressive symptoms as an observed variable, two latent factors were created for mother and
father depressive symptoms: 1) the initial level of depressive symptoms (intercept), and 2) the degree of change of depressive symptoms over time (slope). The modeling of birth mother symptoms and covariates on child anger expression were the same as those in the timing analyses.

**Chronicity of adoptive mother depressive symptoms.** In this model, both AM and BM depressive symptoms were examined as predictors of the quality of toddler anger expression. The latent factors of intercept (initial level) and slope (change over time) of AM depressive symptoms were created. In this model, the intercept of AM depressive symptoms did not predict toddler anger expression, but the slope of AM depressive symptoms did ($\beta = -.31, p < .05$). That is, the more an adoptive mother’s depressive symptoms increased over time, the less quick, long, and intense were her toddler’s anger expressions at 27 months. The intercept and slope of AM depressive symptoms were not significantly correlated. In addition, child fussiness at 9 months predicted the slope of AM depressive symptoms ($\beta = .19, p < .05$). There were no significant changes in how the BM depressive symptoms and other covariates were related to child anger expression from the previous timing model. The model had an adequate fit by most measures (Figure 5, AIC = 433.26, RMSEA = .036; CFI = .96; NNFI = .95; $\chi^2$ (209) = 301.26, $p < .05$).
Figure 5. Adoptive mother chronicity (growth) model.

Note. * = p < .05. Numbers on the unidirectional arrows from AM Intercept and AM Slope to AM Depressive Symptoms indicate loadings in the lambda matrix. For clarity, repeated associations from the timing models (BM depressive symptoms, preeclampsia symptoms, covariates) and associations between BM depressive symptoms and age, openness, and other prenatal risk factors are not pictured.

**Chronicity of adoptive father depressive symptoms.** In this model, both AF and BM depressive symptoms were examined as predictors of the quality of toddler anger expression. The latent factors of intercept (initial level) and slope (change over time) of AF depressive symptoms were created. This model was identical to the AM model, with AF symptoms replacing AM symptoms. In this model, neither the intercept nor slope of AF depressive symptoms predicted toddler anger expression when tested together or alone.
Overview of Chronicity and Severity Analyses

Latent class growth modeling using MPlus 5.21 (Muthén & Muthén, 2004) was used to examine the severity and chronicity of parental depressive symptoms over time. The purpose of this analysis was to clarify whether the change in AM and AF symptoms over time describes the sample as a whole, or whether there are distinct classes of individuals for whom the change over time is different than another class, and whether, if there are distinct classes, those classes relate to child anger expression. In these analyses, birth mother depressive symptoms were not included. Latent class growth modeling was used to determine the number of classes that best described change in parent symptoms over time, and whether those classes predicted the level of toddler anger expression at 27 months of age. MPlus’s EM algorithm was used to account for missing data.

In these analyses, class membership was determined by examining output from MPlus, which provided average estimates of probabilities of class membership, the proportion of individuals in each latent class, and the Vuong-Lo-Mendell-Rubin (VLMR) Likelihood Ratio Test, which determines whether the number of classes modeled is a significantly better fit to the data than one fewer class.

Chronicity and severity of adoptive mother depressive symptoms. First, a model predicting 2 different classes of growth patterns for AM’s was modeled. The VLMR Likelihood Ratio Test indicated that 2 classes was a better fit for the data than 1 class ($p < .05$). The first class, which included 21.16% of the subjects, had an intercept of 0.90 (note that the depressive symptom scores were square root transformed) and a slope that differed significantly from zero of 1.44. This was a group of mothers who were lower in depressive symptoms at the outset and
whose symptoms grew with time. The second class, which included 78.84% of mothers, had an intercept of 3.96 and a slope that also differed significantly from zero of -0.51 (see Figure 6).

This was a group whose depressive symptoms were higher at the outset and declined with time. Class membership only marginally predicted toddler anger expression ($p < .10$). Membership in the first class was associated with lower levels of toddler anger expression. Notably, the slope and intercept of these classes did not predict toddler anger expression. The VLMR Likelihood Ratio Test indicated that 3 classes did not fit the data better than 2 classes ($p > .05$), and thus the 2-class solution was accepted. The marginal association of class membership with toddler anger

Figure 6. Adoptive mother chronicity and severity (latent class growth) model.

*Note.* Each line represents expected growth based on observed values. Both lines have intercepts and slopes significantly different from zero ($p < .05$).
expression is difficult to reconcile with our previous analyses, particularly because neither the slope nor the intercept of either class was associated with anger expression. These two classes will not be examined further in the analyses for this reason, but will be addressed in the discussion.

**Chronicity and severity of adoptive father depressive symptoms.** The 2-class solutions for AFs did not converge due to negative variance in the slope for both estimated classes (the psi matrix was not positive definite). After fixing the slope variance to 0, the VLMR Likelihood Ratio Test indicated that 2 classes did not fit the data better than 1 class \((p > .05)\), and thus further examination of multiple classes was not pursued. The 1-class solution indicated that neither intercept, nor slope of AF depressive symptoms predicted child anger expression.

**Aim 2. To examine how genetic risk and environmental risk operate together to influence negative qualities in the parent-toddler interaction.**

**Overview of Analyses**

The first step in determining how genetic and environmental risk contributed to the parent-toddler interaction was to create variables that represented dyadic qualities. There are multiple options for creating dyadic interaction variables, including cross-correlation function, Hidden Markov Modeling, and contingency analysis. One idiographic method for examining causal relations in a time series is dynamic factor analysis (Fisher, Newman, & Molenaar, in press; Molenaar, 1985). In this method, the procedure accounts for within time associations between partners, and within-subject continuity, and then the causal effects between partners can be estimated (as shown in Figure 7), provided there is not a third unknown variable that explains
the association. A vector autoregressive (VAR) model with a lag of one second was tested by creating a Block-Toeplitz matrix.

\[ \begin{align*}
\text{Partner 1} & \quad \text{Time } t \\
\text{Partner 2} & \quad \text{Time } t
\end{align*} \]

\[ \begin{align*}
\beta_{4,1} & \quad \text{Partner 1} \quad \text{Time } t + 1 \\
\beta_{2,3} & \quad \text{Partner 2} \quad \text{Time } t + 1
\end{align*} \]

*Figure 7. Dynamic Factor Analysis Model.*

*Note.* This model accounts for within time associations between partners, and within-subject continuity. Provided there is not a third unknown variable that explains the association, the cross-lags (\( \beta_{4,1} \) and \( \beta_{2,3} \)) account for how one partner’s behavior in the previous second influences the other partner’s behavior in the next second. For this dissertation, \( \beta_{4,1} \) and \( \beta_{2,3} \) were extracted for each dyad.

A VAR analysis was run for each adoptive parent-child pair in LISREL 8.80, and the causal effects between partners were extracted (see \( \beta \)s in Figure 7). That is, variables were extracted that represented how one partner’s behavior at time \( t \) predicted the other partner’s behavior at time \( t + 1 \). The \( \lambda \) matrix was set to identity and the \( \theta \) matrix was fixed at zero, restrictions that are necessary in LISREL for analyzing relations between latent variables with single manifest
variables. The cross-lagged parameters from the $\beta$ matrix were then extracted from each dyad. Because each model was saturated, the fit of each of the models cannot be evaluated. Two VAR analyses were run for each dyad: 1) the degree to which Parent Positive behaviors predicted Child Positive behaviors and vice versa, and 2) the degree to which Parent Negative behaviors predicted Child Negative behaviors and vice versa. Thus, four variables were extracted from each dyad representing:

1) the degree to which a parent’s positive behavior predicted a child’s positive behavior in the next second (Positive Parent led to Positive Child),

2) the degree to which a child’s positive behavior predicted a parent’s positive behavior in the next second (Positive Child led to Positive Parent),

3) the degree to which a parent's negative behavior predicted a child’s negative behavior in the next second (Negative Parent led to Negative Child), and

4) the degree to which a child’s negative behavior predicted a parent’s negative behavior in the next second (Negative Child led to Negative Parent).

It would have been possible to also run two more VAR analyses for the degree to which the positive behaviors in one partner predicted the negative behaviors in the other partner and vice-versa, but this would have yielded four additional variables for analysis. For parsimony, only the four above were examined, 50% of the possible contingent scores derived from the variables. Structural equation models were next tested in which adoptive parent depressive symptoms (9 and 18 months only) and BM depressive symptoms (modeled as a latent variable) predicted the four dyadic codes. Models were tested separately for mother and father dyads. The covariates included above (child fussiness at 9 months, average score of openness of the adoption process, BM age, AM age, AF age, child gender, pregnancy risk, prenatal exposure to
toxins, prenatal exposure to drugs, preeclampsia risk, and neonatal complications) were also included in these models. Covariation among the four dyadic variables was allowed. There was one outlier for Negative AF led to Negative Child. The reported associations were tested with it present and with it altered to the next lowest value, and the results were unchanged.

**Adoptive mother and child dyads.** As in previous models, AM depressive symptoms were moderately stable from 9 months to 18 months ($\beta = .57, p < .05$). A full model with all hypothesized paths and covariates was analyzed. Next, nonsignificant paths and covariates were systematically removed. The two Positive parent-child dyad variables were significantly correlated with each other, and the Negative variables were correlated with each other but this relation did not reach significance ($p < .10$). In addition, Positive Child led to Positive Parent and Negative Parent led to Negative Child were associated but their relation did not reach significance. Concurrent (18 month) AM depressive symptoms did not predict any of the four dyadic variables, but higher 9 month AM depressive symptoms were associated with higher Negative Parent led to Negative Child ($\beta = .13, p < .05$). Specifically, higher adoptive mother depressive symptoms when the child was 9 months of age were associated with the child responding negatively to maternal negative behaviors at 18 months.

Contrary to expectations, BM depressive symptoms were associated with higher levels of Positive Parent led to Positive Child ($\beta = .15, p < .05$) and Positive Child led to Positive Parent, although the latter relation did not reach significance ($\beta = .13, p < .10$). BM depressive symptoms, however, were associated with several prenatal complications that contributed to the quality of the parent-child interaction at 18 months. BM depressive symptoms also predicted prenatal Drug Use ($\beta = .34, p < .05$) and Exposure to Toxins ($\beta = .13, p < .05$). Drug Use was, in turn, negatively associated with Positive Parent led to Positive Child ($\beta = -.11, p < .05$) and
Positive Child led to Positive Parent ($\beta = -\ .17, p < .05$), and Exposure to Toxins was negatively associated with Negative Parent led to Negative Child ($\beta = -\ .11, p < .05$). Neonatal Risk, unassociated with BM depressive symptoms, was positively associated with Positive Parent led to Positive Child ($\beta = .16, p < .05$), and with Positive Child led to Positive Parent ($\beta = .09, p < .10$) and Negative Parent led to Negative Child ($\beta = .09, p < .10$), although the latter two associations did not reach significance.

Child 9 month fussiness was positively associated with AM depressive symptoms ($r = .12, p < .05$) and with Positive Child led to Positive Parent ($\beta = .12, p < .05$). Additional associations between BM depressive symptoms, perinatal risk factors, openness, and BM Age were also significant and included in the final model. The final model was an excellent fit by all indices (Figure 8; AIC = 260.86, RMSEA = 0.0; CFI = 1.0; NNFI = .99; $\chi^2 (155) = 150.86, p > .05$).
Figure 8. Adoptive mother and child interaction model.

Note. Dashed lines indicate marginal findings where \( p < .10 \); * = \( p < .05 \). For clarity, associations between BM depressive symptoms and age, openness, and other prenatal risk factors are not pictured.

Adoptive father and child dyads. As in previous models, AF depressive symptoms were moderately stable from 9 months to 18 months (\( \beta = .52, p < .05 \)). In parallel with the AM model, a full model with all hypothesized paths and covariates was analyzed. Next, nonsignificant paths and covariates were systematically removed. The two Positive parent-child dyad variables and the two Negative dyad variables were significantly correlated. AF depressive symptoms at 9 months did not predict any of the four dyadic variables, but higher 18 month AF depressive symptoms were associated with lower Negative Parent led to Negative Child (\( \beta = -.11, p < .05 \)).
Specifically, higher adoptive father depressive symptoms predicted fewer exchanges involving children responding negatively to their adoptive fathers’ negative parenting.

BM depressive symptoms were not directly associated with any of the four father-child dyad variables. BM depressive symptoms were, however, associated with prenatal Drug Use ($\beta = .30, p < .05$), which was correlated with Exposure to Toxins. Exposure to Toxins was positively associated with Negative Child led to Negative Parent ($\beta = .21, p < .05$). Additional associations between BM depressive symptoms, perinatal risk factors, openness, and BM age were also significant and included in the final model.

Finally, boys had more Negative Child led to Negative Parent exchanges ($\beta = -.11, p < .05$) and child 9 month fussiness was positively correlated with AF depressive symptoms. Child 9 month fussiness was associated with Positive Parent led to Positive Child ($\beta = .10, p < .10$), but the association did not reach significance. The final model was an excellent fit by all indices (Figure 9; AIC = 253.65, RMSEA = 0.0; CFI = 1.0; NNFI = 1.0; $\chi^2$ (161) = 155.65, $p > .05$).
**Aim 3.** To examine whether the quality of adoptive parent-child dyadic interaction moderated risk associated with either the presence of a depressed adoptive parent (ExE or ExrGE), or of a biological parent (GxE or Gx rGE), on child emotion regulation.

**Overview of Analyses**

For the final set of analyses, the first step was to select a dyadic variable for moderation. Then, we tested whether the timing and chronicity effects revealed in Aim 1 analyses held true for both higher and lower levels of the selected dyadic variable. As described in the analyses of Aim 2, eight dyadic variables had been created (four for mother-child dyads and four for father-child dyads).
child dyads). Splitting these into higher and lower groups would result in 32 (16 timing and 16 chronicity) replications of Aim 1. Therefore, to minimize the number of tests, only one dyadic variable was selected and tested for both mothers and fathers. We chose the Negative Parent led to Negative Child exchange variable (the degree to which children responded negatively to negative parenting) for several reasons. First, the analyses up to this point clearly showed that this pattern of interaction was associated with higher levels of AM depression at 9 months, suggesting that a negative parent-child cycle in which AM depressive symptoms caused more AM negative parenting that led to more negative child responses. Second, this variable provided a way to index change in child behavior over time when considered in the context of infant temperament at 9 months and toddler anger expression at 27 months. Therefore, the sample was split at the median for both AM and Child dyadic variables and AF and Child dyadic variables, resulting in 8 (4 timing and 4 chronicity) separate models for the replication of Aim 1.

Timing of parental depressive symptoms and **higher levels of negative exchanges between adoptive mothers and their 18-month-olds.** The sample was split at the median of the AM-child dyadic interaction (median = -.02), resulting in an N of 163 in the higher group. As in Aim 1, AM, AF, and BM depressive symptoms were examined as predictors of the quality of toddler anger expression. In the best-fitting model from Aim 1 with this half of the sample, child gender remained associated with toddler anger expression but not maternal noncompliance. Child temperament remained significantly associated with both AM and AF depressive symptoms at 9 months. In addition, significant intercorrelations among birth mother age, openness, and prenatal risk factors were included in the model. AM and AF depressive symptoms remained stable, and AF symptoms at 9 months predicted AM symptoms at 18 months ($\beta = .18, p < .05$). As previously shown but now with higher standardized betas, this
model found that higher levels of AM depressive symptoms at 9 months predicted poorer quality toddler anger expression ($\beta = .34, p < .05$), whereas higher levels of AM depressive symptoms at 27 months predicted better quality ($\beta = -.39, p < .05$). Moreover, higher levels of BM depressive symptoms predicted higher levels of preeclampsia symptoms during pregnancy ($\beta = .36, p < .05$), which in turn, predicted higher levels of toddler anger expression ($\beta = .23, p < .05$). The model was an adequate fit by most indices (Figure 10; AIC = 496.84, RMSEA = 0.042; CFI = .92; NNFI = .91; $\chi^2 (285) = 364.84, p = .00096$).

Figure 10. Timing analyses with higher levels of negative AM led to Child.

Note. * = $p < .05$. For clarity, associations between BM depressive symptoms and age, openness, and other prenatal risk factors are not pictured.
Timing analyses of parental depressive symptoms and lower levels of negative exchanges between adoptive mothers and their 18-month-olds. The lower half of the median split had an N of 155 for these analyses. In contrast to the higher group, families with lower levels of Negative Parent led to Negative Child did not show the effects reported for Aim 1. Instead, there were no significant associations between AM or AF depressive symptoms and levels of child anger expression. The relations between AM and AF depressive symptoms (both concurrent and the cross-lag) became marginal. Boys remained more likely to have higher levels of anger expression, and more maternal noncompliance with the task instructions was associated with more toddler anger expression. Although BM depressive symptoms still predicted preeclampsia symptoms ($\beta = .24, p < .05$), preeclampsia symptoms did not predict toddler anger expression. The model was an adequate fit by most indices (Figure 11; AIC = 498, RMSEA = 0.043; CFI = .92; NNFI = .91; $\chi^2 (285) = 366, p = .00084$).
Figure 11. Timing analyses with lower levels of negative AM led to Child.

Note. Dashed lines indicate marginal findings where $p < .10$; * $= p < .05$. For clarity, associations between BM depressive symptoms and age, openness, and other prenatal risk factors are not pictured.

Because effects of AM depressive symptoms were found for one half of the sample and not the other, an ExE or Ex rGE interaction was possible, depending on the interpretation of the Negative Parent led to Negative Child variable. Moreover, because the indirect association from BM depressive symptoms to preeclampsia to toddler anger was replicated for only the higher negative parent-child exchange group, a GxE or Gx rGE interaction was considered.
Timing of parental depressive symptoms and higher levels of negative exchanges between adoptive fathers and their 18-month-olds. The sample was split at the median of the AF-child dyadic interaction (median = -.02), resulting in an N of 164. As in Aim 1, AM, AF, and BM depressive symptoms were examined as predictors of toddler anger expression. Using the best-fitting model from Aim 1 with this half of the sample, no covariates remained associated with toddler anger expression. In addition, significant intercorrelations among birth mother age, openness, and prenatal risk factors were included in the model. AM and AF depressive symptoms remained stable, and AF symptoms at 9 months predicted AM symptoms at 18 months ($\beta = .12, p < .05$). As before, higher levels of AM depressive symptoms at 9 months predicted higher levels of toddler anger expression ($\beta = .17, p < .05$), but higher levels of AM depressive symptoms at 27 months were not predictive. Higher levels of BM depressive symptoms predicted higher levels of preeclampsia symptoms during pregnancy ($\beta = .32, p < .05$), but preeclampsia was only marginally associated with toddler anger expression ($\beta = .14, p < .10$). The model was an adequate fit by most indices (Figure 12; AIC = 492.03; RMSEA = 0.040; CFI = .92; NNFI = .91; $\chi^2 (285) = 360.03, p = .0017$).
Figure 12. Timing analyses with higher levels of negative AF led to Child.

Note. Dashed lines indicate marginal findings where $p < .10$; * = $p < .05$. For clarity, associations between BM depressive symptoms and age, openness, and other prenatal risk factors are not pictured.

Timing of parental depressive symptoms and lower levels of negative exchanges between adoptive fathers and their 18-month-olds. The lower half of the median split for adoptive fathers had an N of 145 for these analyses. In contrast to adoptive mothers, there were no significant associations between 9 month AM depressive symptoms and toddler anger expression, the association found in Aim 1 between 27 month AM depressive symptoms and child anger remained significant ($\beta = -.32$, $p < .05$). AF depressive symptoms at 9 months
predicted 18 month AM symptoms (β = .15, p < .05). Boys remained more likely to have higher levels of anger expression, and higher levels of mother noncompliance were associated with more toddler anger expression. Child temperament was only correlated with AF depressive symptoms, not AM depressive symptoms. BM depressive symptoms remained predictive of preeclampsia symptoms (β = .31, p < .05), as did preeclampsia symptoms of child anger expression (β = .23, p < .05). The model was an adequate fit by all indices (Figure 13; AIC = 454.94, RMSEA = 0.030; CFI = .95; NNFI = .94; χ² (285) = 322.94, p > .05).

* Figure 13. Timing analyses with lower levels of negative AF led to Child.

* Note. * = p < .05. For clarity, associations between BM depressive symptoms and age, openness, and other prenatal risk factors are not pictured.
Chronicity analyses with higher levels of negative exchanges between adoptive mothers and their 18-month-olds. The chronicity analyses mirrored the timing analyses. First we present chronicity effects for those dyads that were higher in negative exchanges between the adoptive mother and toddler. The path from BM depressive symptoms to preeclampsia remained significant ($\beta = .43, p < .05$), as did the path between preeclampsia and toddler anger expression ($\beta = .23, p < .05$). The path from AM depressive symptoms slope to toddler anger expression became marginal, although the beta increased in magnitude ($\beta = -.59, p < .10$), as did the path from child temperament to AM slope ($\beta = .17, p < .10$). The model was an adequate fit by most indices (Figure 14; AIC = 435.76, RMSEA = 0.053; CFI = .91; NNFI = .90; $\chi^2$ (208) = 301.76, $p < .05$).
Figure 14. Chronicity analyses with higher levels of negative AM led to Child.

Note. Dashed lines indicate marginal findings where $p < .10$; * = $p < .05$. For clarity, associations between BM depressive symptoms and age, openness, and other prenatal risk factors are not pictured.

Chronicity analyses with lower levels of negative exchanges between adoptive mothers and their 18-month-olds. For this group, the path from BM depressive symptoms to preeclampsia remained significant ($\beta = .43, p < .05$), but the path between preeclampsia and toddler anger expression did not. The path from AM slope to child anger expression dropped below the standard criterion for significance, as did the path from child temperament to AM slope. The model was an adequate fit by most indices ($\text{AIC} = 412.50$, $\text{RMSEA} = 0.047$; $\text{CFI} = .93$; $\text{NNFI} = .92$; $\chi^2 (209) = 280.50, p < .05$).
**Chronicity analyses with moderation by negative exchanges between adoptive fathers and their 18-month-olds.** As in Aim 1, neither split of the sample revealed significant associations between the intercept nor slope of AF depressive symptoms with toddler anger expression. All associations between BM and prenatal factors with child anger expression were replicated from the timing analyses with the split samples above.
Chapter 4. DISCUSSION

The findings of the present study demonstrate how individual and dyadic factors operate together to influence both genetic and environmental risk to toddler anger regulation. As predicted in Aim 1, we found that the timing of adoptive mother depressive symptoms were predictive of toddler anger regulation, specifically that more maternal depressive symptoms at 9 months was associated with more anger expression in toddlerhood, but that more depressive symptoms at 27 months was associated with less anger expression. Analyses of the chronicity of adoptive mother symptoms support the patterns from infancy to toddlerhood suggested by the timing findings. We found an effect of birth mother depressive symptoms on toddler anger expression, but not as expected. There was no direct effect of genetic risk to toddler anger expression, but birth mother depressive symptoms indirectly influenced toddler anger expressions through a complication in the prenatal environment, symptoms of preeclampsia. However, each of the above-described effects was moderated by the quality of the parent-child interaction. Although we did not find the expected associations between adoptive parent depressive symptoms and adoptive parent contingent responsiveness to child behaviors predicted in Aim 2, we did find adoptive parent depressive symptoms to relate to the amount children reacted negatively to parents’ negative or disengaged behaviors. As expected in Aim 3, this quality of the parent-child interaction played a role in exacerbating or mitigating biological and environmental risk factors. Throughout all of our analyses, we found no direct effects of father timing, chronicity, or severity of symptoms on toddler anger expression, but their relation to mother depressive symptoms underscore the importance of considering fathers in the family context. The discussion section will deal with each of these issues first and then discuss study limitations and questions raised by this study that suggest directions for future research.
The Role of the Adoptive Family Environment

The findings detail important aspects of the effects of the family environment, particularly the significance of the parent-child interaction and the implications of having a mother with elevated depressive symptoms on children’s anger regulation. One of the most novel findings concerned the timing of maternal depressive symptoms, which indicated unique associations between both 9-month and 27-month depressive symptoms and toddler anger expression, but in opposite directions. Children expressed more anger at 27 months if their adoptive mothers were more depressed when the children were 9 months of age, but they expressed less anger if their mothers were more depressed concurrently. Our analyses included the stability of mothers’ symptoms over time, and could not be due to shared genes as the adoptive mother and adopted child were, by design, genetically unrelated. At first the opposite effects for mother depressive symptoms and child outcomes at 9 and 27 months might seem counterintuitive, but there are several possible explanations.

We would not necessarily expect the same pattern of findings at different developmental stages for the child and parent. At 9 months of age, most children have not yet learned to speak, are limited in their mobility and self-efficacy, and are highly dependent on their parents for meeting their basic needs. Depressed mothers have been shown to be less responsive and less contingent in their responses to their children’s needs and emotions (Bettes, 1988; Cohn & Tronick, 1989; Gelfand & Teti, 1990; Rowe, Pan, & Ayoub, 2005). Infants of mothers with elevated depressive symptoms during this developmental stage may learn a pattern of behavior in which they work harder to communicate their needs to their parents. In arm restraint specifically, communicating needs would entail showing more distress in order to convey their desire to free
their arms and play with the attractive toy. At 9 months of age, infants whose mothers are being less responsive could learn to work harder to communicate that distress by showing more anger.

One might expect that we would find similar patterns of increased anger expression due to maternal depressive symptoms at later assessments. In fact, we found no independent influence of mother depressive symptoms at 18 months and that higher levels of depressive symptoms at 27 months were associated with lower levels of anger expression at 27 months. This association is different from that at 9 months in several important ways. Most notably, the children are older. Two-year-old toddlers are typically physically active, can express themselves with words as well as emotions, and have accumulated 18 months more experience than when they were 9 months. During those intervening 18 months toddlers would have had countless interactions with their parents, learning how their parents typically respond to their behaviors. Nine-month-olds will also have learned interaction patterns with their parents, but the additional 18 months, which occur during months of rapid language learning (e.g., Gleason, 2005), advancements in self-locomotion (Campos et al., 2000), and emotion regulatory capabilities (Cole et al., 2011; Kopp, 1982; 1989), could change expectations for both infants and toddlers. Perhaps, by the time infants have reached 27 months of age, if they have learned that their parents are not responsive, they then communicate less distress and expect less help from their parents during situations such as the arm restraint. Previous work has found that 2-year-olds of mothers with elevated levels of depressed and anxious symptoms express less frustration during a mishap, either because they have learned that it will not elicit a response or because they do not wish to upset their mother further (Cole, Barrett, & Zahn-Waxler, 1992). This pattern is supported by an examination of some of the additional behaviors coded during the arm restraint task. The frequency of toddlers’ support-seeking behaviors (e.g., looking towards mother,
speaking to mother) used in the restraint portion of the arm restraint is correlated with the level of adoptive mother depressive symptoms at 27 months ($r = -.20$, $p < .005$). That is, children are less likely to seek support from mothers with higher levels of symptoms. This correlation is not solely due to the association between anger expression and support-seeking strategies, as the relation between support-seeking and mother depressive symptoms remains even when controlling for levels of anger.

Overall, our timing analyses suggest a pattern in which 9-month-old infants communicate more distress to mothers with symptoms of depression, a communication pattern that is evident at 27 months of age. In contrast, 27-month-old toddlers with more depressed mothers show a different pattern, in which they communicate less distress, perhaps expecting less support from their mother.

**Chronicity vs. Timing**

In addition to examining the timing of parental depressive symptoms, we also studied the chronicity and severity patterns over time. Our chronicity analyses lend further support to the timing analyses discussed above. We found that if mothers’ depressive symptoms increase over time, children are less likely to express anger in the arm restraint task. Thus as toddlers learn that their mothers interact with them in an increasingly depressive style over time, they potentially adapt their behaviors to seek less support when in a challenging situation. Additional methodology, such as an intervention design with longitudinal assessment of child behaviors, would be needed to fully support this hypothesis. In an attempt to understand whether this chronicity pattern is true for some parents and not others, we used latent class growth curve analysis to determine if there were specific patterns of severity and chronicity within this sample of adoptive mothers. We found support for two distinct groups of mothers, one that starts with
higher levels of depressive symptoms that slightly decline across the three assessments, and another that starts lower, and increases almost to the level of the other group by the third assessment. However, the prediction of levels of anger expression using these groups did not reach significance.

One way to understand these groups would be to see them as descriptive of our findings reported with the timing analyses. Perhaps the group that starts higher and slightly decreases is similar to the association between 9-month depressive symptoms and higher levels of anger expression. When children’s mothers have higher level of symptoms at this early stage in development, the attachment status and dyadic pattern could be disrupted such that children are more reactive to challenging events and communicate their distress with more negative emotion. However, when mothers’ symptoms start lower and increase over time, this could initiate a different learning cycle. Toddlers whose mothers’ depressive symptoms increase at the age when they are learning to speak and act autonomously could respond to their mothers’ increased irritability and/or nonresponsiveness in ways other than the increased negative emotion seen in children whose mothers were higher in depression at 9 months. If levels are not higher until 18 and 27 months, they may have learned other ways to get their needs met (e.g., speaking, moving autonomously, seeking the other parent). In addition, they may have learned increasingly across time that their mothers are not likely to respond to cries of distress. Without examination of the parent-child interaction across time, these ideas are not empirically supported. The findings from our single assessment of the parent-child interaction are discussed in relation to these ideas below.

Another possibility about the two classes of mother depressive symptoms is that mothers in the class that starts low and increases over time are underreporting their symptoms at 9
months. Previous research found that mothers who scored “0” on the Beck Depression Inventory displayed many of the same characteristics as mothers with high scores, such as lower activity levels, less expressivity, and less frequent vocalizing (Field, Morrow, Healy, & Foster, 1991). However, closer examination of the adoptive mothers in the EGDS sample suggests that this is not the case. Mothers reporting either zero symptoms or very low symptoms (2 and below) at 9 months do not have unexpected differences in either later depression scores, partner scores, child anger expression, or parent-child dyadic scores.

Finally, our timing analyses suggested that there was moderate continuity in mothers’ depressive symptoms from 9 to 18, 18 to 27, and 9 to 27 months. This suggests that for at least some mothers in our sample, their rank order level of depressive symptoms within the sample remains similar to the level in earlier assessments. The relatively little change in some mothers could explain why we find no direct timing effects at 18 months. If a mother’s symptoms were stable from 18 to 27 months, we may only see that her 18-month symptoms predict her 27-month symptoms, which in turn predict child anger expression. There would not be any direct effects of 18-month symptoms in this case. Similarly, if a mother’s symptoms were stable from 9 to 27 months, we would not see a direct effect of her 9-month symptoms on child anger expression. This could suggest that the direct timing effect of mothers’ depressive symptoms at 9 months that we find is in mothers with little chronicity.

Putting together our timing, chronicity, and severity analyses advances our understanding of the adoptive family environment. In families in which we know that the associations are not due to genetic similarity, we find distinct patterns of risk to child anger regulation from maternal depressive symptoms that differ depending on the level and time course of the symptoms. There
are further implications of these findings when considering the effects of biological risk and the parent-child interaction.

**Biological Risk**

Consistent with previous research (e.g., Sullivan et al., 2000), we found evidence for a genetic effect of depressive symptoms on child anger expression; however, we found that this effect seemed to operate via preeclampsia symptoms. Birth mothers with more depressive symptoms, whether their depressive symptoms were during pregnancy, 4 months after birth, 18 months after birth, or measured by a diagnosis of MDD, were more likely to have symptoms of preeclampsia, such as hypertension, weight gain, and proteinuria (urine contains an abnormal amount of protein and can signal kidney problems). In turn, mothers with more preeclampsia symptoms had toddlers with higher levels of anger expression at 27 months. This association was significant in the presence of both adoptive mother and adoptive father depressive symptoms, suggesting that there is significant genetic risk from maternal depressive symptoms operating through the prenatal environment that is unique from the postnatal effects of interacting with a caregiver or caregivers struggling with depressive symptoms. To date, most studies reporting associations between child problems and parental depressive symptoms interpret these associations as a result of disruptions in parenting, family context, and reduced responsiveness or sensitivity on the part of the depressed parent (e.g., Chronis et al., 2007; Cummings et al., 2005). Although our findings support the important role of parental depressive symptoms as an environmental influence on child functioning, these findings also suggest that at least some of the impact of maternal depression on toddler problems is due to shared genes and the intrauterine environment.
This is not the first study to find associations between depressive symptoms and preeclampsia symptoms. Not specific to women during pregnancy, depression is a risk for hypertension after controlling for other known hypertension risk factors more generally, although this finding may be moderated by race (Davidson, Jonas, Dixon, & Markovitz, 2000; Jonas, Franks, & Ingram, 1997). A prospective population-based study found that depressive symptoms, as measured with the short form of the Beck Depression Inventory, were associated with an increased risk (odds ratio of 2.5) for preeclampsia during pregnancy (Kurki, Hiilesmaa, Raitasalo, Mattila, & Ylikorkala, 2000). Depression could lead to preeclampsia risk through altered excretion of vasoactive hormones or other neuroendocrine transmitters, which in turn could increase risk for hypertension. However, their study did not delineate the precise mechanisms involved in the increased risk and had several limitations including a low cut-off score for categorizing a mother as depressed (Alder, Fink, Bitzer, Höсли, & Holzgreve, 2007).

Additional potential mechanisms to preeclampsia risk have been proposed, such as through infections like urinary tract infection and periodontal disease (Conde-Agudelo, Villar, & Lindheimer, 2008). Most hypotheses surrounding the role of infection focus on the indirect effects mediated by enhancing the maternal systemic inflammatory response, although the relationship could be due to confounding factors, such as socioeconomic status (Conde-Agudelo et al., 2008). Depression could play a role in this process, as it has been linked to periodontal disease (e.g., Rosania, Low, McCormick, & Rosania, 2009) and poorer immune function (Kiecolt-Glaser & Glaser, 2002). Although the current study does not examine the underlying mechanism, it replicated a strong association between elevated depressive symptoms and risk for preeclampsia.
This is the first study to find effects of preeclampsia symptoms on child emotional development in toddlerhood. The effects of maternal stress on the developing fetus are well documented (see Van den Bergh, Mulder, Mennes, & Glover, 2005). Of particular interest to this study, Van den Bergh and colleagues refer to the animal literature, where we find that rats with more stress have decreased uterine blood flow, followed by higher blood pressure, raised catecholamine levels, and proteinuria, suggesting a potential mechanism for preeclampsia (Kanayama, Tsujimura, She, Maehara, & Terao, 1997). Impaired uterine blood flow is associated with intrauterine growth restriction, and thus a resulting lack of oxygen to the fetus (Van den Bergh et al., 2005). This lack of oxygen could ultimately lead to impaired neurobiological development. Although the precise mechanisms have not been fully delineated in the human literature, it is plausible that increased physical stress to the fetus, as a result of preeclampsia symptoms, is related to the increased anger reactivity at age two. Newborns of depressed mothers have been found to show a myriad of symptoms associated with abnormal neurobiological development such as delayed habituation, orientation, and autonomic stability, elevated cortisol and norepinephrine, lower dopamine and serotonin levels (Diego et al., 2004; Field et al., 2004), higher levels of crying and lower soothability (Zuckerman, Bauchner, Parker, & Cabral, 1990), and higher heart rate and cortisol during a mother-child interaction (Dawson et al., 2001). Some links have been found between newborn risk factors and later behavior. For example, newborn vagal tone was related to infant 5-month mother temperament ratings of frustration (Stifter & Fox, 1990). Complications from the physical stress of a mother’s preeclampsia, because of either increased rates of infection or stress related to depressive symptoms, could be a potential mechanism between birth mother depression and later child anger reactivity.
Thus, we find evidence of a genetic effect of depressive symptoms on child anger expression at 27 months, but only indirectly through birth mothers’ preeclampsia symptoms. While the literature provides suggestions about the probable pathways to increased reactivity to the arm restraint, the mechanisms are not well delineated. Of note, our proposed indirect pathway through preeclampsia was not found in all families. We found that the association between preeclampsia and toddler anger expression was moderated by the quality of the mother-child interaction.

**Implications of the Dyadic Interaction**

Contrary to most of the research on parent-child interactions with depressed parents (e.g., Cox et al., 1987; Feng et al., 2007), we found no associations between adoptive parent depressive symptoms and how parents responded to their child’s behavior. However, for both adoptive mothers and fathers, associations were found between parental depressive symptoms and how children responded to their parents’ behaviors. For mothers, the higher their depressive symptoms were at 9 months, the more likely children were to respond negatively to their mothers’ negative and disengaged behaviors at 18 months. In contrast, the higher fathers’ depressive symptoms were at 18 months, concurrently, the less likely it was that children would respond negatively to their fathers’ negative behaviors.

For mother-child dyads, this pattern of findings lends further support to the timing effects found between mother depressive symptoms at 9 months and children’s later anger expression. The parent-child interaction at 18 months could be a marker of a mechanism through which mothers’ depressive symptoms influence children over time. If mothers have more depressive symptoms at 9 months, children are more likely to respond to mothers’ negative behaviors with negative behaviors of their own, possibly because they learned to express more anger in response
to their mothers’ depressive symptoms during infancy. This pattern reflects a cycle of mother-child negativity (e.g., Patterson, 1982) that could have developed due to children’s learned reaction to nonresponsiveness in their mothers when they were infants.

We were also interested in examining the adoptive-parent dyad to determine if child genetic risk was driving the interaction with their parents, thus suggesting a presence of evocative gene-environment correlation. While the patterns of genetic and perinatal effects in our parent-child dyad models (as shown in Figures 8 and 9) may not seem clear, there are some factors that could explain the differences in the direction of associations. First, we find a direct association between birth mother depressive symptoms and the positive mother-child interactions, such that there are more positive responses from both partners if the child had higher levels of genetic risk from depression. This finding can be better explained when considering it in the context of the prenatal risk factors. Previous research finds prenatal alcohol exposure to relate to fewer positive parent-child interactions at 9 and 24 months of age (Brown, Olson, & Croninger, 2010) and both child negative affect and maternal emotional connectedness in a mother-child interaction task at child age 18 months (O’Connor & Paley, 2006), although mothers’ depressive symptoms unexpectedly were not related to prenatal alcohol use. The authors concluded that the lack of associations between mother depressive symptoms and alcohol use may have been due to lower levels of depression in the sample overall. Previous work in the current sample has found that lower levels of depression are associated with higher levels of discontinuation of substance alcohol and drug use during pregnancy (Massey et al., 2010). Indeed, the current study found that birth mother depressive symptoms predicted their alcohol and drug use during pregnancy, which was in turn negatively associated with positive responses in the adoptive mother-child interaction. Thus, the risk from depression that was also associated
with the tendency to use more substances during pregnancy made it less likely that mothers and children interacted positively during the teaching task, while the risk from depression unassociated with the tendency to use substances made it more likely to interact positively. It may be that genetic risk only reduces the positive qualities of the mother-child interaction if the genetic risk was also associated with an increased use of alcohol, drugs, and/or tobacco during pregnancy.

Our analysis of the dyadic interaction revealed additional associations between prenatal risk factors and parent-child dyadic qualities (as shown in Figures 8 and 9), some in unexpected directions. In the mother-child model, we find that exposure to toxins and neonatal risk relate to the interaction, but in the directions of fewer negative and more positive interactions. However, these perinatal risk factors may have cause to operate differently than ones such as preeclampsia and drug use. Other studies with these data find birth mother personality factors such as novelty seeking to relate to drug use (Massey et al., 2010), whereas it is more likely that exposure to toxins and neonatal risk factors relate to chance and living environment, rather than relations to birth mother depressive symptoms and genetic risk more generally. Thus, while the significant associations found between exposure to toxins and neonatal risk factors are interesting, they may be outside the scope of this project and represent important covariates that require further study.

Overall, we find continued support for the role of early adoptive mother depressive symptoms on later child behaviors, specifically that higher levels of depressive symptoms in infancy may stimulate a negative cycle of mother-child interactions that are evident in toddlerhood. Moreover, we find a role of children’s genetic risk in the mother-child interaction, but possibly only indirectly through birth mothers’ drug use during pregnancy. These findings highlight how environmental, genetic, and prenatal factors all play a role in parent-child
interactions in toddlerhood. This interaction also seems to play a pivotal role in how children’s anger expression is influenced by such environmental, genetic, and prenatal risk factors.

**Moderation by the Parent-Child Interaction.**

The aspect of the parent-child interaction that was the most related to adoptive parents’ depressive symptoms, child negative responses to parents’ negative behaviors in the previous second, moderated the influence of depressive symptoms from both birth and adoptive parents on child anger expression. EGDS studies have shown moderating influences of parent psychopathology on child outcomes in previous work (Leve et al., 2010; Natsuaki et al., 2010), but this is the first study to find an effect of a dyadic quality on both environmental and genetic influences. The discussion of the moderation will focus on how three predictors influence child anger expression: 1) 9-month adoptive mother depressive symptoms, 2) 27-month adoptive mother depressive symptoms, and 3) preeclampsia symptoms.

First, in our analyses with the entire sample we found that toddlers who had mothers with higher levels of depressive symptoms at 9 months, during an age of attachment formation and high dependency on mothers for meeting everyday needs, were more likely to express anger during the arm restraint at 27 months. This association was only true for toddlers who reacted more negatively to their mothers’ negative behaviors and more negatively to their fathers’ negative behaviors at 18 months. The moderation suggests that the environmental influence of maternal depressive symptoms in infancy is only present if there is also a negative dyadic interaction in toddlerhood. That is, mothers’ depressive symptoms at 9 months are only associated with later child anger reactivity if there are also negative qualities observable in the mother-child interaction. One way to explain this would be if mothers with higher symptoms at 9 months were the same mothers in the more negative parent-child interaction at 18 months.
However, mothers in mother-child dyads with more negative interactions at 18 months were only marginally more likely to have higher levels of depressive symptoms at 9 months ($p < .10$). It could be that higher level of depressive symptoms at 9 months are leading to the more negative interaction, which in turn leads to more anger reactivity at 27 months. This would suggest that the potential disruption of the mother-child relationship has negative consequences for the later parent-child interaction, which would be consistent with the literature (e.g., Matas, Arend, & Sroufe, 1978; Frankel & Bates, 1990). It could also be that having a less negative interaction at 18 months buffers children from the adverse effects on anger regulation associated with having a mother higher in depressive symptoms in infancy, suggesting that the moment-to-moment aspects of the parent-child interaction can serve a protective role, or be a mechanism through which risk is conferred.

Second, our analyses with the entire sample suggested that toddlers of mothers with higher levels of depressive symptoms at 27 months expressed less anger during the arm restraint, possibly because expressing moderate anger and frustration to a depressed parent would not be as likely to elicit helping behaviors. This association was only true for 18-month-old toddlers who reacted more negatively to their mothers’ negative behaviors, but who reacted less negatively to their fathers’ negative behaviors. Moreover, there are no mean differences in adoptive mother 27-month depressive symptoms between the high and low groups of parent-child negative interactions, partially suggesting that the interactions do not lead to differences in depressive symptoms at 27 months. An alternative way to think about the interaction would be that a more positive interaction buffers children from the effects of mother’s depressive symptoms at 27 months. There could be a stronger history of the mother-child relationship that encourages children to ask for support during the arm restraint at 27 months, even in the
presence of current depressive symptomatology. It also possible that our findings reflect differences in how mothers express their depressive symptoms, i.e., whether they are more withdrawn and/or irritable, or whether they are able to maintain positivity with their child even when struggling with internal feelings of depression.

However, the association works in the opposite direction for father-child dyads. The suppressing effect of maternal depressive symptoms on child anger expression is only present in dyads in which children respond to father negativity with less negativity. Children may learn to use the other parent for more support if the other is depressed (Hops et al., 1987; Hossain et al., 1994). The present findings may reveal a related but different pattern. Alternatively, the order of the parent-child interaction tasks, in which fathers almost always interacted with their child after the mother had, may have influenced the findings, e.g., the child was already frustrated and so the task was more difficult the second time. Moreover, the task may not be equivalent for fathers and mothers. Children, especially at this young age, may have spent less time with their fathers, rendering the father-child interaction more unfamiliar for the dyad. The father differences are discussed in more detail below.

Last, we found with the entire sample that birth mothers with more depressive symptoms were more likely to have higher symptoms of preeclampsia, which in turn predicted higher levels of anger expression at 27 months. The association was only found with toddlers who had reacted more negatively to their adoptive mothers’ negative behaviors. This moderation could be explained by differing levels of preeclampsia symptoms for the two groups divided by interaction quality. If children who reacted more negatively also had higher levels of preeclampsia, it could explain the association. That is, higher levels of preeclampsia could result in neurobiological changes that eventually alter how toddlers interact with their mothers.
However, differences in preeclampsia between high and low groups of the negative interaction were not significant. It may instead be possible that the quality of the dyadic interaction buffers some of the effects of preeclampsia, such that the higher likelihood of anger reactivity resulting from neurobiological changes from preeclampsia is only present if toddlers interacted negatively with their adoptive mothers. Notably, this interaction was not found in father-child dyads, suggesting that the interaction was either not as vulnerable to the child risk factors, or other risk and/or contextual factors overwhelmed those contributions.

If we interpret the parent-child interaction negative quality as an environmental effect and preeclampsia symptoms as environmental influence, then we have found several examples of ExE. Yet there are reasons to think there are genetic effects (G) operating here as well. First, because we found that birth mother depressive symptoms predicted drug use, which in turn predicted the mother-child negative interaction, some of the dyadic quality could be described as gene-environment correlation (rGE). That is, the child evoked a negative mother-child cycle of interaction due to genetic factors. Our findings could then be described as Ex rGE. Second, if we interpret the effect of preeclampsia symptoms on anger expression as a genetic effect due to its association with the birth mother depressive symptoms, then we have found examples of both Ex rGE (moderation of the adoptive mother depressive symptoms paths) and Gx rGE (moderation of the preeclampsia path). However, each of these interpretations could be debated as to which label should be assigned.

**Fathers**

Most of the discussion thus far has focused on the environmental contribution of mothers and mothering, with little focus on fathers. We did not find direct effects of father depressive symptoms on child anger expression, but this could be for several reasons. First, we measured
anger expression during the arm restraint task, in which the mothers were present but the fathers were not. When conceptualizing the toddlers’ anger expression as emotional communication of distress, the context of to whom the infant is communicating is very important. It is possible that if the arm restraint task had been carried out with fathers, we would have found a direct link between father symptoms and child anger expression.

Second, our finding that adoptive mothers’ depressive symptoms have a direct influence while adoptive fathers’ do not could reflect the relative amount of time each parent spends with the children. By using information about the number of hours per week that each parent works, we can infer how much time is spent with the child at home. In the EGDS sample, fathers work significantly more hours than mothers; moreover, in fathers only does that amount of work relate to levels of depressive symptoms. The fewer hours that fathers work each week, the more depressive symptoms fathers report at 9 months only ($r = -0.12, p < .05$). It is possible that, for some fathers, being home more often when the child is still an infant is associated with feelings of worthlessness or sadness, although this association could also be due to employment difficulties or other third variables influencing the association. Fathers can behave just as sensitively and appropriately in parent child interactions as mothers (Belsky, Gilstrap, & Rovine, 1984; Lamb, 1997), but research on fatherhood and parenting suggests that, on average, fathers become more engaged in child-rearing as the child becomes able to speak and act independently in toddlerhood (Laflamme, Pomerleau, & Malcuit, 2002). Fathers’ roles during infancy, when much of the time spent with infants is for basic care, could be less prescribed than mothers’, reflected in the fact that during toddlerhood fathers are expected to be playmates more than managers of basic care (Bretherton, 1985; Laflamme et al., 2002; McBride & Mills, 1993; Tiedje & Darling-Fisher, 1993; Wille, 1995). Thus, because fathers are spending less time with their
children with less involvement with basic care, their level of depressive symptoms may not be as detrimental to child outcomes. This aligns with some of the literature on parental depression that finds fewer effects on children from father symptoms than mother symptoms (Connell & Goodman, 2002; Field, Hossain, & Malphurs, 1999). However, previous research with this sample has found direct contributions of father depressive symptoms on child problems (Pemberton et al., 2010), and the previous work and current study finds that fathers’ depressive symptoms influence mothers’ depression. In the current study, fathers play a role in the family system, although direct effects on anger expression were not found.

Some research finds that the level of father involvement moderates the effect of maternal depression on child outcomes (Mezulis et al., 2004) or that maternal postpartum depression only contributes to toddler behavior problems if paternal psychopathology is present (Dietz, Jennings, Kelley, & Marshal, 2009). Our finding that 9-month father depressive symptoms are associated with maternal depressive symptoms at 18 months suggests some link between parental depressive symptoms that could develop in relation to the child, marital factors, or factors unmeasured by the study that may have been present before the child was adopted. For example, depression could alter co-parenting, marital satisfaction (see review by Beach, Fincham, & Katz, 1998), spousal support (Rook, Pietromonaco, & Lewis, 1994), and problem solving skills (Christian, O’Leary, & Vivian, 1994). Future research could benefit from examining mechanisms of risk transmission within a genetically-informed framework and in the family systems context.

**Limitations and Future Directions**

Several of the patterns in this study remain unexplained, and are thus promising avenues for future research. Although early child temperament was not a focus of this study, our findings
suggest that it could play an important role in family dynamics and the transmission of risk. Specifically, at 9 months of age, maternal symptoms of depression and infant propensity to communicate distress could relate to infant temperament. We found that children’s level of temperamental fussiness was associated with adoptive mother depressive symptoms. Children who are more distress-prone, whether that is biologically driven or due to learned responses to the environment or both, could be more likely react with distress when their goals are blocked. They could be more distress-prone because the person with whom they need to communicate is less likely to respond to low levels of distress due to depressive symptomatology. The reverse could also be true. Mothers could be responding to their infants’ temperamental fussiness with higher levels of depressive symptoms. They could feel more stressed while parenting, less efficacious (Porter & Hsu, 2003), or feel that their expectations of the adoption process and parenting have not been met. This hypothesis is supported in the chronicity analyses, where we found that mothers’ depressive symptoms were more likely to increase over time if their infants were higher in fussiness at 9 months of age.

Moreover, dyads in which toddlers are fussier and mothers are more irritable and less likely to be responsive could lead to disruptions in attachment formation. Nine months is a key age in development of attachment formation (Belsky, Rovine, & Taylor, 1984), and maternal history of depression is related to increased risk for infant insecure attachment (Carter, Garrity-Rokous, Chazan-Cohen, Little, & Briggs-Gowan, 2001), which is in turn related to childhood aggression and behavior problems (Cicchetti et al., 1998; Lyons-Ruth, 1992). Although attachment was not measured in this study, it follows that children could learn to express more anger due to an attachment disruption related to the degree of mothers’ depressive symptoms and children’s fussiness.
There are several limitations to this study that should be considered. First, the EGDS participants are representative of birth and adoptive families from the agencies participating in this study; however, the adoptive families have high educational and economic backgrounds in comparison to national norms. There is a need to include demographic and material-economic factors in studies of parental depression (Oyserman, Mowbray, Meares, & Firminger, 2000). For generalizability to more high-risk environments and more ethnically diverse samples, further studies need to be conducted. The demographic differences between the adoptive and biological parents in terms of age and socioeconomic status, both of which could conceivably contribute to the impact of psychological factors on child outcomes, could also be considered (Stoolmiller, 1998, 1999); however, past adoption studies show that the favorability toward adoptive parents has negligible effects on heritability and environmental estimates (McGue et al., 2007).

Second, due to the complex nature of our models, we did not analyze our data including depressive symptoms from birth fathers because fewer birth fathers participated (N = 90). Replication of the findings using birth fathers’ data would have provided a strong confirmation of genetic effects because the effects from a birth father to a child are not confounded with prenatal environmental factors. Without birth father data we are only including half of the child’s genetic makeup. We know, however, that the observed effects from birth mothers to children are not simply the result of prenatal stress from depression during pregnancy because prenatal depression did not make a prediction to child functioning unique of postnatal birth mother depressive symptoms. Future work incorporating careful measurement of genes, prenatal environment, and postnatal environment is needed to further disentangle these influences (Knopik, 2009).
Third, our observational measure indexed but was not a direct assessment of child anger regulation. Due to the limited repertoire of strategies available to a 27-month-old in the arm restraint, it was more prudent to look at anger expression itself rather than how specific strategies relate to the reduction in anger expression. Future work interested in understanding how parental depressive symptoms influence child emotion regulation should include a larger selection of emotion regulation tasks. Moreover, video quality from the parent-child interaction tasks did not allow us to measure parent or child emotion expression during the teaching task. Tasks that allow for emotion coding during parent-child tasks would better allow the measurement of dyadic emotion communication and some possible mechanisms for the transfer of risk from depressive symptoms. We also only examined a small portion of the parent-child interaction from what was available. Future work could examine more specific reciprocity patterns rather than general positive and negative factors, or could examine how one partner’s positive behaviors influence the other partner’s negative behaviors and vice-versa. Such a study was beyond the scope of this dissertation, but given the significance of the parent-child interaction found in this paper, could yield very important information. Similarly, future work should examine additional parent-child interaction moderators, which could either emphasize the unique role of the moderator used in this paper, or could uncover additional patterns of moderation and further targets for intervention.

Fourth, future models could examine reciprocal effects over time, such as one study that found child noncompliance at age 2 to predict concurrent maternal depressive symptoms, which in turn predicted internalizing and externalizing problems at age 4 (Gross et al., 2008). Last, the current study does not examine a number of potentially important contextual factors such as marital interaction, socio-economic status, social supports, and siblings. Some of these factors
have been shown to be important for the variables in this analysis. For example, there are additive effects for maternal depression and marital conflict on child outcomes (Essex, Klein, Cho, & Kraemer, 2003). These variables could be especially important for disentangling the differences in our models for mothers and fathers and better understanding how one parent’s depressive symptoms influence the other’s symptoms.

Expanding this research and addressing the limitations mentioned above would be done best in studies with genetically-sensitive designs. We could not have made conclusions about the unique effects of the prenatal environment, parent depressive symptoms operating through the environment, and the parent-child interaction without the use of the adoption design. Without such a design, our conclusions would have been uncertain because we would have been unable to differentiate genetic and environmental influences. Future research should utilize such designs to further disentangle the processes involved in transmission of risk and promotion of emotion regulation skills. For example, adoption and twin and sibling studies with extensive measurement could advance our understanding of the relative influences of genes, prenatal environment, shared environment, and nonshared environment, identifying who is most at risk and through what mechanisms.

In sum, our findings suggest that there are multiple ways through which parental depressive symptoms influence child anger expression in toddlerhood. This study underscores the importance of measuring parental depressive symptoms over time, and how a genetically-informed sample can highlight the distinct importance of the parent-child interaction as a mechanism for transmission of risk from genes, the prenatal environment, and the postnatal environment. Our findings could explain why interventions for children of depressed parents work for some families and not others (e.g., Cicchetti, Rogosch, & Toth, 2000; Compas,
Forehand, Keller, Champion, & Hardcastle, 2011). Understanding that the quality of parent-child interaction could buffer genetically- and environmentally-mediated effects highlights the need for targeted intervention with depressed parents and their children in early childhood.
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