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GENETIC, PRENATAL, AND FAMILY INFLUENCES ON THE DEVELOPMENT OF SOCIAL COMPETENCE IN EARLY CHILDHOOD

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Amanda M. Ramos

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The dissertation of Amanda M. Ramos was reviewed and approved by the following:

Jenae M. Neiderhiser
Distinguished Professor of Psychology
Dissertation Advisor
Chair of Committee

Karen L. Bierman
Evan Pugh Professor of Psychology

Erika S. Lunkenheimer
Associate Professor of Psychology

Gregory M. Fosco
Associate Professor of Human Development and Family Studies and Psychology

Kristin A. Buss
Professor of Psychology
Head of the Department of Psychology

ABSTRACT

Our understanding of the development of social competence in early childhood is greatly enhanced using an integrated approach merging family systems theory and behavioral genetics. Previous literature suggests that child social competence is influenced by heritable influences (Battaglia et al., 2017), prenatal distress (Carter et al., 2001a; DiPietro et al., 2006; Eichler et al., 2017), and parental responsiveness and hostility (Anthony et al., 2005; Feldman & Masalha, 2010; Hartas, 2011; Kochanska et al., 2008). However, previous literature has often considered the role of these influences separately without considering how these factors can inhibit or increase children's social competence in tandem or as a cascade of negative effects. The main research question of this dissertation is to examine the mechanisms by which children's early environment (prenatal and family) and heritable influences shape the development of social competence. This dissertation uses a longitudinal parent-offspring adoption design to examine two main aims: (1) whether the role of prenatal distress on the development on social competence is a transfer of heritable influences or a pure environmental influence, and (2) examine how children can impact parenting, while considering the role of gene-environment interplay. Structural equation modeling was used to achieve these aims. Results from this dissertation show that prenatal distress was not associated with social competence; however, there were heritable influences on social competence as well as earlier child behaviors (regulation and reactivity) that were associated with social competence at 4.5 years old. Also, results from the second study suggest that children and parents are engaged in a coercive cycle that negatively impact the development of social competence. Additionally, it suggests that family dynamics are either child-led or parent-led, depending on the child behavior being investigated. Taken together these studies support: (1) the necessity of accounting for heritable influences in studies, (2) the importance of considering the child when examining the role of the family, and (3) the implications of the prenatal environment on parenting and child behaviors. By beginning to address these research questions, our research supports the integration of multiple levels of influence in an attempt to not only move closer to elucidating mechanisms of change, but also further understanding the complex nature of the development of social competence.

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Chapter 1

Introduction

The early development of social competence is linked with children's academic, psychological, and socio-emotional wellbeing across the lifespan (Burt & Roisman, 2010; Jones, Greenberg, & Crowley, 2015; Ladd et al., 2014; Masten & Coatsworth, 1998; Masten & Obradović, 2006; Santos, Vaughn, Peceguina, Daniel, & Shin, 2014). Specifically, more socially competent children have higher academic achievement (Jones et al., 2015; Welsh, Parke, Widaman, & O'Neil, 2001) and lower levels of externalizing and internalizing behaviors (Blandon et al., 2010; Bornstein et al., 2010; Burt et al., 2008; Corredor et al., 2017). Social competence is also considered to be a "prerequisite" for school readiness because it is an important component for developing positive relationships with peers and teachers (Ladd, Herald, & Kochel, 2006; Masten & Obradović, 2006). These associations are due to the role of social competence as the child's ability to positively navigate their social environments (Denham, 2006; Garner & Estep, 2001; Ladd et al., 2006; Masten & Obradović, 2006). Therefore, understanding the factors that impact the development of social competence is important to more effectively target these skills in early childhood.

The literature has most often focused on exploring how the parent-child relationship and parenting influence the development of social competence (Fabes, Gaertner, & Popp, 2006; Karreman, van Tuijl, van Aken, & Deković, 2006). However, social competence has also been found to be impacted by prenatal factors (Bandstra et al.,

2010; Jones et al., 2013; Kully-Martens et al., 2012; Latimer et al., 2012), heritable factors (Battaglia et al., 2017; DiLalla et al., 2012; Knafo & Plomin, 2006b), and early child behaviors (Eisenberg et al., 2010). Although these various areas of research on social competence have provided a wealth of information about the potential factors that are associated with social competence, there have been fewer studies examining how these influences work together. Specifically, are prenatal, genetic, parenting and child effects associated with social competence via transactional, interactive, or cumulative processes? Without research explicitly examining how these influences work together it is not possible to have a clear articulation of mechanisms.

This dissertation will draw on several perspectives (i.e., family systems, behavioral genetics, and the fetal programming hypothesis) in an attempt to highlight the importance of integrating these lines of research to better understand the developmental mechanisms underlying social competence. Therefore, this study's research questions (see Figure 1.1) are as follows: (1) What are the unique influences of heritable, prenatal, parenting, and child behaviors on child social competence at age 4.5 years?; (2) Do prenatal influences mediate the association between heritable influences and child social competence, such that heritable influences are transmitted to the child via the prenatal environment?; (3) Do child behaviors evoke parenting behaviors, partially due to heritable influences (evocative *r*GE), which in turn influence child social competence?

First, this dissertation will review family systems theory and the behavioral genetics literature (Chapter 2). Chapter 3 defines social competence based on the current literature and provides a background of heritable, prenatal, and parenting and early child behavior influences on social competence. Chapters 4 and 5 are empirical papers testing

portions of the conceptual framework presented in Figure 1.1. The first paper (Chapter 4) examines the fetal programming hypothesis using a behavioral genetic approach to probe whether the prenatal distress has a direct or indirect influence on child social competence in preschool via early dysregulation and reactivity (path b to d), or whether prenatal influences are an indicator of genetic risk or resilience (path a to b). The second paper (Chapter 5) combines family systems theory and behavioral genetics approaches to examine whether children evoke mother and father parenting behaviors via evocative *r*GE which influences child social competence in preschool (path a to e to g). Finally, Chapter 6 summarizes the findings from the two studies in this dissertation, fits these findings within the current literature, and provides future directions for this research.

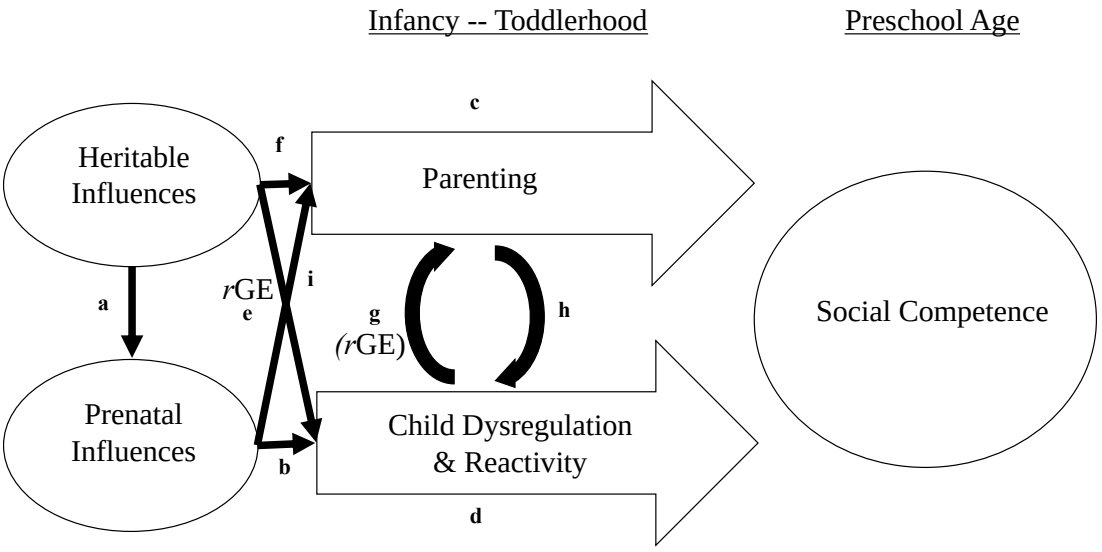

Figure 1.1. Conceptual Model for the Development of Social Competence. This figure is arranged with the earliest influences on the left-hand side. Block arrows indicate behaviors that occur over time.

Chapter 2

Family Systems Theory and Behavioral Genetics

Family Systems Theory

Family systems theory attempts to understand families as a socialization mechanism by examining the interconnected nature of the family (Cox & Paley, 1997; Minuchin, 1985; Nichols & Everett, 1986). Within the family there are multiple subsystems (e.g., interparental relationship, parent-child relationship) that are constantly shifting and are transactional in nature, such that one subsystem influences how another subsystem functions (Minuchin, 1985). These subsystems are organized in a hierarchical structure with the parental subsystem at the top, followed by the parent-child subsystem, and finally sibling subsystems (if there are siblings). In an effectively functioning family, these subsystems are separated by boundaries such that each subsystem knows their role/place; however, individuals are allowed to access other subsystems in order to access resources. For example, when a child is in conflict with one parent the child can access the other parent in order to help with conflict resolution. Although this theory imposes a hierarchical structure, it acknowledges the transactional nature of the subsystems, such that it is not always parent → child, but instead the child can influence the parent-child or interparental relationship (Minuchin, 1985). A majority of the literature particularly in social competence currently focuses on how parents influence their child's development (e.g., Sturge-Apple, Davies, & Cummings, 2006); however, other literature focuses on the more transactional nature of the family (e.g., Fosco, Lippold, & Feinberg, 2014). By

examining the family as a set of interacting systems, family systems theory facilitates our understanding of not only how each of the subsystems affect one another, but how the subsystems might influence the child's development (e.g., Buehler & Gerard, 2002; Gerard, Krishnakumar, & Buehler, 2006). In other words, family systems models examine the development of children as a function of social interactions.

Family systems theory is used in this dissertation as a way to conceptualize the unique or similar roles of mothers and fathers within the family and the bidirectional relationships between parents and children. This theory's strengths include examining complex family processes that influence the developing child and considering the role of the father alongside the mother. Additionally, family systems theory specifically addresses the important role the child plays within the transactional feedback loop of the family. A limitation to much of the research examining family processes, however, is a lack of clarity about possible mechanisms of change both within the family and within individuals. Although there is a feedback loop with parent and child providing inputs and outputs, this is typically described as environmental in nature. Studies using a family systems approach typically do not have the ability to disentangle genetic influences from environmental influences and therefore cannot claim that the effects are unambiguously environmental. As such, support for the influence of the parent-child relationship on children's development, may misinterpret this effect as environmental rather than as a combination of environmental and genetic influences.

Behavioral Genetics Approach

A behavioral genetics approach focuses on understanding how genetic and environmental influences account for individual differences in behavior in a population

(Knopik et al., 2017). Quantitative genetic designs capitalize on the different degrees of family relatedness (e.g., twin, sibling, and adoption designs) to examine how heritable and environmental influences work together to influence behavior. This dissertation will use a parent-offspring adoption design to capitalize on the variations in genetic and environmental contributions among an adoption triad (birth parents, adoptive parents, adopted child). Specifically, in an adoption design, birth parents provide the child's genes (and prenatal environment for birth mother), and adoptive parents provide the postnatal environment when children are placed at or near birth, but are genetically unrelated to the child. Therefore, associations between the birth parents and the adopted child are best explained as heritable (and prenatal influences) and associations between the adoptive parents and adopted child are best explained by rearing environmental influences (Knopik et al., 2017).

Gene-environment interplay. Aside from direct estimation of heritable and environmental influences, behavioral genetics approaches also consider how genes and environments work together (i.e., gene-environment interplay). There are two main ways that gene-environment interplay are examined: genotype x environment (GxE) interaction and genotype-environment correlation (*r*GE). GxE interaction occurs when there is an interaction between genetic and environmental influences, such that the effect of environment is dependent on the genetic influences, or vice versa (Knopik et al., 2017; Reiss et al., 2013). Genotype-environment correlation occurs when there is a correlation between the environment and the genotype of the individual (Knopik et al., 2017; Plomin et al., 1977; Scarr & McCartney, 1983), which is best explained in three different ways: *passive r*GE, *evocative r*GE, and *active r*GE. Passive *r*GE occurs when genes and

environments are correlated because parents and children share genes and parents provide the rearing environment. Evocative *r*GE occurs when an individual's genetically influenced characteristics evoke a response from the environment. For example, a child's genetic propensity to be social evokes a particular behavior from their parent (i.e., warmth). Finally, active *r*GE occurs when an individual's genetically influenced characteristics are correlated with the environment that they select. Peer selection is one example of active *r*GE (Cleveland et al., 2005). Active *r*GE is found less often in early childhood, and is theorized to become more important later in development (Scarr & McCartney, 1983) and is less relevant for parent-child relationships as children cannot select their parents (Neiderhiser et al., 2004). Behavioral genetics studies support that both passive and evocative *r*GE are indicated in studies examining parenting influences on child development (Marceau et al., 2013; McAdams et al., 2014; Narusyte et al., 2008); therefore, even in some genetically-informed designs it can be hard to distinguish which form of *r*GE might be occurring.

Adoption designs are particularly useful for identifying evocative *r*GE because the rearing parents are not genetically related to the child. Therefore, any influence of the child on the way they are parented that is also associated with birth parent(s) constructs represents evocative *r*GE. Passive *r*GE, on the other hand, is not present in a parent-offspring adoption design because adoptive parents and their adopted child do not share genes, thus confounds due to parents providing both genes and rearing environment to the child are removed. Previous studies using the same parent-offspring adoption sample as this proposal have found support for children's heritable characteristics (e.g., low social motivation, social wariness) evoking parenting behaviors (Elam et al., 2014; Natsuaki et

al., 2013). An additional strength of adoption designs is the ability to separate the prenatal and postnatal environments when both birth mothers and fathers are included (Loehlin, 2016; Marceau et al., 2013; Neiderhiser et al., 2016). Incorporating birth fathers' data allows us to examine the influence of the full genetic potential instead of looking at only birth mother characteristics that might be more likely to be associated with her prenatal experiences. Therefore, if estimates between the associations between birth mothers and child are similar to birth father and child, we can assume the associations are due to heritable influences.

Although a parent-offspring adoption design is a powerful way to clarify heritable and environmental influences, there are also limitations (Knopik et al., 2017). These limitations include selective placement, adoption openness, generalizability, and difficulty in distinguishing prenatal from heritable influences. Each of these limitations are discussed here with strategies that have been and/or can be used to address them.

Selective placement refers to placing adopted children into homes that are selected to be similar to, or distinct from, specific characteristics of the birth parents. If selective placement occurs it becomes more difficult to disentangle heritable and environmental effects because the environment being provided to the child may be confounded by genetic similarity between adoptive and birth parents. In other words, if adoptive parents are selected to be similar to the birth parents on IQ, for example, then any correlation between adoptive parent IQ and child IQ may be inflated because of genetic similarity rather than due to the rearing environment, and a correlation between birth parent IQ and child IQ may be inflated due to the rearing environment and not genetic similarity. Selective placement can be tested by examining associations between

birth parent and adoptive parent characteristics (i.e., personality, executive functioning). If the birth and adoptive parents are more similar than expected by chance then selective placement would be indicated. The sample used in this dissertation, the Early Growth and Development Study (EGDS), has found no evidence of selective placement bias (Leve et al., 2019).

Openness of adoption, or contact between birth parents and the adoptive parents and the adopted child, is another possible limitation of parent-offspring adoption designs. If the birth parents have a lot of contact with the child, it may be difficult to know whether the rearing environment is independent of heritable influences. Collecting information about the amount of contact and knowledge exchanged between the birth parents and adoptive parents allows the level of openness to be accounted for in analyses. Although most current adoptions are open to some extent, the level of contact tends to be low and generally does not contribute significantly to child outcomes (Knopik et al., 2017; Leve et al., 2013). One exception to this was reported for the EGDS. Openness of the adoption was significantly associated with child executive functioning at age 27 months, even after controlling for the effects of birth parent verbal IQ and executive functioning (Leve et al., 2013).

Another common criticism of parent-offspring adoption designs is the lack of generalizability. Concerns about generalizability have been raised in regard to all members of the adoption triad (birth parents, adoptive parents, and adopted children). For example, birth mothers might not provide the best prenatal environment to the child, compared to mothers who decide not to place their child, and adoptive parents might be less representative of nonadoptive parents (e.g., make more money, better educated, etc.).

For adoptive parents, there is the potential for restricted ranges on measures of the family environment (i.e., reduced variance distribution) (Stoolmiller, 1999). For example, adoptive families might have positively skewed distributions for negativity in the family and negatively skewed distributions for positivity. However, previous work has found that adoptive and birth parents are representative of unselected samples of biological parents (Petrill et al., 2003). Additionally, studies testing challenges associated with restricted range have found that although the range of environments provided by adoptive parents is somewhat restricted, this does not have an impact on child development (Colpin, 2002; McGue et al., 2007; McMahon & Gibson, 2002; Stoolmiller, 1999).

Finally, behavioral genetic designs still have difficulty disentangling heritable from prenatal influences (Loehlin, 2016). The adoption design allows us to separate heritable and postnatal influences, as any correlations we find between the child and adoptive parents behaviors (with no correlation between birth parent) is an environmental influence because the adoptive parents only provide the postnatal environment (Leve et al., 2013). However, if we find a correlation between social competence and the birth mother prenatal environment, behavioral genetic designs (including some adoption designs) cannot determine whether this is a heritable or prenatal influence. In order to clarify if the finding is due to the prenatal environment—and not heritable influences—we need to (1) include birth father data to account for the full heritable influences, and (2) have direct assessments of the prenatal environment that are being statistically modeled. Another potential option for adoption designs is to examine siblings who have been differentially exposed to a prenatal environment (e.g., mother smoked during one pregnancy and not the other); however, you would need to have siblings with the same

biological mother and father to control for shared genes. The EGDS design is such that we have recruited as many birth fathers as possible to participate in the study along with birth mothers and we model birth fathers along with birth mothers whenever possible in order to better distinguish heritable and prenatal influences.

This dissertation seeks to integrate the behavioral genetics approach with family systems theory. Therefore, when examining the bidirectional interactions between parents (mother and father) and the child, we will be incorporating not just environmental mechanisms, but the potential for the child's heritable influences to influence how the parents' parent the child (evocative *r*GE), as well as the potential for heritable influences to be transmitted through the prenatal environment. In the parent-offspring adoption design, we are able to better understand/examine the potential for these evocative effects because passive *r*GE is not present. The integration of these perspectives within a single study can help to further clarify the complex nature of family interactions between mother and child and father and child that in turn can influence the development of the child, without the confound of shared genes.

Chapter 3

Social Competence in Early Childhood and Early Risk and Protective Factors (Heritable, Prenatal, Parenting Influences, and Child Effects)

Definition

In early childhood, social competence encompasses a wide set of developmentally salient behaviors that include positive peer interactions, prosocial behaviors, empathy, and emotion competence (Corredor et al., 2017; Garner & Estep, 2001) as well as the ability to initiate and not withdraw from social interactions (Ladd et al., 2006; Rubin et al., 2009). Although social competence consists of positive behaviors, some work has utilized assessments of maladjustment (e.g., externalizing behaviors) singularly (Fabes et al., 2006; Masten & Curtis, 2000; Rabinowitz & Drabick, 2017). This linking of social competence with psychopathology was possibly predicated by the developmental psychopathology perspective, which posits that psychopathology is "normal development gone awry" (Cicchetti, 1984; Sroufe, 2009). Therefore, social competence and externalizing and internalizing behaviors were studied in parallel with far fewer studies taking a strengths-based approach to studying social competence. In an attempt to consider how externalizing behaviors and social competence are related, some work has begun to explore the simultaneous and longitudinal nature of social competence and externalizing and internalizing behaviors (Berry & O'Connor, 2010; Burt et al., 2008), and has found that social competence is associated with lower levels of externalizing and internalizing behaviors (Bornstein et al., 2010) and that externalizing behaviors predicted

lower social competence (Keane & Calkins, 2004). One small caveat is that some researchers hypothesize that youth who are high in externalizing could have higher levels of social competence (Mayberry & Espelage, 2006; Sutton et al., 1999); however, additional work does not find these associations (Berger et al., 2015). There is also support for their overlap from behavioral genetics research that has found that the associations between social competence and internalizing behaviors (Scaini et al., 2017; Wang & Saudino, 2015) and social competence and externalizing behaviors (Pesenti-Gritti et al., 2011; Reiss et al., 2000) were partially explained by genetic influences (4% - 33% and 50%, respectively). Therefore, it is possible for these constructs to not only affect one another, but also have etiological overlap that indicate an overall risk.

Effects of Early Regulation and Reactivity

Although social competence is generally first studied during Preschool and Kindergarten (Bornstein et al., 2008; Burt & Roisman, 2010), researchers have outlined a comprehensive list of behaviors that are developmental building blocks to social competence (Denham, 2006; Feldman & Masalha, 2010). These "prerequisites" or building blocks include emotion expression, regulation, and problem solving (Cole et al., 2004a; Denham, 2006; Hubbard & Coie, 1994). Therefore, it is not just about whether the child *can* navigate their social interactions, but whether they positively interact with the world around them by coordinating all of the skills they have been developing.

This dissertation examines child regulation and reactivity because they capture children's ability to control their behaviors and emotions within social situations and effective use of these behaviors and emotions are a precursor to social competence. Regulation is multi-dimensional and encompasses cognitive, behavioral, and emotional

behaviors (McClelland & Cameron, 2011, 2012). Children who develop the ability to regulate at an early age are better able to coordinate responses to situational cues (Rothbart, Sheese, Rueda, & Posner, 2011). For example, well-regulated children have the ability to focus attention and inhibit or engage in behaviors, which is essential for and facilitates positive social interactions and competence with peers (Eisenberg et al., 2001; Denham, 2003). Therefore, in order to initiate positive peer relationships, they need to coordinate their regulatory processes (i.e., emotion regulation, self-control, etc.) to navigate the social world. Reactivity, on the other hand, includes aspects of emotion like negative mood, high intensity emotional responses, and irritability and is different from the regulatory processes of attention, inhibition, etc. (DiPietro et al., 2008; Korja et al., 2017). Reactivity encompasses not just the intensity of emotions, but also frequency and latency (Dennis et al., 2006). The general displays of emotion and the potential for a specific propensity to be angry, sad, etc. (i.e., reactive) plays a role in the child's ability to interact with their peers. If a child is prone to anger, they could be more likely to respond to any form of frustration with anger. For example, if a child always responds in a negative manner when interacting with another child, children are less likely to consider them a friend or interact with them. This dissertation will focus on dysregulation that is specifically behavioral regulation and reactivity that is the proneness of the child to be angry (Blandon et al., 2010; Eisenberg et al., 2000; Kim & Kochanska, 2012). Most studies examine reactivity and dysregulation separately, although they are interrelated (Rothbart & Bates, 2006; Shields et al., 1994). For example, less reactivity in infancy and toddlerhood is associated with higher levels of social competence and prosocial behaviors in early childhood (Denham et al., 2003; Diener & Kim, 2004; Rubin et al., 1995).

Studies examining regulation found that impulsivity was negatively associated with social competence (Spinrad et al., 2006), while effortful control was positively associated with social competence (Fabes & Eisenberg, 1999). Therefore, reactivity and dysregulation in toddlerhood can have detrimental effects on the development of social competence in early childhood. These effects are dependent on both inherited traits and early interactions with the environment (prenatal and family influences) that helps the child learn and practice the skills necessary to become a socially competent individual.

Heritable Influences

One mechanism that social competence researchers should consider is heritable influences. Studies consistently have demonstrated that social competence is heritable from childhood through adolescence (Edelbrock et al., 1995; Hudziak et al., 2003; Roisman & Fraley, 2012; Van Hulle et al., 2007); however, there is less consistency about the amount of variance explained. Some studies show that as children develop, heritable influences increase, while shared environmental influences—nongenetic influences that account for twin similarity—decrease over time (Battaglia et al., 2017; Knafo & Plomin, 2006b). For example, one study examining children from ages two to seven years showed that genetic influences increased from two to four years old, while shared environmental influences decreased from moderate to non-significant by age seven years (Knafo & Plomin, 2006b); however, during middle childhood heritability estimates were not significantly different from estimates at age 4. Other studies focused on early childhood have more consistently found support for the moderate heritability (21 – 38%) of social competence (Roisman & Fraley, 2012; Saudino, Carter, Purper-Ouakil, & Gorwood, 2008; Van Hulle, Lemery-Chalfant, & Goldsmith, 2007). Additional studies

examining social competence during middle childhood through adolescence showed only moderate heritability with larger shared environmental effects (Edelbrock et al., 1995; Hudziak et al., 2003). These estimates might be due to the differences in measurement and sample ages not being consistent across time; however, these studies also show that heritability of social competence does not continually increase throughout the lifespan, but that shared environmental influences matter as well. Thus, the moderate heritability of social competence should be integrated into the conceptualization of influences on social competence in non-genetically informed studies, particularly as it relates to mechanistic effects.

Prenatal Influences

In addition to heritable influences, there is evidence that the prenatal environment is highly influential for children's socio-emotional development and particularly that prenatal exposure to stressors and toxins can have a long-lasting negative effect on social competence (Behnke et al., 2013; Dunkel Schetter & Tanner, 2012; Graignic-Philippe et al., 2014; Latimer et al., 2012; O'Connor et al., 2003; Rice et al., 2007). One theoretical explanation for how this occurs is through prenatal (fetal) programming (Barker, 1998; Kinsella & Monk, 2009; O'Connor et al., 2003; Weinstock, 2008). This hypothesis states that a stimulus during a sensitive period of development (e.g., prenatal) alters the development of the fetus, although the original conceptualization started with a focus on prenatal nutrition (Barker, 1998). This hypothesis has since been extended to additional prenatal experiences like stress that influences fetal development by "reprogramming" neural networks and the hypothalamic pituitary adrenal (HPA) axis (Ping et al., 2015). In addition, the fetal programming hypothesis has been fairly prolific in animal studies with

evidence showing that fetal programming doesn't stop at the second generation, but that the prenatal environment impacts future generations (Drake & Walker, 2004; Drake et al., 2005; Francis et al., 1999). Particularly one study found that rats whose mothers were exposed to a nutrient scarce prenatal environment saw effects similar to rats who had, themselves, been exposed to nutrient scarce prenatal environments (Drake et al., 2005). Although this hypothesis has been tested in numerous human and animal studies, the true test of this hypothesis would be to also include the effects of the maternal grandmother's environment as evidenced in animal models; however, many human studies do not have access to this information. Therefore, this is an intergenerational hypothesis that due to limitations, many studies are unable to fully test. However, studies examining the prenatal environment of the study child can still partially test this hypothesis.

One particularly important prenatal experience to consider that has the potential to reprogram the child's neural networks is stress (including both anxiety and depressive symptoms) because a majority of women experience some form of stress during pregnancy (Dunkel Schetter & Tanner, 2012; Glover, 2014) and approximately 21% of women experience some form of depression during pregnancy (Dunkel Schetter & Tanner, 2012; Glover, 2014; Rice et al., 2010; Woods et al., 2010). Prenatal stress is often defined as the perceived stress of the mother or anxiety assessed through behavioral or physiological measures (de Weerth et al., 2003; Dunkel Schetter & Tanner, 2012; O'Connor et al., 2003). A broader conceptualization of prenatal stress includes a wider range of exposures (e.g., anxiety, support, depression, stress) that can create a stressful fetal environment including negative affect because of the way it makes the mother feel about herself and the world (DiPietro et al., 2002). Generally, studies have found that

children who experienced higher levels of prenatal stress had higher levels of emotional and behavioral problems (O'Connor, Heron, Golding, et al., 2002) and less prosocial behaviors in early childhood (Loomans et al., 2011). Additionally, studies have found that prenatal depressive symptoms were negatively associated with social competence and positively associated with externalizing behaviors (Carter et al., 2001a; Eichler et al., 2017).

However, there is also a possible indirect effect of prenatal influences on social competence during early childhood through children's early dysregulation and reactivity. For example, some studies have found that prenatal stress is associated with higher levels of reactivity and dysregulation in infants and toddlers (Davis et al., 2007; de Weerth et al., 2003; DiPietro et al., 2008; Korja et al., 2017). Additionally, prenatal depressive symptoms have been positively associated with reactivity and dysregulation in children (Davis et al., 2007; Feldman et al., 2009). Studies examining the effects of both depressive and anxiety symptoms (i.e., distress) during pregnancy have found that fluctuations in these symptoms were associated with increased reactivity in infant and toddlers (Austin et al., 2005; Glynn et al., 2018; Nolvi et al., 2016). There has been less research examining the effects of both prenatal depressive and anxiety symptom influences on children's early regulation (DiPietro et al., 2006; Neuenschwander et al., 2018). For the purposes of this dissertation, we are calling this prenatal distress to better conceptualize the inclusion of prenatal anxiety and depression. One of the few studies to do this found that prenatal anxiety and depressive symptoms were associated with worse effortful control through increased reactivity (Neuenschwander et al., 2018). Therefore, child reactivity and regulation might represent a unique feedback loop that is influenced

by prenatal distress, but also influences children's social competence. Overall, these studies show that prenatal distress can increase early child reactivity and dysregulation, which can in turn influence the development of social competence. The relationships between prenatal distress and early child dysregulation and reactivity will be further explored in empirical paper 1.

Parenting Influences

In early childhood, mothers and fathers play an important role in the development of social competence due in large part to parenting (Clark & Ladd, 2000; Driscoll & Pianta, 2011; Haskett & Willoughby, 2007; Karreman et al., 2006; Masten & Coatsworth, 1998; Smeekens et al., 2007). Generally, the literature on parenting behaviors can be separated into positive behaviors (i.e., responsive, warm, and structured) (Anthony et al., 2005; NICHD Early Child Care Research Network, 2004), and negative behaviors (i.e., harsh, conflictual, and controlling) (Pettit et al., 1988; Waller et al., 2015; Ziv et al., 2016). Developmentally salient parenting behaviors are important to understand for the development of children's competency (e.g., self-regulation, positive peer relationships) during early childhood (Domitrovich & Bierman, 2001; Rabinowitz & Drabick, 2017). Positive and negative parenting behaviors will be further explored in empirical paper 2, focusing on the developmental saliency of specific parenting behaviors (responsivity and overreactivity) for social competence.

Positive parenting. Positive parenting encompasses a wide variety of behaviors; however, the most salient behaviors for early childhood [years zero to five] is responsivity and structure. For this dissertation, we will focus on parental responsivity. Responsiveness is presumed to be a parent-driven process whereby parents are

emotionally supportive and contingently responding to the child's needs in appropriate ways (Kochanska & Aksan, 2004; Landry et al., 2006). Importantly, the way that parents respond to different child bids could be important for understanding children's outcomes. The ability of parents to be responsive and sensitive to the child's needs helps to facilitate the development of a secure sense of self in the child (Thompson, 2000; Waters & Cummings, 2000). Numerous cross-sectional studies of parenting suggest that parental responsivity is important for the development of social competence (Feldman & Masalha, 2010; Mize & Pettit, 1997). Longitudinal studies have found that parent responsivity during infancy and toddlerhood have effects on social competence and prosocial behavior that last into preschool (Hartas, 2011; Narvaez et al., 2013; Rispoli et al., 2013). These studies suggest that responsivity might be how parents' model and reinforce positive reciprocal interactions that children enact with their peers. For example, responsivity might instill reciprocal feelings of reciprocity that the child will adopt in their future interactions (Kochanska & Aksan, 2004). Additionally, parents who are responsive could also be scaffolding the child as they are adapting to the child's developmental changes and potentially developmentally salient requests. Additionally, there is support that these positive parenting behaviors are having not just a short-term effect, but a lasting effect on children's positive behavior development.

While it is possible that parenting has a lasting effect through socialization and modeling, it is also possible that positive parenting is influencing social competence because they are increasing children's early regulatory and emotional capabilities through their parenting behaviors (Brophy-Herb et al., 2013; Feldman, 2015; Karreman et al., 2006; Kim & Kochanska, 2012; Leerkes et al., 2009; NICHD Early Child Care Research

Network, 2004). A few studies have found that mothers responsiveness in infancy or toddlerhood was positively associated with regulation in toddlerhood (Bernier et al., 2010; Leerkes et al., 2009; Vernon-Feagans et al., 2016). Another study found that maternal sensitivity was associated with infant reactivity from 4 to 16 months, but it was not specifically associated with anger reactivity (Braungart-Rieker et al., 2010). Therefore, early parental responsiveness could be providing opportunities for children to practice and learn to regulate themselves, which in turn is critical for their later positive development. Additional longitudinal examinations are needed to clarify pathways of family strengths by which parenting behaviors influence self-regulation and in turn children's social competence.

Negative parenting. Negative parenting, on the other hand, includes control—when structure and limit-setting become intrusive – and negativity. For the purpose of this dissertation, we will be focused on negativity. Negativity is characterized by coercion, harshness, and hostility. Negative parenting has been associated with lower levels of concurrent social competence and fewer prosocial skills (Anthony et al., 2005; Eddy et al., 2001; Laible et al., 2004; MacDonald & Parke, 1984; Pettit et al., 1988; Walker & MacPhee, 2011). Longitudinal studies find that parent hostility in toddlerhood is negatively associated with social competence at 5 years old (Hartas, 2011; Pettit et al., 1988). One possible explanation for the relationship between negative parenting and social competence is the coercive process between parents and children. Coercion theory hypothesizes that children's negative behaviors are due to a process of reciprocal reinforcement of harsh and coercive behaviors between parent and child (Patterson, 1982, 2016; Smith et al., 2014). Specifically, the parent reinforces the child's negative

behaviors, those child negative behaviors elicit more parental hostility, which elicits more child negative behaviors continuously until the interaction ends. This is presumed to not only model negative interaction patterns, but also impede the child's ability to regulate their own behaviors in other contexts. However, this process might also be a detriment to children's positive behaviors as well due to the consistent negative cycles they are receiving from their environment.

Some studies suggest that the coercive process can begin earlier in childhood and be associated with early dysregulation and reactivity (Eddy et al., 2001; Smith et al., 2014). This specifically might be due to the child's inability to regulate behaviors and emotions that they would normally begin to learn from the parent due to the parents modeling and displaying dysregulated behaviors. For example, parent negativity at age 2 was positively associated with children's later negativity (Rispoli et al., 2013) and parent aversive behavior was positively associated with children's antisocial behaviors (Eddy et al., 2001). However, this coercive process might be dependent on the type of behaviors that are examined (positive or negative), especially in early childhood. In one study parental negativity in toddlerhood was not associated with child prosocial behaviors, and child prosocial behaviors were not associated with parent negativity from 18 to 36 months (Daniel et al., 2016). Therefore, to further understand these processes, more work needs to be done to explore how parenting influences the development of social competence and whether and when early child dysregulation and reactivity plays a role.

Considering multiple parenting behaviors together. While current research supports the importance of numerous early parenting behaviors for the development of children's social competence, these behaviors are often examined independently of one

another. The studies that have begun to examine these distinct parenting behaviors together have found unique effects of each. For example, parental negativity at age 3 was associated with lower levels of prosocial behavior at age 5, after accounting for parenting sensitivity (Hartas, 2011). It is also possible that specific parenting behaviors may function uniquely to impact different child behaviors. For example, one study that examined multiple parenting behaviors (i.e., warmth, scaffolding, limit setting, and negativity) found that only warmth was associated with children's social competence 6 months later, while scaffolding and limit setting were associated with children's effortful control, and negativity was not associated with either social competence or effortful control (Lengua et al., 2007). While studying the influence of single parenting behaviors on child outcomes are important, this assumes that all parenting behaviors are equally important or uniquely influence the same child behavior; however, just as Lengua and colleagues (2007) found, certain parenting behaviors might directly impact child social competence or indirectly through their effects on other child behaviors like dysregulation. This is particularly important to consider in early childhood because children are experiencing physical, cognitive and social changes that can add strain to parents (Campbell et al., 2000). Parents must adapt to the developmental changes of their child as well as orchestrate positive social interactions within the child's world. Therefore, examining these parenting behaviors simultaneously can better help us to understand whether specific aspects of parenting might influence social competence or whether specific parenting behaviors might operate through different child behaviors. This will be addressed in empirical paper 2, specifically assessing longitudinal associations between parenting behaviors over time on the development of social competence.

Child-driven effects. One other reason that certain parent behaviors might matter at specific times in child development is because children might be eliciting those behaviors from their parents (Bornstein et al., 2008). Therefore, research needs to include child-driven effects, as parents might be more sensitive to children's behaviors at different developmental periods. Research including child-driven effects has focused particularly on the coercive process (Patterson, 1982; Smith et al., 2014). For example, a study found that children whose parents used harsh strategies in response to their children's negative emotions were more dysregulated and in turn was associated with less social competence (Fabes et al., 2001). However, children can also evoke less positive parenting behaviors (Eisenberg et al., 2010). One study examining parental responsivity found that while dysregulated children had parents who were less responsive at 24 and 36 months, children's dysregulation was also associated with less responsive parenting during the same developmental period (NICHD Early Child Care Research Network, 2004). Thus, not only does parenting behavior have a direct and indirect effect on social competence in children through child regulation, but children's early behaviors directly influence parents' early behaviors. Therefore, it is possible that developmentally salient parenting shown to influence social competence might be partly due to children eliciting these behaviors from their parents, supporting the need for examining parent and child-driven effects together. Therefore, this dissertation will not only explore the relationship between early parenting behaviors on children's later social competence, but also the role that the child plays in parenting behavior in paper 2.

Examining the Interplay among Genetic, Prenatal Environmental, and Postnatal Environmental Influences

There is a large literature exploring the individual effects of heritable, prenatal, and postnatal influences on the development of social competence in children; therefore, the future direction of the field is *how* genes and environments work together to influence the development of social competence, which this dissertation will explore.

Genetic and prenatal interplay. Findings from studies exploring the interplay of genetic and prenatal environmental influences, especially smoking during pregnancy (SDP), highlight the importance of considering these two influences together (Dolan et al., 2016; Gaysina et al., 2013; Knopik, 2009), particularly because of the complexity of prenatal influences. For example, one study found that birth mother depressive symptoms, measured as a heritable influence, were associated with toddler externalizing behaviors, while prenatal risks (e.g., drug use, toxin exposure) were not directly associated with toddler externalizing behaviors (Pemberton et al., 2010). However, birth mother depressive symptoms also had an indirect effect on toddler externalizing behaviors through prenatal risk (Pemberton et al., 2010), suggesting heritable risk for depressive symptoms might operate through prenatal risks to influence the externalizing behaviors in the child. Literature examining prenatal stress suggests that prenatal stress directly influences behavior in children (Rees et al., 2018); however, research incorporating heritable influences suggest inconsistencies in the effects of prenatal stress on children's early behaviors. While a few genetically-informed studies found that prenatal stress mediates the association between heritable influences and child behaviors (Kerr et al., 2013; Marceau et al., 2013; Rice et al., 2010), other studies have shown that prenatal stress has no effect on behaviors (e.g., ADHD, externalizing behaviors) after controlling for inherited influences (Gjerde et al., 2017; Rice et al., 2010). It is possible

that prior studies examining the effects of the prenatal environment (including meta-analyses) overestimate the influence of the prenatal environment because they do not control for heritable influences. Therefore, exploring these associations using a genetically informed design can help to disentangle heritable and prenatal influences and help researchers build converging evidence to support the effects of prenatal or heritable influences.

Prenatal environment and parenting interplay. Additionally, the inconsistent findings of the prenatal environments' direct effects on social competence (Dunkel Schetter & Tanner, 2012; Glover, 2014; Graignic-Philippe et al., 2014) might be due to the postnatal environment in which the child is raised (Bekkhus et al., 2011). While prenatal stress can be deleterious to the child, the postnatal environment can buffer or exacerbate these effects. One study found that after controlling for parenting behaviors, prenatal anxiety was still positively associated with emotional problems and ADHD at 4 years old (O'Donnell et al., 2014). A few studies have also examined the potential for the postnatal environment to mediate the association between the prenatal environment and child behavior. For example, prenatal stress negatively influenced the mother-child relationship (i.e., more intrusive), which in turn negatively influenced children's cooperative behaviors and increased the child's use of aggressive strategies while interacting with other children at four years old (Booth et al., 1991). Another study found that prenatal anxiety was associated with child externalizing behaviors at 3.5 years, but only for children of mothers who did not engage in skin contact (Pickles et al., 2017). Therefore, it seems that those children who are exposed to an adverse prenatal environment and have more negative parents have a greater risk for negative outcomes.

Although many of the studies examining prenatal and postnatal effects are conducted during later childhood, there is some evidence of this interplay impacting early dysregulation in children. One study found that maternal responsiveness, but not prenatal stress, was associated with child regulation at 7 months, but additional results found that mothers who were low in responsivity and had prenatal anxiety had children who were the most dysregulated, compared to mothers who were high in responsivity (Grant et al., 2010). Additionally, prenatal stress was associated with early child dysregulation through lowered maternal positive discipline (supportive, responsive, calm) (Kok et al., 2013) and lowered maternal sensitivity (Edwards & Hans, 2016). Therefore, the effects of the prenatal environment might leave children susceptible to negative outcomes if they are also exposed to risk in the early rearing environment.

Genetic and parenting interplay. The majority of studies examining the effect of parenting behaviors on child outcomes attribute these associations to environmental influences, disregarding potential heritable influences. Parents and children share the same genes, thereby causing a potential bias due to the inability of these studies to account for heritable influences (Moore & Neiderhiser, 2014). Therefore, the use of genetically informed designs can disentangle genetic and environmental influences to help provide a better understanding of the mechanisms involved in the development of social competence. Some of this work has found that parenting behaviors moderate the genetic propensity for social competence and other positive behaviors (Cheung et al., 2014; Deater-Deckard, 2000; Van Ryzin et al., 2015). Specifically, in an analysis using the same sample as will be used in this dissertation, Van Ryzin and colleagues (2015) found that when adoptive parents were low in responsivity at child age 2.5 years,

heritable influences were stronger for social competence in children at 6 years, but when adoptive parents were high in responsivity there was no association between heritable influences and social competence. These studies are important because they highlight that heritable influences can buffer children from their environment, and vice versa. Therefore, these studies help us to better understand why children might be resilient to their family environment and to their inherited risk.

Examining the role of gene-environment interplay can also help us to understand not only how parents impact development, but also, the role of the child in their own development. As previously mentioned, family studies have shown that children influence parenting behaviors (Bell, 1968; Heatly & Votruba-Drzal, 2017; Jia et al., 2012) and one mechanism for this is explained by heritable pathways via evocative *r*GE in infancy and toddlerhood (Hajal et al., 2015; Harold et al., 2013; Klahr et al., 2013, 2017; Natsuaki et al., 2013; Scarr & McCartney, 1983; Ulbricht et al., 2013). For example, children's impulsivity (Harold et al., 2013) and negative emotionality (Lipscomb et al., 2011) were associated with parents' higher hostility and overreactivity during early childhood, and some of these associations were explained by heritable influences. Few studies examine how these parenting behaviors, evoked by genetically influenced characteristics, influence outcomes directly (Elam et al., 2014; Knafo & Plomin, 2006b). One study found that low social motivation in birth parents was associated with more maternal and paternal hostility in adoptive parents, which was associated with higher levels of disruptive behaviors in adopted children at preschool age (Elam et al., 2014). These findings suggest an evocative *r*GE effect because the birth parent characteristic is associated with the adoptive parents' behavior and subsequently

the child's behavior with peers. These studies taken together provide support that children are actively engaged in their development, but also heritable characteristics are a mechanism that help to explain how development unfolds. This potential mechanism will be examined in paper 2.

Genetic, prenatal, and postnatal environment interplay. Finally, although the above literature focuses on gene-environment interplay, there are very few studies that incorporate all three sources of influence (genetic, prenatal, and postnatal), and none of these studies include specific measures of social competence. However, to begin considering how the interplay among heritable influences, prenatal environment, and postnatal environment might influence the development of social competence, this dissertation will review studies that examine heritable, prenatal environmental, and postnatal environmental influences on externalizing behaviors (Hannigan et al., 2018; Marceau et al., 2015; Marceau et al., 2013; Neiderhiser et al., 2016). One such study examined the effects of inherited risk (i.e., substance use, internalizing problems, and externalizing problems), prenatal risk, and adoptive parent overreactivity at 18 months on toddlers externalizing and internalizing behaviors (Marceau et al., 2013). This study found that prenatal risk did not mediate the association between inherited risk and child externalizing behaviors at 27 months. However, prenatal internalizing symptoms mediated the association between inherited risk (birth mother internalizing symptoms) and toddler withdrawal behavior, while accounting for parenting influences. Another study using the same sample also found that inherited risk (birth mother internalizing symptoms) was associated with child internalizing problems at 6 years through prenatal risk (prenatal internalizing symptoms and prenatal drug use) (Marceau et al., 2015).

Additionally, for both of these studies, overreactive parenting was uniquely positively associated with child internalizing and externalizing behaviors at 27 months and 6 years. Therefore, these two studies show that there might be specific types of prenatal influences that transmit heritable influences to child outcomes highlighting the importance of different mechanistic pathways. Additionally, these studies still support the importance of parenting behaviors along with heritable and prenatal influences. By using a genetically-informed design that also captures the prenatal and postnatal environment these studies highlight the complex (equifinal) nature of child development. In addition, these studies provide converging evidence for mechanistic pathways that prior studies have illuminated.

The Present Study

The core question of this dissertation is to understand how heritable influences and sensitive periods of development (prenatal and early postnatal) influence the development of social competence in early childhood. Specifically, this proposal will focus on key mechanistic pathways previously found to be influential in early childhood (inherited influences, prenatal stress, positive and negative parenting, and child effects) in 2 papers outlined in Figure 3.1. In addition, this dissertation will explore the role of the child, through evocative *r*GE, to partially shape their own development by influencing their rearing environment. **Paper 1** will examine heritable and prenatal distress influences on the development of social competence with child dysregulation and reactivity as potential mediators. There are three hypotheses for Paper 1. First, (1) birth parent traits will be directly related to child social competence at 4.5 years and indirectly related to child social competence through prenatal distress. We also hypothesize that (2) prenatal

distress will negatively influence social competence through child dysregulation and reactivity in toddlerhood. Finally, we hypothesize that (3) birth parent heritable traits will be associated with children's social competence through child dysregulation and reactivity during toddlerhood. **Paper 2** will investigate the mutual influences of parent and child from infancy to toddlerhood to further understand a potential heritable mechanism (evocative *r*GE) that is driving developmentally salient parenting behaviors that then influence social competence. This model will be testing both adoptive mother and father responsiveness and overreactivity in the same model. We hypothesized that (1) birth parent (BP) temperament (heritable influences) will be associated with parents overreactivity and responsivity, which in turn would be related to child social competence at 4.5 years. In addition, we hypothesized that (2) BP temperament would be associated with parenting behaviors via child dysregulation/reactivity, supporting evocative *r*GE effects. Finally, we expected that (3) there would be a bidirectional relationship over time between child dysregulation/reactivity and parenting during toddlerhood that in turn would influence child social competence at age 4.5 years. Such that, child reactivity and dysregulation would be associated with higher levels of overreactive parenting and lower levels of responsive parenting which would be related to lower levels of social competence. Also, overreactive parenting would be associated with higher child reactivity and dysregulation. This proposal is addressing novel questions that will attempt to elucidate mechanisms of change to get a better understanding of how children develop their social competence.

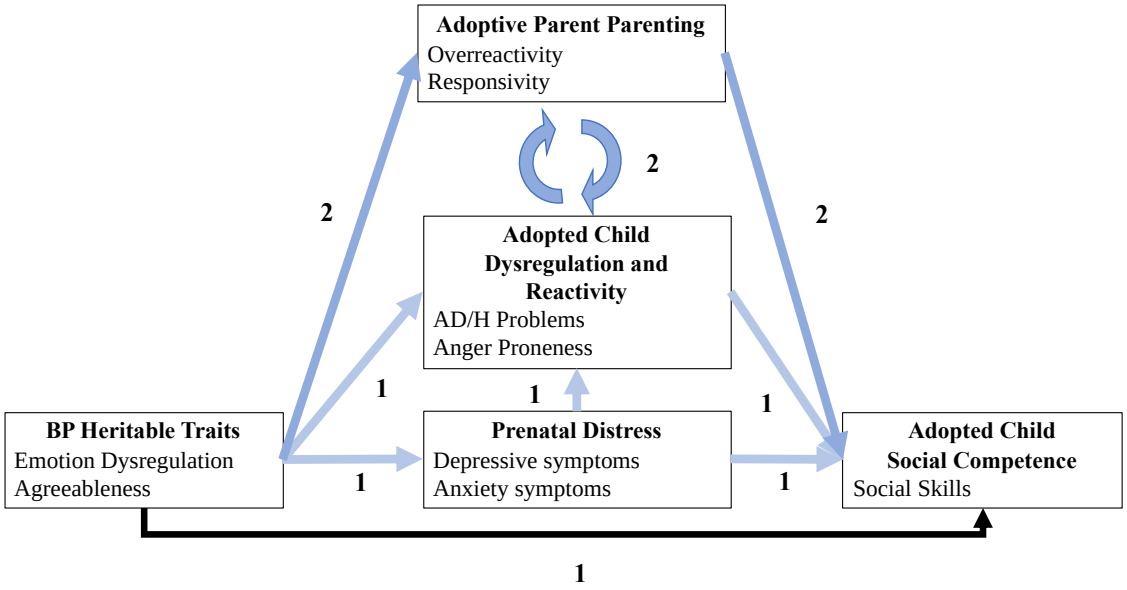

Figure 3.1 Conceptual outline of papers.

Chapter 4

The Role of Prenatal Distress on Children's Early Social Competence: Disentangling Heritable and Environmental Influences

Authors: Amanda M. Ramos, Elizabeth A. Shewark, David Reiss, Leslie D. Leve, Misaki N. Natsuaki, Daniel S. Shaw, Jody M. Ganiban, and Jenae M. Neiderhiser

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Abstract

Prenatal depressive and anxiety symptoms are linked with children's lower social competence in early childhood. However, it is unclear whether this is due to genetic risk transmitted through the prenatal environment, or the prenatal influence alone. Using data from the Early Growth and Development Study, a prospective parent-offspring adoption sample (N = 561), we examined heritable (birth parent temperament) and prenatal environmental influences on child social competence at 4.5 years old. We also examined whether associations between the prenatal environment and social competence were mediated by children's early dysregulation and reactivity at 18 and 27 months. Results showed heritable influences on children's social competence at 4.5 years old and prenatal distress was not directly related to social competence or indirectly via children's regulatory abilities. Additionally, children's reactivity and dysregulation at 18 and 27 months were negatively associated with children's social competence at age 4.5 years old. The findings reflect the importance of examining the effects of the prenatal period within a genetically informed design.

Introduction

The prenatal period has been shown to be a highly influential time for children's social and emotional development that can be negatively affected by external stressors such as maternal depressive and anxiety symptoms (Class et al., 2012; Kerr et al., 2013; Latimer et al., 2012; Natsuaki et al., 2010; Rice et al., 2007). One way to begin to understand how exposure to prenatal distress may manifest in children is to examine behaviors during early childhood, a time when children are developing early regulatory abilities and social competence (Cole et al., 2004a; Denham et al., 2003; Fabes & Eisenberg, 1999; Garner & Estep, 2001). Associations between the prenatal environment and social competence are typically presumed to be a direct influence of the prenatal environment, however, many of these studies do not account for the possible confounding effects of genes shared between the parent and the child (D'Onofrio et al., 2013; Knopik et al., 2016), or of the links between genetic risk and prenatal risks (Hannigan et al., 2018). As such, these studies are unable to delineate whether the influence is due to (1) genetic influences that increase the risk for prenatal distress, but also increase the risk for negative child behaviors or (2) if it is a direct influence of the prenatal environment on children's behavior. The major aim of this study was to investigate the influence of prenatal distress on the development of children's social competence by using a genetically informed design that is better able to disentangle heritable and prenatal influences.

Prenatal Distress on Social Competence

A majority of women experience some form of stress during pregnancy (Dennis et al., 2017; Dunkel Schetter & Tanner, 2012; Glover, 2014) and approximately 21% of

women experience some depressive symptoms during pregnancy (Dunkel Schetter & Tanner, 2012; Rice et al., 2010; Woods et al., 2010). In addition, prenatal anxiety and depressive symptoms often co-occur adding difficulty to the study of these influences (Ibanez et al., 2012). This study will be examining prenatal distress, which is a combination of prenatal anxiety and depression. Depressive and anxiety symptoms create a stressful fetal environment that effects the child both behaviorally and physiologically (DiPietro et al., 2002). Prenatal depressive symptoms, anxiety symptoms, and prenatal distress have been shown to negatively impact children's social development (Dunkel Schetter & Tanner, 2012; Hentges et al., 2019; Madigan et al., 2018; Rees et al., 2018; Waters et al., 2014). Mothers prenatal depressive symptoms are associated with lower children's social competence (Comaskey et al., 2017; Eichler et al., 2017) and higher externalizing and emotional problems (Hanington et al., 2012; Lahti et al., 2017; Van Batenburg-Eddes et al., 2013; Wolford et al., 2017) in preschool age children. Similarly, prenatal anxiety symptoms are associated with fewer prosocial behaviors (Loomans et al., 2011) and more externalizing behaviors (O'Connor, Heron, Glover, et al., 2002; O'Connor, Heron, Golding, et al., 2002). In addition, when studied together, prenatal distress has been shown to have a negative influence on children's social competence and externalizing behaviors (de Bruijn et al., 2009; Hentges et al., 2019; Madigan et al., 2018). Therefore, findings to date demonstrate the importance of understanding the prenatal mechanisms of influence that may alter children's social developmental trajectories.

The associations between prenatal distress and early child development are hypothesized to be due to fetal programming (Barker, 1998; Goodman & Gotlib, 1999;

Kingston & Tough, 2014; Ping et al., 2015). Fetal programming suggests that stress (i.e., a stimulus) alters the development of the fetus by reprogramming the neural network and hypothalamic pituitary adrenal axis (Barker, 1998; Goodman & Gotlib, 1999; Kingston & Tough, 2014; Ping et al., 2015). Two potential child behaviors that might act as an intermediary to children's social competence is regulation and reactivity. Regulation in children has been identified as an important prerequisite of social competence because early regulation allows the child to coordinate specific responses to situational cues (Rothbart, Sheese, Rueda, & Posner, 2011). For example, well-regulated children have the ability to focus attention and inhibit or engage in behaviors, which is essential for and facilitates positive social interactions and competence with peers (Denham et al., 2003; Eisenberg et al., 2001). Whereas reactivity, including latency and intensity of emotions, is a response to changes in the environment (Braungart-Rieker et al., 2010). For example, children can be prone to quick anger in situations, which can make it difficult to interact with peers who might be seeking different goals (Korja et al., 2017). Therefore, in order to initiate positive peer relationships, children need to be able to manage their emotions and coordinate their regulatory processes (i.e., emotion regulation, self-control, etc.) to navigate the social world. The current study intends to use these early child behaviors as two potential risk factors that prenatal distress can be influencing prior to social competence to examine the fetal programming hypothesis further.

However, there are some inconsistencies in the findings for the fetal programming hypothesis when examining these earlier child behaviors. The majority of the studies find that prenatal depressive and/or anxiety symptoms, when examined separately, are associated with children's early behaviors and in turn later social development (Blair et

al., 2011; Carter et al., 2001b; DiPietro et al., 2002; Feldman et al., 2009). Additional studies that have examined prenatal depressive and anxiety symptoms simultaneously found that they are associated with infant and toddlers's early reactivity (de Bruijn et al., 2009; Neuenschwander et al., 2018; Nolvi et al., 2016), while others find that they are associated with infant and children's early regulatory abilities (Austin et al., 2005; Barker et al., 2011). However, other studies find no effect of prenatal symptoms on infant and toddler's regulation or reactivity (DiPietro et al., 2006; McMahon et al., 2013). The current study focused on toddlerhood to further examine regulation and reactivity as a pathway by which prenatal distress can impact social competence, therefore, allowing a further test of the fetal programming hypothesis within a genetically informed design.

Confounding due to Heritable Influences

The assumption is that very early environmental influences set the child on a risk trajectory; however, it is possible that heritable influences also play a role. The majority of prenatal studies examine a mother rearing her biological child. This presents a confound that is not often acknowledged because the mother and child share genes in addition to the mother providing the prenatal environment. For this reason, associations found for prenatal influences on child behaviors may be due to prenatal environments and/or to genes shared between mother and child (Knopik et al., 2016, 2017; Moore & Neiderhiser, 2014). Studies have consistently shown that social competence and early regulatory abilities and emotions are heritable during childhood (Clifford et al., 2015; Edelbrock et al., 1995; Hudziak et al., 2003; Roisman & Fraley, 2012; Van Hulle et al., 2007). One study has also shown that the association between prenatal depression and children's depression was explained by heritable influences, and was not an

environmental effect (Hannigan et al., 2018). Therefore, genetically informed designs are needed to help disentangle the heritable and environmental (including prenatal) influences on children's social development. In particular, parent-offspring adoption designs of children placed at or near birth that include both birth mothers and birth fathers help to disentangle heritable influences from prenatal influences (Loehlin, 1989; Marceau, Hajal, et al., 2013; Neiderhiser et al., 2016). This is because the birth parents provide the heritable influences, the birth mother provides the prenatal environment, and the adoptive parents provide the postnatal environment. Therefore, if estimates between the associations between birth mothers and child are similar to birth father and child, we can assume the associations are due to genetic influences.

While there are no genetically informed studies examining the influence of prenatal distress on children's social competence, such studies have examined other child behaviors like externalizing and internalizing. While meta-analyses suggest that prenatal stress has an impact on children's behavior (Rees et al., 2018); genetically informed research suggest inconsistencies in the effects of prenatal stress on children's early behaviors (Hannigan et al., 2018; Kerr et al., 2013; Rice et al., 2010). A few genetically-informed studies have found that prenatal stress only partially mediates the association between heritable influences and child behaviors (Kerr et al., 2013; Marceau et al., 2013; Rice et al., 2010), but other studies have shown that prenatal stress has no effect on child behaviors (e.g., ADHD, externalizing behaviors) after controlling for inherited influences (Gjerde et al., 2017; Rice et al., 2010). For example, one study found that maternal prenatal depressive symptoms were associated with child externalizing behaviors in early childhood through heritable influences with no direct effect of maternal prenatal

depressive symptoms (Hannigan et al., 2018). This study's findings suggest that the genes that are influencing the child's behaviors are also influencing the type of prenatal environment experienced. Therefore, it is possible that prior studies examining the effects of the prenatal environment (including meta-analyses) overestimate the influence of the prenatal environment because they do not control for heritable influences. Therefore, exploring these associations using a genetically informed design can help to disentangle heritable and prenatal influences and help researchers build converging evidence to support the effects of prenatal or heritable influences.

The Current Study

The present study examined how heritable influences (i.e., birth parent agreeableness and emotion dysregulation) and prenatal distress influenced the development of social competence at child age 4.5 using a prospective parent-offspring adoption design. This design can help to advance our understanding of the influences of prenatal environments by helping to clarify the mechanistic pathways that may be directly related to prenatal influences from those due to underlying genetic propensities, while also considering how the two may operate together. Additionally, this study examined the influence of early child behaviors and emotions (dysregulation and reactivity) as a mechanistic pathway. We hypothesized that the prenatal distress pathway to social competence would be partially explained by heritable influences (i.e., birth parent emotion dysregulation and agreeableness). Additionally, we expected to find that prenatal distress negatively impacted social competence through higher child reactivity and higher child dysregulation.

Method

Participants

The Early Growth and Development Study (EGDS) is a longitudinal parent-offspring adoption study of birth parents (BP), adoptive parents (AP), and adopted children (Leve et al., 2019). This sample includes 561 families followed from birth to early adolescence. Families were recruited from 45 adoption agencies in 15 states, with adoption agencies ranging from public, private, religious, and secular. Due to family movement over time, the EGDS families reside in 46 states, the District of Columbia, and 7 other countries. Data collection for the first wave ranged from 2003 to 2010. Data collection for child age 4.5 occurred from 2007-2014.

Child demographics. The majority of children (57.2% male) were White (55.3%), with 19.6% multiracial, 13.2% Black, 10.9% Latino, .5% American Indian or Alaskan Native, .2% Asian, .2% Native Hawaiian or Pacific Islander, and .2% did not report. The mean age of the child at the time of placement in the adoptive home was 5.58 days (*SD* = 11.32 days, Range = 0-91 days).

AP demographics. Most of the time AP 1 was the mother (96.6%) and AP 2 was the father (94.5%). The majority of AP 1's were White (91.8%), with .4% multiracial, 3.9% Black, 2% Latino, .2% American Indian or Alaskan Native, .9% Asian, and .4% did not report. The majority of AP 2's were White (90.4%), with 1.1% multiracial, 4.9% Black, 1.6% Latino, .5% Asian, .5% Native Hawaiian or Pacific Islander, and .9% did not report. At the first data assessment, there were 41 same-sex parent families. Our adoptive parents were generally well-educated (on average obtaining a college degree), had a median income of $100,000-125,000 (Range: <$15,000 to > $300,000), and were in their

late thirties at the time of the child's birth (adoptive parent 1: *M(SD)* = 37.43 (5.59); adoptive parent 2: *M(SD)* = 38.30 (5.83).

BP demographics. The majority of birth mothers were White (70.1%), with 4.9% multiracial, 13.3% Black, 6.7% Latino, 2.5% American Indian or Alaskan Native, 1.8% Asian, .2% Native Hawaiian or Pacific Islander, and .5% did not report. The majority of birth fathers were White (69.9%), with 4.8% multiracial, 11.5% Black, 9.6% Latino, .5% American Indian or Alaskan Native, .5% Native Hawaiian or Pacific Islander, and 3.3% did not report. Our birth parents had on average a high school degree (Range: no high school degree to graduate degree), had a median income of < $15,000 (Range: <$15,000 to > $300,000), and were in their mid to late twenties at the time of the child's birth (birth mother: *M(SD)* = 24.35 (6.03); birth father: *M(SD)* = 26.08 (7.77).

Informed consent was obtained from all participants and the institutional review boards at each of the participating universities responsible for recruitment and assessment approved this study. Details about sample recruitment, demographics, and assessment can be found in Leve et al., 2019.

Measures

Birth parent temperament. We used latent temperament factors that were constructed for both birth mothers (BM) and birth fathers (BF) to estimate heritable influences (Shewark et al., *under review*). We used two factors: emotion dysregulation and agreeableness. Emotion dysregulation consisted of attentional control, activation control, fear and frustration subscales from the Adult Temperament Questionnaire – short form (ATQ; Rothbart et al., 2000). Agreeableness consisted of the sociability subscale from the ATQ, and the nurturance and intimate relationship subscales from the Harter

Adult Self-Perception Profile (Messer & Harter, 1986). Higher scores on emotion dysregulation were associated with higher levels of dysregulation with lower scores indicative of higher levels of attentional control and activation. Higher agreeableness indicates higher levels of sociability, nurturance, and positive intimate relationships. More information on the construction of these factors can be found in Shewark et al. In analyses, BM and BF factor scores were used and paths were constrained to be equal to provide a single heritable influence estimate for each temperament construct.

Birth mother prenatal distress. When the child was approximately 5 months old, birth mothers reported on their depressive and anxiety symptoms during pregnancy. Birth mothers completed shortened versions of the Beck Anxiety Inventory (BAI; Beck & Steer, 1993) and the Beck Depression Inventory (BDI; Beck & Steer, 1993) adapted for the pregnancy period. Mothers who endorsed sadness or anhedonia for at least a 2-week period during pregnancy were asked an additional 5 items from the BDI. Similarly, mothers were asked about anxiety symptoms with an additional 4 items from the BAI. Both subscales were on a 4-point Likert scale and had good reliability (BDI α = .86; BAI α = .80). Example items include: "I have been able to laugh and see the funny side of things" and "Things have been getting on top of me". Prenatal depressive symptoms and anxiety symptoms were combined by summing the scores (r = .55, p < .001).

Adopted child dysregulation. To measure child dysregulation, we used adoptive parent reports on the Attention-Deficit/Hyperactivity Problems subscale of the Child Behavior Checklist (Achenbach et al., 1987). This subscale includes 6 items on a scale from 1 (Not True) to 3 (Very True), for example, "Can't stand waiting; wants everything now". This subscale shows good reliability at 18 months (AP1 α =.85; AP2 α =.87) and

27 months (AP1 α =.77; AP2 α =.80). AP1 and AP2 scores were averaged at each timepoint to create composites (18 months: *r* = .39; 27 months: *r* = .42).

Adopted child reactivity. To measure child reactivity, we used parent reports on the anger proneness subscale of the Toddler Behavior Assessment Questionnaire (Goldsmith, 1996). This subscale consists of 28 items that assess the child's likelihood of presenting anger in situations, for example, "While shopping, if you did not agree to buy your child a toy that he/she wanted, how often did she/he: physically struggle when you tried to separate her/him from the toy." Each parent reported on these child behaviors at 18 (AP1 α =.89; AP2 α =.89) and 27 months (AP1 α =.85; AP2 α =.87). AP1 and AP2 scores were averaged at each timepoint to create composites (18 months: *r* = .42; 27 months: *r* = .44).

Adopted child social competence. Parent reports on child social competence at child age 4.5 years on the Social Skills Rating System (Gresham & Elliott, 1990) were used to measure social competence. The 39-item total social skills score was used (AP1 α =.87; AP2 α =.87), which includes items about cooperation, communication, responsibility, and self-control during interactions with peers. Example items include "Ends disagreements with you calmly" and Requests permission before leaving the house". Parent responds to items on a 3-point Likert scale. AP1 and AP2 scores were averaged to create a composite (*r* = .48, *p* < .001).

Covariates. Openness of adoption, child sex, and obstetric complications were included as covariates of all study variables. In addition, birth parent education, income, and age at child birth were examined as covariates of birth parent temperament and birth mother prenatal anxiety and depressive symptoms. Significantly related covariates were

controlled for in subsequent analyses by regressing them out of the study constructs and creating standardized z-scores.

Results

Descriptive and correlational results using raw data are presented in Table 4.1. To test our hypotheses, we conducted a structural equation model (SEM) using Mplus 8 (Muthen & Muthen, 2010). Models were estimated using full information maximum likelihood (FIML) estimation to reduce bias due to missing data (Graham, 2003). We used auxiliary variables to assist in better estimating missingness including: openness of adoption, obstetric complications, placement age, birth mother age, birth and adoptive family income, adoptive parent education, total kids in the adoptive family home, adoptive parent overreactivity, and adopted child Oppositional Defiant Disorder. We used four indexed criteria to assess model fit including chi-square ($p > .05$), CFI (> .90), SRMR (< .08), and RMSEA (< .08) (Kline, 2016). In addition to direct effects, we tested the indirect effects of BP temperament to child social competence through prenatal and child behavior constructs.

This model fit well (χ^2 (15) = 60.31, $p < .001$, RMSEA = .07, CFI = .97, SRMR = .04). Results (depicted in Figure 4.1) showed that higher levels of BP emotion dysregulation were associated with higher BMs prenatal distress, but BP agreeableness was not associated with BM prenatal distress. Additionally, higher levels of BP emotion dysregulation were associated with lower levels of child social competence at 4.5 years old. Prenatal distress was negatively marginally associated with adopted child reactivity at 18 months, but was not associated with any other child behavior at 18 and 27 months, or with social competence at 4.5 years. We found that higher levels of child reactivity and

dysregulation were associated with lower levels of social competence at child age 4.5 years. The indirect effect from birth parent emotion dysregulation to child reactivity through prenatal distress was marginal (β = -.014, *SE* = .01, *p* = .08). There were significant indirect effects from child dysregulation at 18 months to social competence through child dysregulation at 27 months (β = -.13, *SE* = .05, *p* = .004). This indirect effect was found for child reactivity as well (β = -.11, *SE* = .05, *p* = .03). Finally, there was a marginal effect from birth parent emotion dysregulation to child social competence at 4.5 years through child reactivity at 27 months (β = .02, *SE* = .01, *p* = .058).

Discussion

This was the first genetically informed study to examine the effect of prenatal distress on social competence, while also examining heritable influences. This study helps to advance our understanding of whether prenatal distress might influence children's positive social development. The findings suggest that there is a heritable influence on social competence at child age 4.5 years, but prenatal distress was not associated with social competence. There was only a marginal association of prenatal distress with child reactivity at 18 months and no indirect pathway of prenatal distress to social competence through earlier child reactivity or dysregulation. However, there were negative effects of child dysregulation and reactivity from 18 to 27 months on children's social competence at 4.5 years. Finally, there were heritable effects on prenatal distress. These findings together help us to better understand potential risk pathways for children's social competence in early childhood.

Contrary to our hypotheses, there were neither direct effects of prenatal distress on children's social competence, nor was there an indirect effect through children's early dysregulation. These null findings present a particularly interesting result that are situated in contrast to the majority of the prenatal literature to date. While there is work supporting the effect of prenatal distress on children's social competence and regulation (Carter et al., 2001b; Comaskey et al., 2017; Eichler et al., 2017; Feldman et al., 2009; Sharp et al., 2015), this is the first study to examine these relationships in conjunction with heritable influences. These null findings are, however; somewhat consistent with previous genetically informed studies examining the effects of the prenatal period on ADHD and externalizing behaviors (Gjerde et al., 2017; Rice et al., 2010). Therefore, these results suggest that it is possible that prenatal distress might not be associated with children's social competence or regulation once heritable influences are accounted for. Instead, these associations are associated with the genetic propensity to be higher (or lower) in social competence and regulation, as evinced by the association of BP emotion dysregulation with child social competence. We also see from these results that BP emotion dysregulation is associated with prenatal distress, highlighting that prenatal distress might be due to the genetic risk for dysregulation. Additionally, it is possible that the prenatal environment might act as a vehicle for the intergenerational transmission of this heritable propensity.

There was a marginal effect of prenatal distress on child reactivity at 18 months. Prenatal distress was not associated with any other child behavior (dysregulation or social competence). It is possible that the negative association found between prenatal distress and child reactivity, although marginal, might indicate an overly inhibited reaction

(reactive control) for those children exposed to more distress in the prenatal environment, which can lead to higher internalizing symptoms during childhood (Block & Block, 1980). However, interpretation of this association should be interpreted with caution.

This study confirms previous twin research that examined the heritability of child social competence (Edelbrock et al., 1995; Hudziak et al., 2003; Van Hulle et al., 2007). One previous study using the EGDS showed heritable influences on children's social competence at age 6 (Van Ryzin et al., 2015), and the current report shows that earlier social competence is also partially heritable. This is informative for research considering the role of the environment on early school readiness and social competence, prior to school entry, because heritable influences should be considered as a potential mechanism of effect to be accounted for.

This work also confirms previous work examining the influence of children's early regulatory capacity and reactivity, or lack of, on children's social competence in the preschool age. Mainly, the indirect effects of both child reactivity and dysregulation from infancy to toddlerhood to social competence at preschool age highlight an important developmental trajectory for understanding children's social development, after accounting for heritable influences. These findings also support previous studies highlighting the importance of these behaviors as pre-requisites or risk factors for children's social competence (Denham et al., 2003; Fabes & Eisenberg, 1999; Spinrad et al., 2006). These early regulatory processes allow for the child to practice the coordination of specific responses to their environment, for example, displaying appropriate emotions. In this study we examined the early child risk behaviors (reactivity and dysregulation), which indicates that children who are unable to appropriately regulate

and react to their environment could be on a risk trajectory that can negatively impact their readiness for school and interacting with their peers. This study not only supported prior work, but it supported this prior work even after accounting for potential genetic confounds, highlighting the importance of these early child behaviors for the development of social competence as an environmental influence. Therefore, more research should be done to understand these risk trajectories within genetically informed designs to further clarify mechanisms of influence (e.g., parenting).

Limitations

This study has a few limitations. First, the items in the prenatal distress construct were collected as part of a retrospective task. Therefore, it is possible that the mothers recall of their anxiety and depressive symptoms might not be as accurate compared to collecting this information from mothers during the prenatal period. However, this study used a modified Life History Calendar method (Caspi et al., 1996; Freedman et al., 1988) to collect this information in an attempt to help mothers to better remember this period. This method allows for mothers to indicate specific events on a calendar, which may include pregnancy related and non-related events to help them focus on the time frame being referenced. Previous EGDS work also suggests that mothers are reliable in remembering past prenatal experiences (Neiderhiser et al., 2016). Second, the child's reactivity and dysregulation were examined using adoptive parent reports on questionnaires. While this is a less precise measurement of reactivity and dysregulation, much of the prenatal literature uses parent reports on children's negative affect and impulsivity similarly to this study. Therefore, these measures are used in an attempt to better understand the associations found within the literature. Finally, this sample might

not be generalizable due to the unique nature of the birth mothers placing their children for adoption. Compared to the national estimates, the birth mothers in this study have a higher percentage of some substance use during pregnancy (e.g., smoking); however, this sample attended on average the recommended number of prenatal care visits (11), indicating that the experienced prenatal period might not be as risky as presumed (Kilpatrick & American Academy of Pediatrics, 2017).

Conclusions

In conclusion, this is the first study to examine the association between prenatal distress and social competence in a genetically informed design. Our study shows that the association between prenatal distress and children's social competence might be due to inherited characteristics instead of prenatal influences. While this study is the first to examine this association, it might be possible that prior prenatal studies have overestimated the effects of the prenatal environment due to the confound of shared genes between parents and children. However, these findings need to be examined further in other genetically informed designs to determine whether, similar to the smoking during pregnancy literature, this association is due to the genetic risk or the environmental impact of smoking (D'Onofrio et al., 2012; Knopik, 2009). This one finding in no way suggests that prenatal depressive symptoms and anxiety symptoms are not negatively influencing child development, but that researchers need to further understand and account for heritable influences that might be conflating risk. Overall, further assessing the relationship between prenatal influences and children's social competence in a genetically informed design is useful for understanding the mechanisms that impact social competence in preschool. This is particularly important as we think about the

importance of social competence for school readiness and later adjustment (Burt & Roisman, 2010; Jones et al., 2015; Masten & Coatsworth, 1998).

Table 4.1.
Means, standard deviations, and correlations using the raw study variables.

Construct	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.
1. BM Emotion Dysregulation	1									
2. BM Agreeableness	-.48**	1								
3. BF Emotion Dysregulation	.33**	-.58**	1							
4. BF Agreeableness	-.35**	.45**	.19*	1						
5. Prenatal Distress	.28**	-.22**	.12**	-.06	1					
6. AC Reactivity B	-.02	-.03	-.03	.05	-.09+	1				
7. AC Reactivity C	-.01	-.07	.06	-.05	-.04	.69**	1			
8. AC Dysregulation B	.01	-.09*	.01	.00	-.02	.37**	.35**	1		
9. AC Dysregulation C	.06	-.09*	.01	.00	.00	.29**	.37**	.67**	1	
10. AC Social Competence	-.08+	.12*	-.12*	.08	-.01	-.27**	-.29**	-.21**	-.30**	1
Mean	0.00	0.00	0.00	0.00	8.65	3.39	3.58	5.11	4.56	49.04
SD	.82	.77	.52	.53	7.43	.63	.61	2.01	2.18	7.84

Note. BM = birth mother, BF = birth father, AC = adopted child, B=Wave B, C =Wave C. + $p < .06$, * $p <. 05$, ** $p < .01$.

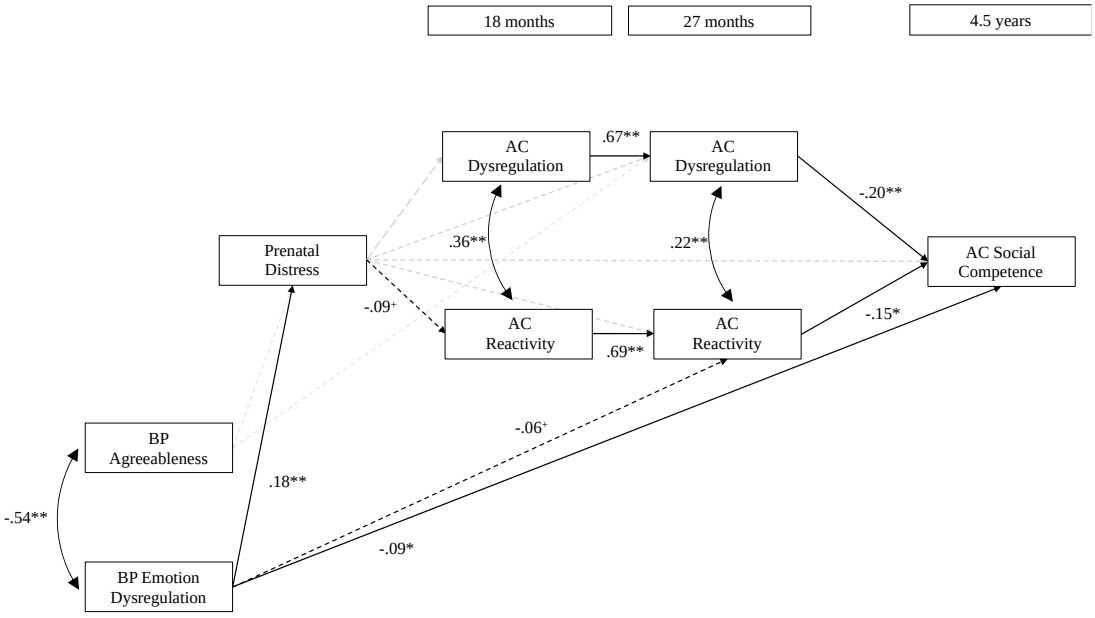

Figure 4.1. Modeling examining genetic and prenatal influences on social competence.

Note: Non- significant paths are dotted grey lines, significant paths are solid black lines, marginal paths are dotted black lines. BP = birth parents, AC = adopted child. + $p < .06$, * $p < .05$, ** $p < .01$.

Chapter 5

Family Interactions in Toddlerhood Influence Social Competence in Preschool Age: Accounting for Heritable and Prenatal Influences

Authors: Amanda M. Ramos, Elizabeth A. Shewark, David Reiss, Leslie D. Leve, Misaki N. Natsuaki, Daniel S. Shaw, Jody M. Ganiban, and Jenae M. Neiderhiser

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Abstract

Identification of early promotive and risk factors for social competence is not only important for fostering children's successful social development but is also essential for children's later academic and psychological well-being. While we know potential mechanisms of influence (e.g., parenting, prenatal, genetics), there is less work exploring how these different influences work together to impact children's social competence. Using a genetically informed sample, the Early Growth and Development Study (N = 561), we examined multiple levels of influence (i.e., heritable, prenatal, parenting, and child-driven effects) on social competence in children at 4.5 years old. Results from structural equation models showed that there were heritable, early child behavior, and parenting effects on child social competence at 4.5 years. Specifically, we found that adoptive mother overreactivity at child age 18 months was positively associated with child dysregulation at 27 months, which in turn was associated with lower levels of social competence at 4.5 years. Additionally, we found that child reactivity at 18 months was associated with adoptive mother overreactivity at 27 months, which was associated with

lower levels of social competence at 4.5 years. Finally, we found both an evocative rGE effect on adoptive fathers' overreactivity at 18 months, but also that prenatal distress was negatively associated with adoptive fathers overreactivity at 18 months. This study first shows that children's interactions with their parents are important for the development of social competence, and that this unfolds across infancy and toddlerhood. Finally, the examination of heritable, prenatal, and postnatal environmental influences simultaneously allows us to better examine the complex nature of how social competence develops in early childhood.

Introduction

Children's social competence not only has immediate impacts on children's academic and overall wellbeing, but also throughout their lifetime, highlighting children's ability to appropriately engage in their social world as a crucial skill to master for successful lifelong adjustment (Blandon et al., 2010; Jones et al., 2015; Masten & Coatsworth, 1998). While the literature has focused on exploring how parenting influences the development of social competence (Fabes et al., 2006; Karreman et al., 2006), prenatal (Bandstra et al., 2010; Jones et al., 2013; Kully-Martens et al., 2012; Rice et al., 2010), heritable (Battaglia et al., 2017; Knafo & Plomin, 2006b), and even child effects (Eisenberg et al., 2010) are often disregarded or not considered in tandem. Although research supports all of these influences on the development of social competence, there is no work examining the more complex interconnected processes that these multilevel factors might have on one another to influence the development of social competence in early childhood. Therefore, the focus of the current study was to use a genetically informed design to disentangle heritable, prenatal, parenting, and child-driven effects that together shape children's social competence in early childhood.

Parenting Influences on Children's Social Competence

In early childhood, developmentally salient parenting behaviors like responsiveness and warmth, as well as overreactive and hostile parenting, have been found to be important promotive and hindrance factors for the development of social competence (Clark & Ladd, 2000; Driscoll & Pianta, 2011; Feldman & Masalha, 2010; Haskett & Willoughby, 2007; Masten & Coatsworth, 1998). These different parenting behaviors are assumed to influence child development because they serve unique

functions to socialize the child. For example, parents' emotional responsivity to their child's needs is presumed important for facilitating the development of a secure sense of self in the child (Thompson, 2000; Waters & Cummings, 2000). Longitudinal and concurrent studies suggest the importance of parental responsivity for children's development of social competence (Feldman & Masalha, 2010; Leerkes et al., 2009; Martin et al., 2010; Raby et al., 2015; Rispoli et al., 2013). One longitudinal study found that parental responsivity toward their infant was associated with higher levels of social competence in the preschool-aged child (Rispoli et al., 2013). On the other hand, overreactive and hostile parenting (i.e. yelling, criticizing, and harsh punishment) is assumed to be detrimental to the development of the child's confidence, by modeling negative interaction styles that they act out with their peers (Anthony et al., 2005; Patterson, 1982). Concurrent and longitudinal studies have found that hostile parenting was associated with lower levels of social competence and fewer prosocial skills in preschool-aged children (Anthony et al., 2005; Eddy et al., 2001; Hartas, 2011; Laible et al., 2004; Walker & MacPhee, 2011). This parenting literature is based on assumptions that parenting influences child outcomes because parents are socializing their child, and this is the major influence on the child. However, these "environmental" mechanisms studied by researchers are potentially biased due to their inability to account for shared genes, thereby inflating the estimates of the associations. Therefore, while these parenting constructs are assumed to influence child social competence, more work is needed to explore parenting outside of the vacuum of parenting as a "pure" environmental influence.

In addition, most research examining parenting focuses on specific types of parenting (i.e., negative or positive), with fewer studies focused on which parenting behaviors might be most influential for the development of social competence. Parenting behavior is not unidimensional and children experience positive and negative parenting behaviors sometimes within the same interaction. To understand the dynamic nature of parenting it is important to consider both positive and negative parenting simultaneously (Hartas, 2011; Laible et al., 2004; Lengua et al., 2007; Rispoli et al., 2013). One cross-sectional study examining warm and harsh parenting of preschool-aged children found that harsh parenting was associated with child externalizing behaviors, while warm parenting was associated with child social competence (Laible et al., 2004). A longitudinal study found that parental responsivity at 9 months and parental supportiveness with preschoolers was associated with child social competence in preschool, while parental negativity at 2 years was not associated with child social competence in preschool (Rispoli et al., 2013). However, a different longitudinal study found that parental negativity at age 3 was associated with lower prosocial behaviors in children at age 5, after accounting for the effects of sensitive parenting (Hartas, 2011). This inconsistency across studies may be due in part to the differences in the child ages or to the different study designs (e.g., concurrent versus longitudinal). It is also possible that certain parenting behaviors are more salient at different developmental periods for the development of social competence in early childhood.

Additionally, much of this literature is dominated by examining mother-child dyads, to the detriment of understanding the role of fathers. Fathers could, for example, provide additional support to mothers, or influence the child directly, or most likely,

some combination of the two (Cabrera et al., 2014; Lamb, 2004). One study found that fathers', not mothers', mutual responsivity with their child was associated with children's social competence in early childhood (Feldman et al., 2013). However, much of the work on father behavior focuses on involvement and support (Jia et al., 2012), but they also can provide unique ways for young children to be socialized, expressed with the type of play fathers engage in to the external activities they include their children in (Cabrera & Tamis-LeMonda, 2013). Therefore, this study will be incorporating fathers in an attempt to fully understand the role of the family system during this young age, particularly to better understand how parenting contributes to child social competence.

Child-Driven Effects

Parent-based models rely on the assumptions that it is a parent-driven process; however, other work contends that family process is transactional in nature (Bell, 1968; Kiff et al., 2011; Patterson, 1982). Specifically, parents respond to the child's needs and demands, and the child responds to the parents' requests and bids and vice versa. During early childhood, regulation and reactivity may be key early child behaviors that evoke behaviors from parents as these are some of the first behaviors exhibited by the child (Belsky, 1984; Kiff et al., 2011). Regulation consists of multidimensional components, like behavior and emotions, that allow the child to coordinate responses to situational cues (McClelland & Cameron, 2012). In comparison, reactivity is an individual's response to their environment with emotional reactions and encompasses high intensity negative emotions (DiPietro et al., 2008; Rothbart & Bates, 2006). Higher levels of regulation and less reactivity are considered prerequisites for the development of social competence because regulatory abilities help children to appropriately respond to their

peers, and children who are overly negative (e.g., sad, angry) have difficulty keeping good quality friendships (Denham et al., 2003; Diener & Kim, 2004; Korja et al., 2017; Rubin et al., 1995). Studies have shown that children's early reactivity and dysregulation are associated with increases in parents negative parenting (Bridgett et al., 2009; Morrell & Murray, 2003) , and decreases in parents positive parenting (Braungart-Rieker et al., 2001; Eisenberg et al., 2010). Work has also suggested that these early interactions influence the development of social competence (Eisenberg et al., 2010; Fabes et al., 2001; Smith et al., 2014). For example, one study found that when parents used harsh parenting strategies in response to negative emotions from their child, they had children who were more dysregulated and in turn were less socially competent (Fabes et al., 2001). These negative interactions between parents and children are considered a coercive cycle that can elicit further negativity within the dyad (Patterson, 1982; Smith et al., 2014). Therefore, not only does parenting have a direct and indirect effect on children's social competence through regulation and reactivity, but children's early behaviors could similarly impact social competence through eliciting particular parental responses. Without accounting for the child-effects, previous parenting literature is unable to clearly delineate whether the parenting behavior is the guiding environmental force or a potentially developmentally salient response elicited by the child. In an attempt to consider the whole family, this work will incorporate child-driven effects as a crucial factor that impacts the development of social competence.

Interplay of Genetic and Rearing Environmental Effects

These child-driven effects on parenting may be operating through evocative gene-environment correlation (*r*GE) (Knopik et al., 2017; Plomin et al., 1977; Scarr &

McCartney, 1983). In other words, the effect of the child in the way they are parented may be due, in part, to inherited characteristics or behaviors of the child. Evocative *r*GE occurs when individuals evoke particular responses from their environment that are correlated with their inherited characteristics (Knopik et al., 2017; Plomin et al., 1977; Scarr & McCartney, 1983). Most family studies are confounded and are unable to separate these influences because parents and children share genes and the parents also provide the rearing environment (Moore & Neiderhiser, 2014). Many genetically informed studies have found that evocative *r*GE influences parenting (e.g., Hajal et al., 2015; Harold et al., 2013; Klahr & Burt, 2014). One meta-analysis found that the moderate heritability estimates decreased over time from childhood to adolescence for negative parenting, but were stable over time for positive parenting (Klahr & Burt, 2014). These findings suggest that infancy and toddlerhood is a particularly important time for examining evocative *r*GE effects.

Twin designs have shown that early regulation, reactivity, and social competence are partially heritable (Clifford et al., 2015; Edelbrock et al., 1995; Hudziak et al., 2003; Roisman & Fraley, 2012; Van Hulle et al., 2007). This makes these behaviors crucial to examine heritable influences as a potential mechanism through evocative *r*GE that impacts early parenting and social competence. Twin and adoption studies have found that child regulatory behaviors and emotions in infancy and toddlerhood evoke parenting (Hajal et al., 2015; Harold et al., 2013; Klahr et al., 2013, 2017; Natsuaki et al., 2013; Ulbricht et al., 2013). For example, child negative emotionality from infancy to toddlerhood was positively associated with higher levels of parent overreactivity from infancy to toddlerhood, which was partially explained by heritable influences (Lipscomb

et al., 2011). Other studies have shown that evocative effects on parenting in toddlerhood and preschool age were associated with higher levels of child disruptive behaviors (Elam et al., 2014) and prosocial behaviors (Knafo & Plomin, 2006a). Due to the larger evocative effects occurring in early childhood, examining early child behaviors within a genetically informed design can help us to further understand how bidirectional family processes occur. Additionally, these findings support the contribution of the child on the family and provide evidence that heritable influences may be one mechanism to explain how development unfolds.

Prenatal Influences on Children's Social Competence

Outside of family interactions in early childhood, another important sensitive period to consider is the prenatal environment (Behnke et al., 2013; Graignic-Philippe et al., 2014; Latimer et al., 2012; Rice et al., 2007). In particular, prenatal distress (i.e., anxiety and depression) has been associated with higher levels of child emotional and behavioral problems, and with lower levels of social competence in early childhood (Carter et al., 2001a; Dunkel Schetter & Tanner, 2012; Eichler et al., 2017; Glover, 2014; Loomans et al., 2011; O'Connor, Heron, Golding, et al., 2002). In addition, prenatal distress has been negatively associated with earlier child behaviors that have been associated with child social competence (Blair et al., 2011; Feldman et al., 2009; Korja et al., 2017; Sharp et al., 2015). One theoretical explanation for this potential mediated pathway is the fetal programming hypothesis (Barker, 1998). This hypothesis proposes that events that happen during the prenatal period can influence fetal development through reprogramming neural networks (Barker, 1998; Ping et al., 2015). Therefore, the prenatal environment might negatively alter the child's neural network, in the womb, that

starts the child at a higher threshold for being emotional or dysregulated, which in turn has the potential to impact their normative developmental trajectory. This hypothesis is supported by studies that have found that prenatal distress is associated with higher levels of negative affect and irritability in infants and toddlers (Davis et al., 2007; DiPietro et al., 2008; Glynn et al., 2018). Additionally studies have found that dysregulation or reactivity mediated the relationship between prenatal distress and later social development (Blair et al., 2011; Carter et al., 2001a; DiPietro et al., 2008). However, this work occurs almost exclusively within family studies, with biological parents rearing their biological children. As such, this literature is unable to distinguish prenatal effects from heritable and postnatal influences, increasing the likelihood that these associations between the prenatal environment and child behavior might be biased. Therefore, this work will use a genetically informed design in an attempt to better estimate heritable, prenatal, and postnatal environmental influences.

Genetic-prenatal interplay. There has been an overwhelming focus on child negative social behaviors (e.g., externalizing problems) in research using genetically informed designs that include prenatal effects. For example, one parent-offspring adoption study found that birth mother depressive symptoms (heritable influence) was associated with toddler externalizing behaviors through prenatal risk (Pemberton et al., 2010). However, the genetically informed literature on the influence of prenatal anxiety and depressive symptoms on children's behavior is mixed, with some studies finding that prenatal stress mediates the association between heritable influences and child behaviors (Kerr et al., 2013; Marceau, Hajal, et al., 2013; Rice et al., 2010), while other studies have shown there is no effect of prenatal stress on child behaviors after controlling for

heritable influences (Gjerde et al., 2017; Hannigan et al., 2018; Rice et al., 2010). Therefore, more work needs to be done to understand the complex nature of the influence of the prenatal environment, and postnatal environment, while accounting for heritable influences.

The Current Study

The present study is one of the first studies to examine multiple levels of influence associated with the development of social competence in young children by considering the effects of heritable, prenatal, and postnatal environmental influences. There are a limited number of studies that have considered all three levels of development: genetic, prenatal, and postnatal and these few studies (Marceau et al., 2015; Marceau et al., 2013; Neiderhiser et al., 2016) have found different associations with child outcomes that could be dependent on the prenatal influences used. The present study used a longitudinal adoption design of adopted children, adoptive parents, and birth parents that allows postnatal and heritable influences to be teased apart, because the birth parents provide only the heritable influences (and prenatal environment for birth mother) and the adoptive parents provide the postnatal environment. It is also possible to distinguish heritable from prenatal influences when birth father information is included in the model (D'Onofrio et al., 2013; Loehlin, 2016). In this report, we examine how heritable influences and toddler regulation and reactivity are associated with adoptive parents' positive and negative parenting of toddlers, that in turn are associated with child social competence at age 4.5 years. We hypothesized that (1) birth parent (BP) emotion dysregulation would be positively associated with parents' overreactivity and negatively associated with parents' responsivity, which in turn would negatively and positively

influence, respectively, child social competence at 4.5 years and (2) BP agreeableness would be positively associated with adoptive parents' responsivity, which in turn would positively influence child social competence at 4.5 years. In addition, we hypothesized that (3) BP temperament would be associated with parenting behaviors via child dysregulation/reactivity, supporting evocative *r*GE effects. Finally, we expected that (4) there would be a bidirectional process between child dysregulation/reactivity and parenting during toddlerhood that in turn would influence child social competence at age 4.5 years. We hypothesized that child dysregulation would be associated with lower levels of parent responsiveness and higher levels of coercive parenting, resulting in lower levels of social competence in children at preschool age.

Method

Participants

Participants were from the Early Growth and Development Study (EGDS), a longitudinal adoption design, with 561 linked sets of adopted children, adoptive parents, and birth parents (Leve et al., 2019). Recruitment of these families occurred through 45 adoption agencies in 15 states, and families were eligible to participate if the adoption was domestic, placement occurred within 3 months (*M* = 5.58 days, *SD* = 11.32 days), the child was placed with a nonrelative, the child had no major medical conditions, and the birth parents and adoptive parents could understand English at an 8th grade level. The adopted children were majority male (57.3%), and about half Caucasian (55.6%), with 19.3% multiethnic, 13% African American, and 10.9% Hispanic. The mean age of placement for the child was 6.19 days (*SD* = 12.45 days). Demographic information about the adoptive parents and birth parents are provided in Table 5.1. Birth parents

(BPs) were generally younger than adoptive parents (APs), had less education, and lower income. For this study, same-sex couples and single parents were removed from the analyses.

Measures

Birth parent temperament. We used latent temperament factors that were constructed for both birth mothers (BMs) and birth fathers (BFs) to estimate heritable influences previously created (Shewark et al., *under review*). We used the following factors: emotion dysregulation and agreeableness. Emotion dysregulation consisted of attentional control, activation control, fear and frustration subscales from the Adult Temperament Questionnaire – short form (ATQ; Rothbart et al., 2000). Agreeableness consisted of the sociability subscale from the ATQ, and the nurturance and intimate relationship subscales from the Harter Adult Self-Perception Profile (Messer & Harter, 1986). Higher scores on the emotion dysregulation factor are indicative of higher levels of dysregulation with lower scores indicative of higher levels of attentional control and activation. Higher agreeableness indicates higher levels of sociability, nurturance, and positive intimate relationships. More information on the construction of these factors is provided in Shewark et al., *under review*. In analyses, BM and BF factor scores were used and paths were constrained to be equal to provide a single heritable influence estimate for each temperament construct.

Birth mother prenatal distress. When the child was approximately 5 months old, birth mothers reported on their depressive and anxiety symptoms during pregnancy. Birth mothers completed shortened versions of the Beck Anxiety Inventory (BAI; Beck & Steer, 1993) and the Beck Depression Inventory (BDI; Beck & Steer, 1993) adapted

for the pregnancy period. Mothers who endorsed sadness or anhedonia for at least a 2-week period during pregnancy were asked an additional 5 items from the BDI. Similarly, mothers were asked about anxiety symptoms with an additional 4 items from the BAI. Both subscales were on a 4-point Likert scale and had good reliability (BDI α = .86; BAI α = .80). Example items include: "I have been able to laugh and see the funny side of things" and "Things have been getting on top of me". Prenatal depressive symptoms and anxiety symptoms were combined by summing their scores (r = .55, p < .001).

Adopted child dysregulation. To capture dysregulation, we used adoptive parent reports on the Attention-Deficit/Hyperactivity Problems subscale of the Child Behavior Checklist (Achenbach et al., 1987). This subscale includes 6 items on a scale from 1 (Not True) to 3 (Very True), for example, "Can't concentrate". This subscale shows good reliability at 18 months (AM α =.85; AF α =.87) and 27 months (AM α =.77; AF α =.80). AM and AF scores were averaged to create composites (18 months: r = .39; 27 months: r = .42).

Adopted child reactivity. To capture reactivity, we used parent reports on the anger proneness subscale of the Toddler Behavior Assessment Questionnaire (Goldsmith, 1996). This subscale consists of 28 items that assess the child's likelihood of presenting anger in situations, for example, "When you did not allow your child to do something for her/himself, for example, dressing or getting into the car seat, how often did your child try to push you away?" Each parent reported on these child behaviors at 18 (adoptive mother (AM) α =.89; adoptive father AF) α =.89) and 27 months (AM α =.85; AF α =.87). AM and AF scores were averaged to create composites (18 months: r = .42; 27 months: r = .44).

Adoptive parents parenting. We assessed both positive and negative parenting behaviors at 18 and 27 months. For *positive parenting behaviors*, we assessed parents' responsive parenting with the Home Observation for Measurement of the Environment (HOME) Inventory. The HOME was designed to measure the emotional and verbal responsiveness of the parent to the child. This assessment was completed by the study interviewers upon the completion of the in-home interview. The responsivity subscale consists of 11 items. An example item is: "mother/father responds to the child's vocalizations with verbal or vocal response." This measure showed good reliability for both parents at both waves (18 months: adoptive mother α = .76, adoptive father α =.71; 27 months: adoptive mother α =.58, adoptive father α = .70). Responsivity was negatively skewed and we reflected this variable and then performed a reciprocal transformation all with the intention of keeping the high scores meaning higher responsivity.

For *negative parenting behaviors,* parents overreactivity was assessed using self-reports on the Parenting Scale (Arnold et al., 1993) at 18 and 27 months. We used the overreactivity subscale, which reflects displays of anger, meanness, and irritability. An example item is: "When my child misbehaves, I get so frustrated or angry that my child can see that I am upset". This subscale consisted of 10 items on a 7-point Likert scale and showed good reliability for both parents across both timepoints (18 months: adoptive mother α = .79, adoptive father α =.77; 27 months: adoptive mother α =.79, adoptive father α = .77).

Adopted child social competence. We used parents report on child social competence at child age 4.5 on the Social Skills Rating System (Gresham & Elliott, 1990). We used the total social skills score that includes 39 items (AM α =.87; AF α =.87), which includes items about cooperation, communication, responsibility, and self-control during interactions with peers. Example items include "Ends disagreements with you calmly" and "Requests permission before leaving the house". Parent responds to items on a 3-point Likert scale. AM and AF scores were averaged to create a composite (r = .48, p < .001).

Covariates. Openness of adoption, child sex, and obstetric complications (e.g., prenatal complications, neonatal complications, substance use), parent age at the child's birth, education, and income were tested as covariates on all study variables. Significantly related covariates were controlled for in subsequent analyses by regressing them out of the study constructs and creating standardized z-scores.

Analytic strategy. Hypotheses were tested using structural equation models in Mplus 8 (Muthen & Muthen, 2010). Four models (BP emotion dysregulation-adopted child dysregulation, BP emotion dysregulation-adopted child reactivity, BP agreeableness-adopted child dysregulation, and BP agreeableness-adopted child reactivity) were estimated using full information maximum likelihood (FIML) estimation to reduce bias of missing data (Graham, 2003). Fit statistics were used to examine the fit of the models including, Chi-square goodness of fit index (p > .05), CFI (.90 or above), SRMR (less than .08), and RMSEA (less than .08). Main effects as well as indirect effects were examined within each model. Birth mother and birth father emotion

dysregulation and agreeableness paths were constrained to be equal in the models to estimate a single heritable influence.

Results

Descriptive statistics and correlations among the raw study variables are presented in Table 5.2. Correlations reflect that adoptive parents overreactivity was significantly associated with early child regulation, reactivity, and social competence with moderate effect sizes (*r*'s range from -.25 to .30). Also, child early dysregulation and reactivity were significantly associated with later social competence (*r*'s range from -.19 to -.28). Finally, birth parent temperament was significantly correlated with social competence at 4.5 years (*r* = -.12 - .12).

Birth Parent Emotion Dysregulation Models

Both models fit well: child dysregulation (χ^2 (25) = 29.82, p = .23, RMSEA = .02, CFI = 1.00, SRMR = .02), and child reactivity (χ^2 (26) = 29.15, p = .30, RMSEA = .02, CFI = 1.00, SRMR = .02). Results (depicted in Figure 5.1) indicate that birth parent emotion dysregulation was negatively associated with child social competence at 4.5 years. In addition, both adoptive mothers' overreactivity and adoptive mothers' responsivity was associated with child social competence (β = -.22, p < .05; β = .13, p < .05, respectively), but adoptive fathers parenting was not associated with child social competence. Adopted child dysregulation was also negatively associated with child social competence. Finally, we found that birth parent emotion dysregulation was positively associated with prenatal distress and that prenatal distress was negatively associated with adoptive fathers' overreactivity at 18 months.

We found differences when examining the bidirectional effects of child dysregulation and parenting and child reactivity and parenting. For the *child dysregulation model* (Figure 5.1A), we found that adoptive mothers' overreactivity at 18 months was positively associated with child dysregulation at 27 months; however, adoptive fathers' overreactivity was not associated with child's dysregulation. Additionally, adoptive fathers' responsivity was positively associated with child dysregulation at 27 months, but not adoptive mothers' responsivity. We also found that adoptive mothers' responsivity at 18 months was negatively associated with adoptive fathers' responsivity at 27 months. Additionally, child dysregulation at 18 months was not associated with adoptive parents' parenting at 27 months. For the *child reactivity model* (Figure 5.1B), we found that adoptive parents' parenting at 18 months was not associated with children's reactivity at 27 months. However, we found that child reactivity at 18 months was positively associated with adoptive mothers' overreactivity at 27 months; however, child reactivity was not associated with other parenting behaviors. We also found that adoptive mothers' responsivity at 18 months was negatively associated with adoptive fathers' responsivity at 27 months.

Indirect effects. Overall, there was an indirect effect from adoptive mothers' overreactivity at 18 months to child social competence at 4.5 years through adoptive mothers' overreactivity at 27 months ($\beta = -.15$, $SE = .05$, $p < .01$). There was a marginally significant indirect effect from birth mother emotion dysregulation to adoptive fathers' overreactivity through prenatal distress ($\beta = -.01$, $SE = .01$, $p = .06$). For the *child dysregulation model,* adoptive mothers' overreactivity at 18 months to child social competence at 4.5 years was significant through child dysregulation at 27 months (β = -

.02, *SE* = .01, *p* = .05). Additionally, there was an indirect effect of child dysregulation at 18 months to child social competence through child dysregulation at 27 months (β = -.11, *SE* = .04, *p* < .05). Finally, adoptive fathers' responsivity to child social competence was not significant through child dysregulation at 27 months (β = -.02, *SE* = .01, *p* = .09). There were no additional significant evocative *r*GE pathways in this model. For the *child reactivity model,* child reactivity at 18 months was associated with child social competence at 4.5 years through adoptive mother overreactivity at 27 months (β = -.02, *SE* = .01, *p* = .05). However, because birth parent emotion dysregulation was not associated with adopted child reactivity, there were no additional significant evocative *r*GE pathways.

Birth Parent Agreeableness Models

Both of the models fit well: child dysregulation (χ^2 (25) = 26.34, *p* = .39, RMSEA = .01, CFI = 1.00, SRMR = .04) and child reactivity (χ^2 (26) = 23.51, *p* = .60, RMSEA = .00, CFI = 1.00, SRMR = .02). Results (depicted in Figure 5.2) showed that birth parent agreeableness was positively associated with child social competence at 4.5 years old. Birth parent agreeableness was also negatively associated with child reactivity at 27 months, but not child dysregulation. Birth parent agreeableness was also negatively associated with prenatal distress and adoptive fathers' overreactivity at 18 months. We also found that prenatal distress was negatively associated with adoptive fathers' overreactivity at 18 months. In addition, both adoptive mothers' overreactivity and responsivity was associated with child social competence (β = -.22, *p* < .05; β = .13, *p* < .05, respectively), but adoptive fathers' parenting behaviors were not associated with

child social competence. Child dysregulation during toddlerhood was negatively associated with child social competence.

When examining the bidirectional effects of parents to children, we found for the *child dysregulation model* (Figure 5.2A) that adoptive mothers' overreactivity at 18 months was positively associated with child dysregulation at 27 months; however, adoptive fathers' overreactivity was not associated with child dysregulation. Additionally, adoptive fathers' responsivity was positively associated with child dysregulation at 27 months, but not adoptive mothers' responsivity. We also found that adoptive mothers' responsivity at 18 months was negatively associated with adoptive fathers' responsivity at 27 months. Additionally, child dysregulation at 18 months was not associated with adoptive parents' parenting at 27 months. For the *child reactivity model* (Figure 5.2B), we found that adoptive parents' parenting at 18 months was not associated with child reactivity at 27 months. However, we found that child reactivity at 18 months was positively associated with adoptive mothers' overreactivity at 27 months; however, child reactivity was not associated with other parenting behaviors. We also found that adoptive mothers' responsivity at 18 months was negatively associated with adoptive fathers' responsivity at 27 months.

Indirect effects. Overall, there was an indirect effect from adoptive mothers' overreactivity at 18 months to child social competence at 4.5 years through adopted mothers' overreactivity at 27 months ($\beta = -.15$, $SE = .05$, $p < .01$). However, the indirect effect from birth mother emotion dysregulation to adoptive fathers' overreactivity through prenatal distress was marginally significant ($\beta = .01$, $SE = .01$, $p = .06$). For the *child dysregulation model,* adoptive mothers' overreactivity at 18 months was associated

with child social competence through child dysregulation at 27 months (β = -.02, *SE* = .01, *p* = .05). Additionally, there was an indirect effect of child dysregulation at 18 months to child social competence through child dysregulation at 27 months (β = -.11, *SE* = .04, *p* < .05). Finally, adoptive fathers' responsivity to child social competence was not significant through child dysregulation at 27 months (β = -.02, *SE* = .01, *p* = .09). For the *child reactivity model,* child reactivity was associated with child social competence at 4.5 years through adoptive mothers' overreactivity at 27 months (β = -.02, *SE* = .01, *p* = .05). However, there were no additional significant evocative *r*GE pathways.

Table 5.1. Sample Descriptives

	Adoptive Parent 1	**Adoptive Parent 2**	**Birth Mother**	**Birth Father**
Age at child birth [*M (SD)*]	37.43 (5.59)	38.30 (5.83)	24.35 (6.03)	26.08 (7.77)
Race/Ethnicity				
White	91.8%	90.4%	70.1%	69.9%
African American	3.9%	4.9%	13.3%	11.5%
Hispanic	2%	1.6%	6.7%	9.6%
More than 1 race	.4%	1.1%	4.9%	4.8%
Asian	.9%	.5%	1.8%	0%
American Indian or Alaskan	.2%	0%	2.5%	.5%
Native	0%	.5%	.2%	.5%
Native Hawaiian or Pacific	.4%	.9%	.5%	3.3%
Islander				
Other				
Income (median)	$100,000-$125,000		<$15,000	$15,000-$40,000
Education (median)	College Degree		High School Degree	

Table 5.2. Correlations and Mean and Standard Deviations of the Raw Study Variables.

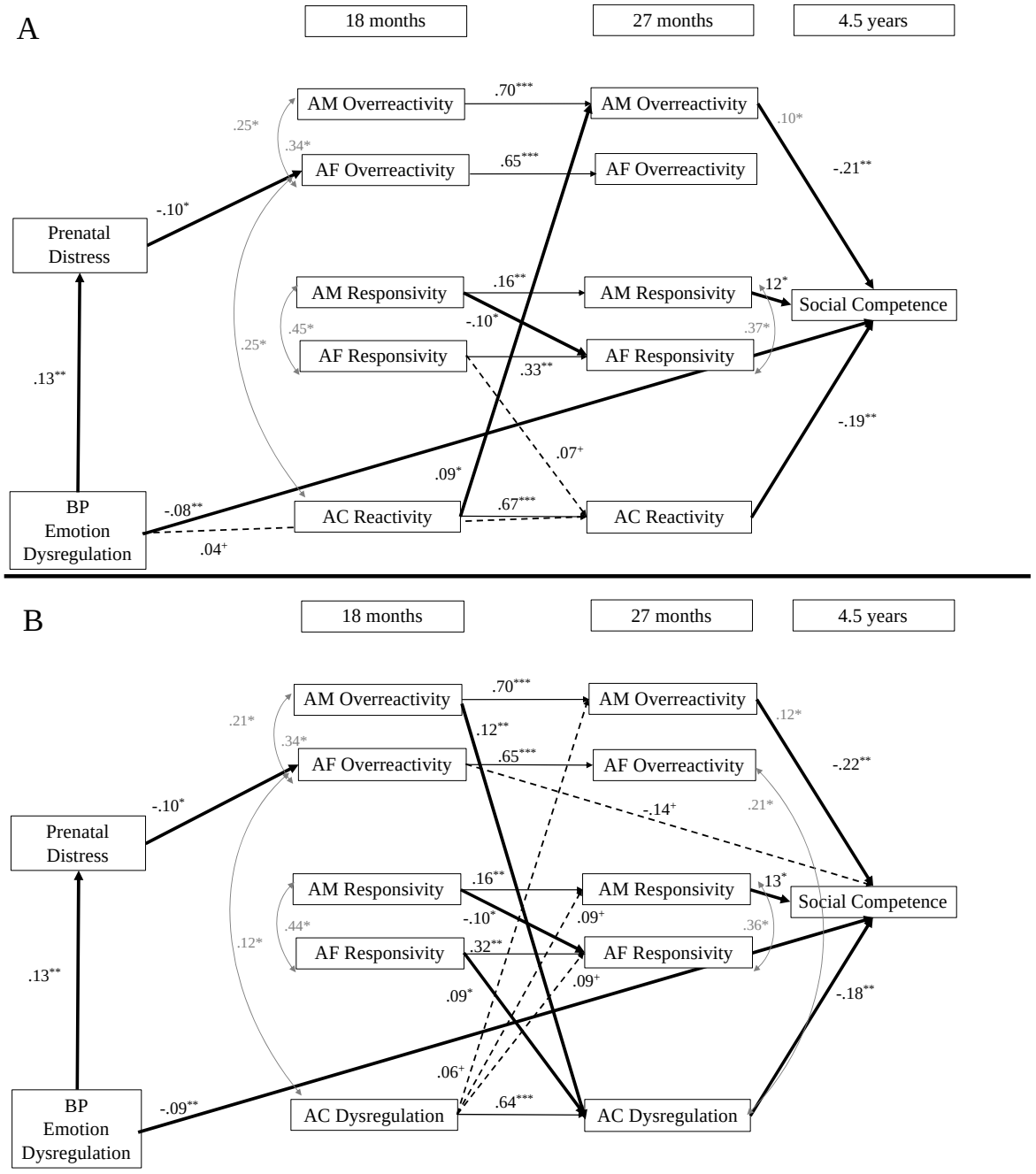
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1.BP Emotion Dysregulation	--	-.67**	.11*	-.02	.05	.03	-.02	.01	-.04	-.01	.03	-.01	-.08	.07	-.01	-.12*
2. BP Agreeableness	-.47**	--	-.05	-.02	-.10*	-.02	.02	-.01	.04	.01	-.04	-.07	.02	-.06	-.01	.08
3. Prenatal Distress	.25**	-.21**	--													
4. AM Overreactivity, 18 mos	-.01	-.03	-.05	--												
5. AF Overreactivity, 18 mos	-.03	-.07	-.10*	.34**	--											
6. AM Responsivity, 18 mos	.03	-.01	.01	-.02	-.01	--										
7. AF Responsivity, 18 mos	.01	.04	-.07	-.01	.01	.54**	--									
8. AC Dysregulation, 18 mos	.00	-.08	-.02	.22**	.12**	.05	.08	--								
9. AC Reactivity, 18 mos	-.03	-.02	-.09+	.24**	.24**	.02	.03	.36**	--							
10.AM Overreactivity, 27 mos	-.04	.06	-.07	.73**	.27**	.02	.03	.24**	.26**	--						
11.AF Overreactivity, 27 mos	-.02	-.03	-.11**	.27**	.69**	-.05	-.05	.16**	.24**	.27**	--					
12. AM Responsivity, 27 mos	.01	-.02	.00	-.07	-.06	.23**	.22**	.10*	-.05	.02	-.02	--				
13. AF Responsivity, 27 mos	.01	.04	-.07	-.04	-.04	.10**	.27**	.10*	-.01	-.01	.00	.37**	--			
14. AC Dysregulation, 27 mos	.04	-.08	-.01	.27**	.10**	.02	.10+	.67**	.26**	.27**	.24**	.04	..07	--		

15.AC Reactivity, 27 mos	.00	-.08	-.04	.23**	.24**	.05	.05	.35**	.68**	.30**	.25**	-.02	-.01	.37**	--	
16. AC Social Competence 4.5 years	-.07	.12*	.00	-.23**	-.18**	.03	.02	-.19**	-.27**	-.25**	-.12*	.14**	.02	-.27**	-.28**	
Mean	.00	.00	8.70	1.86	1.90	10.58	10.05	5.13	3.40	2.07	2.06	10.70	10.27	4.59	3.58	48.87
Standard Deviations	.83	.77	7.43	.60	.60	1.14	1.57	2.03	.62	.62	.62	.78	1.43	2.13	.60	7.90

Note: BP = Birth mother correlations are shown only, AM = adoptive mother, AF = adoptive father, AC = adopted child. + $p <$ .06, * $p < .05$, ** $p < .001$. First two column correlations are for BM, first two row correlations are for BF.

Figure 5.1.

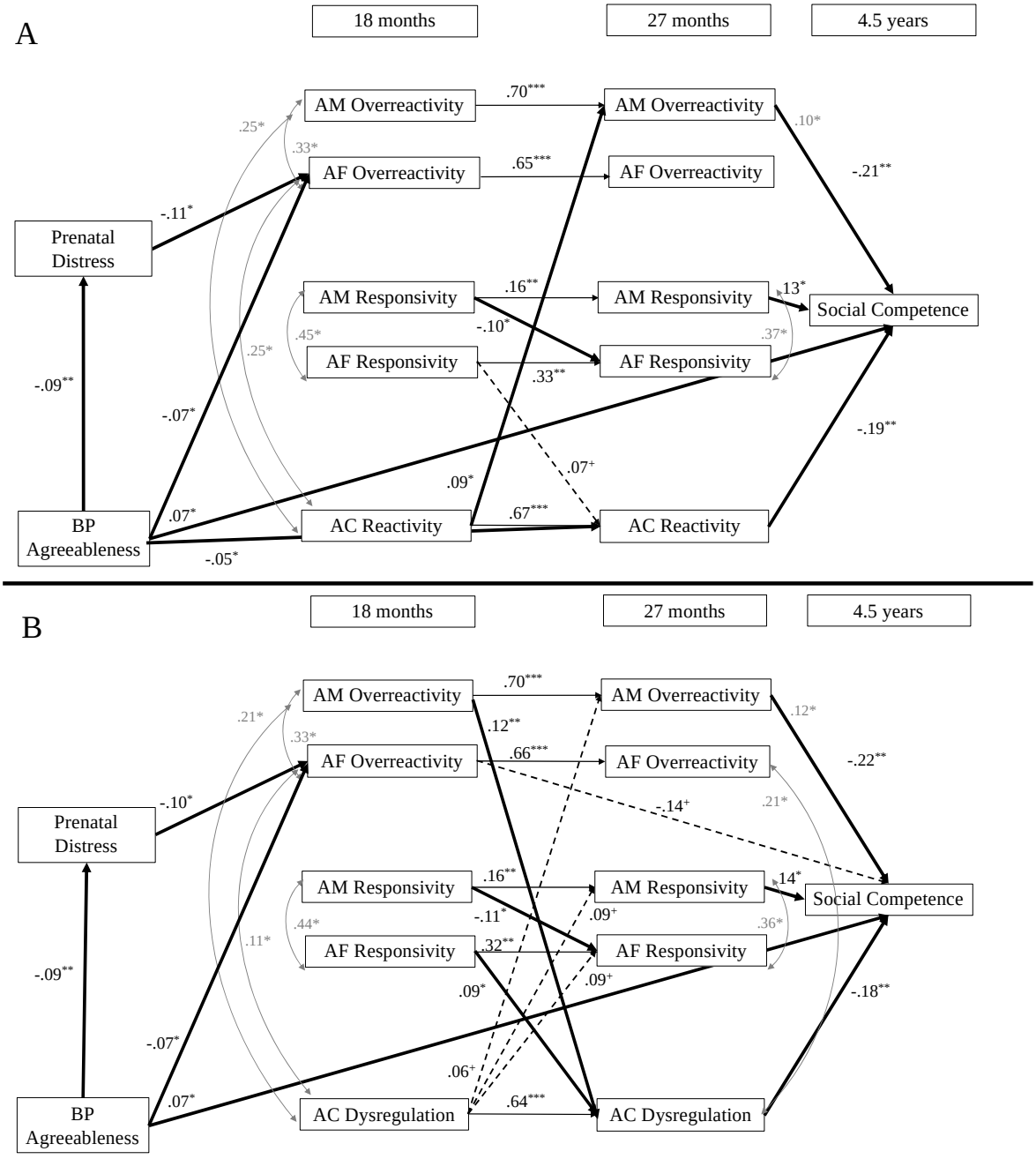
Birth Parent Emotion Dysregulation Models



Note. Figure 1A presents the results with adopted child reactivity, and 1B presents the results with adopted child dysregulation. BP = birth parent, AM = adoptive mother, AF = adoptive father, AC = adopted child. + $p < .07$, * $p < .05$, ** $p < .01$, *** $p < .001$.

Figure 5.2.

Birth Parent Agreeableness Models



Note. Figure 2A presents the results with adopted child reactivity, and 2B presents the results with adopted child dysregulation. BP = birth parent, AM = adoptive mother, AF = adoptive father, AC = adopted child. + $p < .07$, * $p < .05$, ** $p < .01$, *** $p < .001$.

Discussion

This is one of the few genetically informed studies that examines heritable, prenatal, and postnatal influences in early childhood simultaneously, and the first to examine children's early social competence. The findings from this study suggest that not only are there unique heritable influences on children's social competence, but that there are specific child behaviors that elicit specific parenting behaviors, and parenting behaviors that elicit specific child behaviors. There was also a prenatal effect on parenting that might suggest the potential for heritable influences being transmitted through the prenatal environment that in turn influences parenting behaviors. These findings could potentially help us to better understand the risk and promotive factors for early social competence in children.

First, we found different pathways that directly influenced child social competence at 4.5 years. The specific direct pathways we found were (1) heritable influences (birth parent emotion dysregulation and agreeableness), (2) child early behaviors (dysregulation and reactivity), and (3) adoptive mother parenting behaviors, but not adoptive father parenting. These findings support previous research that social competence is at least partially heritable (Battaglia et al., 2017; DiLalla et al., 2012), that early regulatory capacities help social competence to develop (Cole et al., 2004a; Denham, 2006; Hubbard & Coie, 1994), and that parents are influential in the development of social competence (Clark & Ladd, 2000; Driscoll & Pianta, 2011; Haskett & Willoughby, 2007; Masten & Coatsworth, 1998). However, fathers' parenting was not uniquely associated with child social competence. This is consistent with another study (Martin et al., 2010)—that one "good" parent is enough to help promote the child's

social competence and then the child could hit a “ceiling”. It is also important to note that we examined only two parenting behaviors and that fathers may help to advance the child’s social competence development in different ways, like through involvement or scaffolding (Torres et al., 2014). Therefore, examining these specific parenting behaviors (responsiveness and overreactivity) might not be enough to capture the role that the father plays in the child’s development. These direct effects highlight the complex nature of equifinality, whereby multiple risk and promotive factors might come together over time to influence social competence in early childhood.

Second, we found indirect pathways that influenced child social competence at 4.5 years. Specifically, we found that (1) child anger reactivity at 18 months was associated with lower levels of social competence at 4.5 years through evocative effects on adoptive mother overreactivity at 27 months, and (2) adoptive mother overreactivity at 18 months was associated with less social competence through child dysregulation at 27 months. These two findings show that the coercive cycle between mother and child is detrimental to the development of early social competence. Also, these findings highlight that this cycle is both child-directed and parent-directed. Therefore, intervening on either the parent or child alone might not be enough for an intervention work, but instead should focus on the family as different family members might be driving similar coercive patterns. This indirect pathway could also indicate that the coercive cycle is occurring between overreactive parenting and child reactivity, which in turn increases child dysregulation, which in turn negatively impacts their social competence; however, more work needs to be done to examine this potential direction of effects. These indirect pathways support previous work exploring the unfolding of the coercive cycle for

adolescents (Patterson, 1982), but also how the coercive cycle could happen in early childhood (Eddy et al., 2001; Smith et al., 2014). These findings also illustrate how the coercive cycle could negatively impact positive "typical" developmental milestones rather than simply increase negative child behaviors.

We also found potentially unique pathways from parent to child and child to parent that might suggest situational responses to the child's behavioral or emotional cues. Specifically, we found that child reactivity at 18 months was associated with adoptive mother overreactivity at 27 months, while adoptive mother overreactive parenting and adoptive father responsiveness at 18 months was associated with child dysregulation at 27 months, with some of these paths not indirectly related to social competence. These findings might allude to the potential functional differences in these child behaviors. First, reactivity is a situation specific emotional response—often high intensity emotional cues—that the parent is often unable to ignore, particularly because it is not considered to be a positive child behavior. In this instance, this is a child reacting in an angry way. Second, the parent-driven effect on child dysregulation at 27 months highlights the potential importance of mothers' parenting behavior on broader child regulatory capacities. At an early age, children are still using the parent to help regulate their behaviors and emotions because the child is not able to regulate themself alone. High levels of overreactive parenting suggests that the parent themself could be dysregulated and unable to control their behavior towards their child. Therefore, not only is this young child experiencing the mother's hostility— that could be considered extreme—, which could inhibit the child's ability to effectively regulate themself, the mother is also modeling this dyregulated behavior for the child. Over time, the child

might consider this behavior to be expected and in turn can alter how the child addresses particular displays of emotions or behaviors with other children. In addition, we found that fathers' responsivity at 18 months was associated with more child dysregulation at 27 months. While studies generally find that being a responsive parent is important for the child's development (Feldman et al., 2009; Raby et al., 2015; Rispoli et al., 2013), fathers might not be responsive in appropriate ways, as responsivity might serve different functions dependent on the child behavior (Kochanska & Aksan, 2004). For example, fathers could be positively responding whenever the child bids for his attention, whether in positive or negative ways, which could increase the likelihood of reinforcing child dysregulation. There is also research to suggest that when fathers interact with their children their dyadic synchrony of play is categorized by high peaks of intensity which could increase the child's dysregulation (Feldman, 2003). Therefore, while fathers might be trying to find ways to interact with their children, their interactions might be characteristically different than moms and because of that researchers need to further explore this potential negative relationship.

This study also has the unique opportunity to disentangle heritable and environmental influences to examine whether bidirectional effects between parent and child might be due to heritable influences. The evocative *r*GE effect on adoptive fathers' overreactivity suggests that even before toddlerhood, children are eliciting behaviors from their parents. Specifically, a heritable characteristic of the child (e.g., child warmth or easy temperament) is eliciting less overreactive parenting from the father. This finding supports a meta-analysis finding that evocative *rGE* has a larger effect in early childhood and decreases over time (Klahr & Burt, 2014). This also supports previous genetically

informed studies that found early evocative effects on fathers and not mothers (Hajal et al., 2015; Ulbricht et al., 2013), which could suggest that fathers might be more susceptible to characteristics of the child at an early age that are at least partially heritable. We did not however, find other evocative effects on mother or father behaviors in toddlerhood, suggesting that these particular genetic propensities (emotion dysregulation, agreeableness) might be relevant only for this specific parenting behavior, whereas other genetic propensities (e.g., psychopathology) might be more relevant for other parenting behaviors (Trentacosta et al., 2019).

Findings from the present study also examined the potential influence of the prenatal environment on both child and parent behaviors. While we did not find direct associations between prenatal distress and child behaviors (reactivity, dysregulation, social competence), we found that higher prenatal distress was associated with less adoptive fathers' overreactivity. This could partially support the fetal programming hypothesis, because prenatal distress is presumably influencing father negativity through child behavior. Some research suggests that prenatal distress blunts children's cortisol responses, which is associated with less reactivity, and this could be why the father responds less negatively with the child (Laurent et al., 2013; O'Connor et al., 2013). This is supported by previous work with this sample which found that children had a blunted cortisol response at 4.5 years when exposed to prenatal depression (Laurent et al., 2013). We also found that both birth parent emotion dysregulation and agreeableness were associated with prenatal distress; however, the indirect effects to adoptive father overreactivity were not significant. Therefore, while the heritable influences are correlated with the prenatal environment, this association is not in turn related to

parenting. Therefore, it is less likely that this child evocative effect is due to heritable influences and might be specifically the influence of the prenatal environment, supporting previous prenatal work (Barker et al., 2011; Booth et al., 1991).

Finally, one unexpected finding was that mothers' responsiveness at 18 months was associated with less father responsiveness at 27 months. It might be that fathers are seeing the mothers being extremely responsive to their child and therefore we see them engage in this behavior less over time. Also, it is possible that fathers could be adapting more to the developmental behavioral bids from the children. Toddlers present a new challenge for parents, with cognitive and motor advancements, and therefore, fathers might be engaging in other types of parenting behaviors that address this new development. For example, in toddlerhood fathers begin to find ways to engage their children in play and activities outside of the home and so the fathers might be using more scaffolding types of behaviors instead of responsivity.

Limitations. While interpreting these results, some limitations should be considered. The first limitation is our ability to fully disentangle genetic and prenatal influences. This is because birth mothers are reporting on their behaviors as well as their prenatal experiences, increasing the potential for reporter bias. However, compared to other study designs, the adoption design is better able to disentangle genetic and prenatal influences, especially if birth fathers participate (Loehlin, 2016). In an attempt to address this limitation, we have included birth father information. The inclusion of birth fathers allows us to model the full genetic influence (birth mother and birth father), and better separate the genetic and prenatal influences. Second, adoptive parents are reporting on both parent behavior (overreactivity) and child behavior; therefore, increasing the

potential for reporter bias. In an attempt to address this, we combined parent reports of the child behaviors. Finally, these findings might not be generalizable to other populations (e.g., at risk families, non-adoptive families) due to the potential of restricted range (Stoolmiller, 1999). However, one study suggests that this restricted range does not differentially impact child outcomes compared to non-adoptive families (McGue et al., 2007).

Conclusion. These limitations do not minimize the strengths of this study, particularly that this is one of a few studies that have examined multiple levels of influence (heritable, prenatal, postnatal) on the development of early social competence. The current study simultaneously assessed both promotive and risk factors to better capture the complex multifaceted mechanisms of influence on children's social competence. These findings suggest not only that child and parent interactions are impacting child social competence, but that some of the parenting behaviors might be being elicited by heritable characteristics of the child. Additionally, the lack of prenatal influences on child behavior does not imply that these influences do not exist. Rather, the child behaviors examined in this report may not be the most sensitive to the effects of pure prenatal distress. Future genetically informed studies should continue to examine the influence of the prenatal environment as a risk mechanism on positive child outcomes, and consider examining child cortisol as a mechanism through which the prenatal environment influences child development. The heritable influences found here are not trivial; therefore, research moving forward needs to continue using genetically informed designs to examine the role of reciprocal relationships between parents and the child to influence child social competence.

Chapter 6

Conclusion and Future Directions

The purpose of this dissertation was to examine the influence of four risk and protective factors on the development of social competence in early childhood: heritable influences, prenatal distress, parenting, and child behavior. The development of social competence in early childhood is important for school readiness, and for the child's later academic achievement and mental health (Blandon et al., 2010; Bornstein et al., 2010; Jones et al., 2015; Masten & Obradović, 2006). Most of the literature examining the influences of these important factors has examined them in isolation (Battaglia et al., 2017; Denham, 2006; Eichler et al., 2017; Eisenberg et al., 2010; Fabes et al., 2006); however, the development of child behavior is more complex. The first paper found that children's early behaviors (dysregulation and reactivity) and heritable influences were associated with the development of social competence; however, prenatal distress was not associated with social competence or early child behaviors as found in previous literature (Carter et al., 2001a; Davis et al., 2007; Korja et al., 2017; Nolvi et al., 2016). The second paper found that there are different developmental paths by which parent and child behaviors can negatively influence social competence. Additionally, there was evidence for evocative *r*GE and prenatal influences on parenting behaviors. Together these complementary papers attempt to provide a comprehensive understanding of how family

systems, behavior genetics, and the fetal programming hypothesis can be examined together.

The results from these studies found that there are unique direct effects on children's social competence when heritable, prenatal, parenting, and child behavior are examined simultaneously. Specifically, we found that heritable influences, adoptive mother overreactivity and responsiveness, and adopted child dysregulation and reactivity were all associated with child social competence at age 4.5 years. These findings directly corroborate previous studies suggesting heritable influences partially explain social competence during childhood (Battaglia et al., 2017). Additionally, previous findings and conceptualizations of child dysregulation and reactivity as risk factors for lower social competence and school readiness are supported (Cole et al., 2003; Denham, 2006). Finally, this work highlights the importance of parenting for children's social development (Feldman & Masalha, 2010; Hartas, 2011). Therefore, these findings provide evidence for the mechanistic pathways suggested in the conceptual model (Figure 1.1) and the equifinality of these pathways for the development of social competence. These findings also highlight that multiple factors are influencing the development of social competence and further studies need to address this complexity to better understand the mechanisms of influence.

Although previous studies have found a direct effect of the prenatal environment on children's social competence (Carter et al., 2001a; Eichler et al., 2017), this study found no direct effect. It is important to note that the studies reporting direct effects of prenatal environment on children's social competence did not use genetically informed designs; therefore, it is not clear within these studies whether such findings represent a

prenatal effect or a conflated estimate that includes both heritable and prenatal influences. We also did not find that prenatal distress influenced child behaviors (dysregulation and reactivity) in toddlerhood, there was only a trend level association at 18 months (Figure 1.1, path b). However, Study 2 found that prenatal distress was associated with adoptive father overreactivity at 18 months (path i). Since the prenatal environment is provided by the birth mother, the only way the prenatal environment can influence the adoptive parents' parenting is through the child. Therefore, these findings may suggest that prenatal distress might be influencing child behaviors in infancy that in turn is impacting adoptive father overreactivity towards the child. Future studies should further explore the relationship between prenatal distress and early parenting by incorporating child behaviors in infancy to better understand how effects of the prenatal environment may influence parenting.

Finally, the current findings supported bidirectional effects between parent and child behaviors in infancy and toddlerhood and demonstrated that these bidirectional effects were associated with child social competence at 4.5 years old (path g to h). Specifically, we found that higher levels of adopted child reactivity at 18 months was associated with more adoptive mother overreactivity at 27 months, and higher levels of adoptive mother overreactivity at 18 months was associated with more adopted child dysregulation at 27 months. Both of these pathways were indirectly related to lower levels of social competence at 4.5 years. These findings support the need to examine bidirectional effects between parents and children to better understand children's social development. These findings also suggest that studies that examine parents' parenting as the main driving force for socialization should incorporate the role of the child, consistent

with research examining coercive cycles between parents and young children (Chang & Shaw, 2016). This is a particularly important implication for interventions to consider attempting to target the dyad or family instead of a specific person (e.g., parent, child) in an attempt to correct potential long-lasting negative outcomes.

The bidirectional effects between parents and children found in this dissertation were not influenced by evocative *r*GE (path e to g). We did, however, find evidence of an evocative effect from birth parent agreeableness to adoptive father overreactivity at child age 18 months (path f). This finding partially supports the path from genetic influences to parenting, although we did not identify the child behavior that mediated this path. Previous studies have found evocative effects, partially explained by heritable effects in toddlerhood (Natsuaki et al., 2013); however, this is the first study to examine multiple parenting behaviors and child behaviors at once.

Findings from Study 2 also suggest that there might be unique bidirectional patterns between parent and child during early childhood. Specifically, we found that child reactivity at age 18 months (and not dysregulation) was associated with parent overreactivity at child age 27 months, while parent overeactivity was associated with child dysregulation at 27 months (and not reactivity). This suggests that there are specific behaviors in children that elicit specific behaviors in parents. In other words, child dysregulation at this age seems to be parent-driven, whereby parents are influencing their child's ability to regulate. However, child reactivity (displays of intense emotions or the proneness to display a negative emotion) is a behavior that elicits overreactivity from their parent, instead of the parent eliciting reactivity. This might be due to the functional differences of regulation and reactivity (Dennis et al., 2006). Reactivity presumably is a

way for children to cue their parent to a distressing situation or to convey frustration, while dysregulation is the inability to inhibit inappropriate responses. This functional difference is supported particularly in the regulation literature, where children need their parents to help them regulate and over time they learn how to regulate from their parents and transition to being able to self-regulate (Cole et al., 2004b). Meanwhile, the emotion literature would suggest that reactivity elicits parental responses (Buss & Kiel, 2004; Hane et al., 2008; Zeman et al., 2006). Therefore, the findings from this study, while further illustrating the functional differences of these child behaviors/emotions, could potentially help researchers to better target and understand how family processes unfold over time.

Overall, this dissertation provides support for multiple aspects of the conceptual model (Figure 1.1). Because this is one of the first studies that has examined heritable, prenatal, and postnatal environmental influences together in regard to the development of children's social development, these findings offer insights for understanding different mechanistic pathways (environmental versus heritable) and potential new directions. Overall, the first research question, that multiple factors: heritable, prenatal, parental, and child behaviors would be associated with social competence, found that all factors, except the prenatal environment, were associated with social competence. The second research question, that prenatal distress would mediate the association between heritable influences and social competence, was not supported. Birth parent temperament was associated with prenatal distress, which might indicate that some of the prenatal environmental influences were due to heritable characteristics. The third research question, that parenting is elicited by children's heritable characteristics, was partially

supported. Taken together, these findings suggest that parents and children influence one another, with some evidence suggesting that families are also influenced by both the prenatal environment and heritable factors. This dissertation provides a starting point for exploring the underlying risk and resiliency pathways, and highlights the importance of considering genetic, prenatal, and family influences to understand the development of social competence.

These findings should be considered in light of general limitations of the studies and the design. First, the sample used in these studies are generally homogenous. Adoptive parents were mostly Caucasian and were more educated and had higher incomes than birth parents. This could limit the generalizability of the findings; however, one study's findings suggest that the potential for restricted range for adoptive families had minimal effects on children's outcomes compared to non-adoptive families (McGue et al., 2007). Another limitation is the ability of the study to disentangle heritable and prenatal environmental effects. Genetically informed designs are limited in their ability to disentangle genetic and prenatal effects, similarly to non-genetically informed studies, unless researchers use an adoption at conception sample, where both birth parents' information is accessible. Our use of birth father data along with birth mother data increases our ability to estimate the full heritable influence and disentangle prenatal effects; however, this is not without some potential bias because more birth mothers than birth fathers participated. Finally, adoptive parents reported on both their own parenting and the child behaviors, which might result in shared method variance. In an attempt to reduce this, we used observer ratings of one of the parenting constructs (responsiveness) and we averaged parent reports on the child behaviors.

This dissertation provides a direct example of combining genetic, prenatal, and family environmental influences to better understand the development of social competence. By examining all of these within the adoption design we are not only able to disentangle the different levels of influence, but we are able to simultaneously examine the multiple potential mechanisms that are occurring in a child's early life. This is a direct test of integrating behavioral genetics within family systems research and prenatal research to better examine and understand what aspects of the child's environment and genetics might be mechanistically involved in children's social development. Findings from these studies can inform developmental interventions that are attempting to target specific developmental timepoints/milestones. For example, the first study's findings might suggest that interventions targeting child behaviors in early childhood might be more effective for boosting social competence. Findings from the second study might suggest that family interventions might want to consider the different functions of parenting behaviors and whether it is important to address the family as a whole (e.g., coercive process) or attempt to intervene at the individual level (parenting or child behavior). Additionally, this work might suggest that early prevention work like Family Foundations might help to decrease potential risks during the prenatal period that could affect parenting (Feinberg & Kan, 2016; Solmeyer et al., 2014). Future studies need to explore the association of prenatal risk on positive child behaviors (e.g., social competence) in order to better understand the relationship between the prenatal environment on positive outcomes. Future studies should also continue to examine these complex family relationships within genetically informed designs in order to understand the mechanisms that lead to positive child adjustment. Ultimately, examining the

influence of these different risk and protective factors simultaneously within a genetically informed design can help researchers to not only “close in on” important mechanisms of change, but also important development periods where change might be more influential.

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VITA

Amanda M. Ramos

EDUCATION

Candidate for Ph.D. in Psychology, 2020, The Pennsylvania State University, Area of Study: Developmental Psychology
M.A. in Psychology, 2015, California State University, Long Beach, CA
B.A. Psychology (Summa Cum Laude), 2010, Humboldt State University, Arcata, CA

GRANTS & FELLOWSHIPS

2016-2019 John Templeton Foundation: Genetics and Human Agency junior researcher award
2016-2020 Training Interdisciplinary Educational Scientists (TIES) Fellowship R305B090007

SELECTED AWARDS

2016 NSF Funded Award (SES 1258678)
2015 Strumpf Liberal Arts Centennial Graduate Scholar Award
2013 Outstanding Student Research Division 45, APA Convention

RESEARCH EXPERIENCE AT PSU

2014-2020 **Graduate Researcher,** Gene-Environment Interplay Lab, PI: Dr. Neiderhiser
2015-2019 **Study Coordinator**, Pennsylvania Twin Registry, PI: Dr. Neiderhiser,
2016-2019 **Graduate Researcher,** Family P.O.W.E.R. Lab, PI: Dr. Fosco

PUBLICATIONS

Borriello, G. A., **Ramos, A.M.,** Natusaki, M. N., Reiss, D., Shaw, D. S., Leve, L. D., & Neiderhiser, J. M. (in press). Investigating the etiology of children's mathematics achievement using a prospective adoption design. *Developmental Science.*

Ramos, A.M., Chen, T., Hatemi, P., Cleveland, H.H., Neiderhiser, J.M. (2019). The Pennsylvania Longitudinal Study of Parents and Children (PALSPAC) twin registry. *Twin Research and Human Genetics, 22*(6), 765-768. Doi:10.1017/thg.2019.98.

Ramos, A.M., Griffin, A. M., Reiss, D., & Neiderhiser, J.M. (2019). Did I inherit my moral compass? Examining socialization and evocative mechanisms for virtuous character development. *Behavior Genetics, 49*(2), 175-186.

Fosco, G., Mak, H.W., **Ramos, A.M.**, LoBraico, E., & Lippold, M. (2019). Exploring the promise of assessing dynamic characteristics of the family for predicting adolescent risk outcomes. *Journal of Child Psychology and Psychiatry, 60*(8), 848-856.

PROFESSIONAL ACTIVITIES AT PSU

Assessment Consultant, Park Forest Preschool (Fall 2018-Spring 2020)
Methodology Consultant (Spring 2017 – 2020)
Search Committee Member, CCSA search (2018-2019)
Graduate Student Mentor (Fall 2018-Spring 2020)
Ad Hoc Journal Reviewer: Behavioral Genetics, Infant and Child Development