The role of gene and environment interplay in understanding potential mechanisms underlying parenting and children's social-emotional development

Dissertation

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Abstract

Children's social-emotional development is important for the quality of one's life. Based on the bioecological theory and family process model, this dissertation aims to understand the mechanisms underlying parenting and children's social-emotional development, focusing on gene and environment interplay using serotonin genes. Grounded on the literature review in Chapter 1, Chapter 2 investigated whether children with sensitive genes were more likely to show social competence if they experienced positive relationships in an earlier period. The results showed that children with sensitive alleles on 5-HTTLPR or STin2 were likely to show greater social competence if they were securely attached to mothers. Chapter 3 tested mediated and moderated paths from mothers' and children's genes to child behavior problems via child temperament and mothers' negative parenting behaviors. Based on the conceptual framework about gene and environment interplay, I tested gene and environment correlations (passive, active, and evocative) and interaction. The results showed that mothers' sensitive allele(s) on TPH2 rs4570625 inherited to their children and children with sensitive allele(s) were more likely to show anxious and withdrawn behavior problems via mothers' psychological aggression (passive rGE). Children with sensitive allele(s) on 5-HTTLPR were likely to show difficult temperament in infancy and anxious behavior problems after entering schools (active rGE). Children with sensitive allele(s) on 5-HTTLPR were also likely to receive mothers' negative parenting behaviors and to experience more

internalizing behavior problems. However, sequential mediation paths supporting evocative rGE were not statistically significant, and there was no significant moderation (G x E) found. After I examined the association between children's genes and their social-emotional behaviors in Chapters 2 and 3, I investigated the association among mothers' genes, fathers' support, and mothers' parenting behaviors in Chapter 4. Mothers who had at least one or more sensitive alelle(s) on 5-HTTLPR were less likely to show physical aggression to children if they received more coparenting support from fathers. However, if mothers received greater childcare provision from fathers, mothers with sensitive allele(s) were more likely to show physical and psychological aggression to their children. Chapter 5 summarized major findings of these studies, discussed weaknesses, and provided suggestions for future research.

Dedication

To my Jesus and beloved parents who support me with endless love

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Fields of Study

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Table of Contents

List of Tables

List of Figures

Chapter 1. Introduction

Children's Social-Emotional Development

Strong social-emotional development can help children to have a better quality of life. Children with social-emotional competence can show more passions to learn, more enthusiasm to participate and communicate, and better patience and understanding by regulating one's emotions and behaviors so they are ready for early learning and academic achievement (Denham, 2006). Children and adolescents in low-income families are likely to develop limited social-emotional skills, to experience school failures, and to be involved with more antisocial and violent behaviors (Aviles, Anderson, & Davila, 2006). Fostering healthy social-emotional development can benefit in regulating emotions, identifying one's needs, exploring to satisfy one's desire in a productive way, and building positive relationships with others. However, young children in the U.S. commonly experience social-emotional problems (Cooper, Masi, & Vick, 2009). Young children's social-emotional problems are categorized as internalizing and externalizing behavior problems (Achenbach, 1982). Depression or anxiety are representative types of internalizing behavior problems (Gresham & Kern, 2004), and violent or aggressive behaviors belong to externalizing behavior problems (Furlong, Morrison, & Jimerson, 2004). Positive social-emotional development is less studied, but it is also important to investigate positive social-emotional outcomes because revealing significant factors

contributing to positive social-emotional outcomes can provide useful information about what to focus in prevention programs for at-risk children.

Guiding Theories

To understand the roles of parenting in children's social-emotional development in low-income families, researchers have developed two major theories such as family process model and family investment model (Vernon-Feagans, & Cox, 2013). Family process model explains that the lack of family economic resources increases parental stress, and therefore increases the risks of children's behavior problems (McLoyd, 1990). Family investment model explains that limited family socioeconomic resources restricted parental investment on toys, books, or educational resources so children have less opportunities to build their capacities (Mayer, 1997). While family investment model is more useful to explain cognitive development, family process model is very helpful to explain social-emotional development. Both models share social causation perspective which assumes that social condition affects parenting and individual well-being (Simons et al., 2016).

In terms of children's social-emotional development, the debate between nature versus nurture has a long history. So far, it is still debatable which one comes first or which one is more important than the other, and scholars concur that both make important contributions to children's development. However, with the development of new technology, researchers with expertise in different areas have attempted to collaborate more and include both genetic and environmental factors to better understand the mechanisms underlying child development. Understanding the roles of genetic

sensitivity can inform about the role of nature and investigating the roles of parenting can reveal the influence of nurturing in human development.

Before the advent of social scientists' academic interests about genetic sensitivity, parenting, child attachment, temperament, and children's social-emotional development are key topics to understand human development, and this approach is still valid. For example, Kiff, Lengua, and Zalewski (2011) examined how mothers' parenting and child temperament are associated with child behavior problems. The results showed that children with high impulsivity, frustration, and low effortful control received greater effects of mothers' negative parenting on developing behavior problems and elicited mothers' negative parenting. The increase in mothers' negative parenting behaviors made children more fearful. Bronfenbrenner (1979)'s ecological theory provides a strong theoretical foundation to encourage the interaction between individual characteristics, parent-child relationships, family contexts such as socioeconomic conditions, and environmental factors to explain children's proximal developmental processes. In Bronfenbrenner and Ceci (1994)'s bioecological theory, an updated version highlighting the role of genetic sensitivity, individual's characteristics are more sophisticated identified such as one's genotypes and phenotypes. They thought revealing which genotypes are associated with behavioral phenotypes as well as how environmental factors interact with individual characteristics are critical question for a better understanding of child development. In this bioecological theory, individual genotypes do not fix the person's behavioral outcomes. From the same gene, a variety of phenotypes can appear. Individual genotypes are inherited from one's parents, but they are not

exactly the same as those from parents. Therefore, unexplained parts by the concept of heritability in behavioral genetics might be solved by the concept of proximal developmental process in bioecological theory, which explains how individual genetic sensitivity interacts with surrounding environments (Bronfenbrenner & Ceci, 1994).

Gene and Environment Interplay

Recently, there is growing interest about gene and environment interplay. For example, the differential susceptibility hypothesis actively includes the moderation effect by individual genetic sensitivity on the path from environmental factors to social-emotional behavioral outcomes (Belsky & Pluess, 2009). At the same time, even individual genetic sensitivity is interpreted as the consequence of the interaction between a fetus in a mother's womb and a mother's environments during pregnancy (Belsky & Pluess, 2009).

Although the influence of child attachment and parenting on children's social-emotional development might differ by children's genetic characteristics, in the past developmental scientists rarely included genetic sensitivity. Understanding genotype, phenotype, and endophrenotype is important in genetic studies (Beauchaine, Hinshaw, and Gatzke-kopp, 2008). Due to technological developments in gene sequencing, interest has intensified in understanding how individual genetic characteristics and environmental risk or protective factors contribute to children's development.

The way that individual genetic characteristics and environmental conditions are associated with each other to predict human development is called gene and environment interplay (Rutter, 2006). Gene and environment interplay consists of gene and environment interaction (G x E) and gene and environment correlation (rGE). Gene and

environment interactions focus on moderation effect of genetic characteristics on the influence of environmental risk/protective factors. In terms of gene-environment correlation, there are three types: passive, active, and evocative correlations. A passive correlation means that parents who have particular genes are more likely to provide their children with environments consistent with those genes. An active correlation highlights the role of children's own genetic characteristics when children choose what to do rather than following parental genetic characteristics (i.e., niche-picking). An evocative correlation explains that the responses children elicit from other people are the result of the child's genetic characteristics (Rutter, 2006).

As regards gene and environment interaction, there are two major perspectives on the influence of genes and environments on child development. The traditional approach is called the diathesis model (Monroe & Simons, 1991), and a recent approach called differential susceptibility hypothesis extended the traditional approach (Bakermans-Kranenburg & van IJzendoorn, 2007; Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007; Belsky & Pluess, 2009). Both theories share the idea that children with greater genetic sensitivity will show poorer developmental outcomes when they experience unfavorable environments compared to children with less genetic sensitivity. While the diathesis stress perspective highlights the risks of developing negative social-emotional outcomes for children with sensitive alleles experiencing disadvantaged environments, the differential susceptibility hypothesis focuses on the "bright side" of having sensitive alleles for positive social-emotional outcomes under advantageous environments. To summarize these ideas, the differential susceptibility hypothesis

describes children with greater genetic sensitivity as “orchids” and children with less genetic sensitivity as “dandelions” (Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van Ijzendoorn, 2011). However, there is relatively little empirical research to support the hypothesis about the “bright side” of differential susceptibility. This is because previous research has usually compared children’s development in negative and non-negative environments, and has not compared children’s development in negative and positive environments (Ellis et al., 2011). Moreover, the diathesis model is not a singular model as proponents of the differential susceptibility hypothesis have portrayed. There are several models in the diathesis model, and they also assume differences in individual genetic sensitivity toward environmental stimuli (Ingram & Luxton, 2005). Recently, the concept of resilience in the diathesis model also includes the variation in individual responses toward positive outcomes. It is interesting to see that the differential susceptibility hypothesis and diathesis model share more in common than expected.

Serotonin Genes

In our brain, serotonin (5-hydroxytryptamine; 5-HT) influences our mood or emotion regulation (Brown & Hariri, 2006). Thus, its neurotransmission relates to child temperament, individual personality, and susceptibility to mental health problems (Murphy, Andrews, Wichems, Tohda, & Greenberg, 1998). The serotonin transporter (5-HTT) system affects the release of 5-HT in our brain by regulating reuptakes of 5-HT (Brown & Hariri, 2006). Therefore, 5-HTTLPR is the most widely used gene in studies of genetic sensitivity in developmental psychopathology. Generally, having a short allele on 5-HTTLPR is associated with lower amygdala activity (Hariri et al., 2002), and greater sensitivity toward stressful events (Caspi et al., 2003).

In this dissertation, I focused on the roles of the serotonin genes 5-HTTLPR, STin2, and TPH2 as indicators of genetic sensitivity. 5-HTTLPR and STin2 are collected from the same gene but located at different loci (Mitchell et al., 2015). Caspi, Hariri, Holmes, Uher, and Moffitt (2010) conceptualized genetic sensitivity as stress sensitivity and reviewed previous research that operationalizes it with the serotonin transporter gene (5-HTT). In the genotypes of a functional polymorphism of the serotonin transporter gene (5-HTTLPR), two common alleles are the short (S) 14-repeat allele and the long (L) 16-repeat allele. Combinations of these alleles form three genotypes: LL, SS, and LS. Children with one or more short (S) allele(s) show greater sensitivity to environmental influences. In addition to 5-HTTLPR, another serotonin gene is STin2 VNTR (Mitchell et al., 2015). The number of 17 base pair repeats (VNTR) varies in Stin2 and the two common alleles are the 10-repeat allele and 12-repeat allele. Among them, having one or two 12-repeat alleles is assumed to reflect greater sensitivity and to confer a higher risk of depression. I also used TPH2 rs4570625. Tryptophan hydroxylase (TPH) is used to make 5-HT. TPH2 is located on Chromosome 12 and affects the synthesis of 5-HT by regulating the release of TPH (Walther et al., 2003).

Parenting

Many studies reported the association between children's genetic characteristics, parenting, and children's social-emotional behavior outcomes (i.e., Sulik et al., 2012). Relatively, there are only a few studies about the roles of mother's genetic sensitivity on mothers' parenting behaviors. Cents and colleagues (2014) is one rare empirical example. They investigated the association between mothers' 5-HTTLPR and their sensitive parenting, and the results suggested that mothers with an S-allele were more likely to

provide sensitive parenting to their children. Interestingly, children's 5-HTTLPR did not explain the association between maternal 5-HTTLPR and their sensitive parenting. In other words, the research found a unique contribution of maternal genetic characteristics to predict mothers' parenting behaviors.

Moreover, whether the role of mothers' genetic characteristics in parenting is affected by fathers' contributions to parenting or coparenting has never been explored in studies about genetic sensitivity and mothers' parenting. Mileva-Seitz, Bakermans-Kranenburg, and van IJzendoorn (2016) reviewed genetic mechanisms of parenting and highlighted the need to incorporate mothers' genetic information and extend the research to include other caregivers such as fathers. Previous studies using the family process model have typically considered only mothers' parenting. In a review of research using the family process model, Barnett (2008) suggested that researchers should incorporate fathers more, because fathers may play important roles in family processes. A father's active involvement in coparenting a child can relieve the mother's stress and help to improve the quality of her parenting.

Some might question whether fathers' parenting plays a significant role, particularly in single-mother families. This is worth considering because of the increase in nonmarital births and family instability, especially for socio-economically disadvantaged populations (Lundberg, Pollak, & Stearns, 2016). In fact, modern academic interest in the influence of fathers' parenting on children's development originated in attempts to measure the effects of father absence on child development (Leidy, Schofield, & Parke, 2013). However, nonresident fathers do play an important

role in their children's development. For example, a recent study (Choi & Pyun, 2014) showed the influence of nonresident fathers' financial and instrumental supports on child development. It mainly supported the family investment model by revealing that nonresident fathers' financial support was significantly associated with children's positive development. However, the results also supported the family stress model by revealing that nonresident fathers' instrumental support and financial support significantly reduced single mothers' parenting stress and significantly increased mothers' positive parenting behaviors, which were significantly associated with children's social-emotional outcomes. There have not been enough attempts to reveal the role of fathers' parenting/coparenting based on the family process model. In this dissertation, I focus on psychological benefits (i.e., children's social-emotional behavioral outcomes or mothers' parenting behavior toward children) from fathers' parenting/supportive coparenting.

Structure of Dissertation

The major goal of this study is to fill in the identified gaps such as (1) the lack of studies connecting individual genetic sensitivity and children's positive social-emotional development, (2) the lack of studies connecting individual genetic sensitivity to family process model, and (3) the lack of studies integrating fathers' support under the genetic mechanism of mothers' parenting. To be specific, through this investigation, I examined the interplay between genes and environments in three different studies. The first study focused on testing whether genetically sensitive children actually experience more social competence than other children in positive environments. The second study tested gene and environment correlations and interaction models with mother's and child's genetic

sensitivity, mother's psychologically aggressive behaviors, and children's internalizing behavior problems. Lastly, the third study incorporated fathers' support by examining whether genetically sensitive mothers provided less aggressive parenting behaviors if they received greater support from fathers. The three studies used the survey, home observation, and genetic contract data from the Fragile Families and Child Wellbeing Study (FFCWS). I analyzed data from 2,646 mothers and children in FFCWS who completed the surveys for parents and focal children from childbirth to child age 9 years and participated in the collection of saliva samples for genetic analysis at 9 years old.

According to Erikson's psychosocial theory (1950), children experience different developmental pathways depending on whether they achieve required tasks at each developmental stage. For example, children establish basic trust or distrust from childbirth to 1 year old by experiencing sensitive care from parents. Then, children develop autonomy or shame and doubt though the experience of toilet training from 1 to 3 years old. Subsequently, children can develop imagination or guilt through make-believe play and adjusting to parental demands about self-control from 3 to 6 years old. After entering schools, children can learn how to cooperate with others and develop the feeling of productiveness or inferiority from 6 to 11 years old (Berk & Meyers, 2016). Given that children's social-emotional functioning established by early childhood predicts their future success, relationships, and social-emotional wellbeing in adulthood (Asendorpf, Denissen, & van Aken, 2009; Jones, Greenberg, & Crowley, 2015), this dissertation aimed to reveal how genetic and environmental factors were associated with

mothers' parenting and children's social-emotional development in early and middle childhood.

Study 1. The first study tested the association between children's differential susceptibility and their social competence in early childhood. In order to explore whether children with higher genetic sensitivity show more social competence if they experienced more advantageous environments, I examined the associations between children's serotonin genes (5-HTTLPR and STin2), child temperament, child attachment, parental adjustment (parental mastery/stress), and children's social competence through latent structural equation modeling with tests of moderation.

Study 2. The second study tested gene and environment interplay through mediations and moderations. In this research, I tested how children's and mothers' serotonin genes are associated with child temperament, mothers' psychological aggression, and children's later internalizing behavior problems. There are three correlations to explain gene and environment interplay. One is a passive correlation focusing on inheritance from mothers and genetically sensitive mothers' tendency to provide more negative parenting. Another is an active correlation highlighting children's active roles in choosing risky environments guided by their own genetic sensitivity beyond mothers' influence. The other is an evocative correlation assuming that genetically sensitive children's tendency toward difficult temperament triggers mothers' negative parenting. In addition, I also examined the moderation model of gene and environment interaction by testing the interaction between children's serotonin genes and mothers' aggressive parenting. After testing each model, I combined all significant

results and tested the multiple sequential mediation model. In doing this, I tried to draw a map to understand how mother's or children's differential susceptibility affects their behavioral outcomes (parenting behaviors for mothers and behavior problems for children) in given environments.

Study 3. The last study tested the roles of fathers' supportive coparenting and childcare provision on the association between mothers' serotonin genetic sensitivity and aggressive parenting behaviors. The basic research question started from how mothers' genetic sensitivity affects their parenting behaviors and which factors interact with mothers' genetic sensitivity to predict their parenting behaviors. There is no prior research that has incorporated fathers' supports in the genetic mechanisms underlying mothers' parenting.

These three studies were designed to extend our knowledge about gene and environment interplay – especially our understanding of the mechanisms underlying children's social-emotional development and mothers' parenting. In sum, the major goal of these three studies was to reveal hidden mechanisms involving gene and environment interplay to ultimately inform prevention and intervention efforts directed at families and parents to promote children's healthy social-emotional development.

The Data Analyzed

In this dissertation, I used data from the Fragile Families and Child Wellbeing study (Reichman, Teitler, Garfinkel, & McLanahan, 2001). This dataset reflects a representative sample of nonmarital births in U.S. cities. FFCWS collected data from 4,898 couples at childbirth at hospitals and followed up at focal children's age 1, 3, 5, and 9 years old. At each wave, FFCWS collected data through phone interviews for core

mother's and core father's survey, and via home visitation to observe mothers' reactions to children and provide direct assessments of children's developmental outcomes (FFCWS, 2008; FFCWS, 2013). FFCWS also collected saliva samples for genetic assay from 2,884 focal children and 2,670 of their biological mothers at children's age 9 (FFCWS, 2015).

This large data set is beneficial for testing the roles of genetic sensitivities because it has more statistical power than many prior studies of gene-environment interplay in children's development. Given that children in low-income families are more likely to have higher risks for internalizing or externalizing behavior problems (Qi & Kaiser, 2003; Shaw, Keenan, Vondra, Delliquardi, & Giovannelli, 1997; Shaw, Vondra, Hommerding, Keenan, & Dunn, 1994), the FFCWS sample is also appropriate for the identification of risk and protective factors in children's social-emotional development. Identification of protective factors, in particular, in this disadvantaged sample may be helpful to inform prevention or intervention programs for children in low-income families. In terms of genetic sensitivity, because this sample is likely to show greater variation in child behavior problems and mothers' parenting behaviors than the average, it was advantageous for testing how genetic sensitivity is associated with mothers' and children's behavioral outcomes in both positive or negative directions. For example, I tested how genetic sensitivity is associated with children's positive behavioral outcomes in the first study, and tested how children's genetic sensitivity is associated with children's internalizing behavior problems in the second study. Finally, given that low-income families experience more instability in parents' relationships, and that FFCWS

oversampled nonmarital births, the last study tested how fathers' support was differently associated with mothers' genetic sensitivity in predicting mothers' parenting behaviors by fathers' marital/residential context.

Chapter 2. Differential susceptibility or diathesis model? Gene and attachment interaction in predicting preschoolers' social competence

Recently, there is a growing interest across diverse fields in the interactions between candidate genes and environments to understand the role of children's genetic characteristics in children's social-emotional outcomes (i.e., Dick et al., 2015; Duncan & Keller, 2011; Weeland, Overbeek, de Castro, & Matthys, 2015). These attempts to incorporate gene and environment interaction are meaningful because they promote a shift in academic interests from ascribing the causes of children's outcomes to only a sole area toward a greater integration of nature and nurture in understanding the etiology of psychological symptoms of mental health problems or developmental processes of social-emotional development.

Theoretical Frameworks to Interpret Gene and Environment Interaction

Gene and environment interaction can be interpreted into two ways. One interpretation is that the influence of individual genetic characteristics on one's developmental outcomes can differ by one's environmental conditions. An alternative interpretation is that the effects of environmental factors can vary depending on individual genetic characteristics (Spinath & Bleidorn, 2017). There are two major theoretical frameworks used to explain gene and environment interaction in psychopathology - the diathesis model (DM) and the differential susceptibility (DS)

hypothesis. Whereas the DM tends to focus on the moderation effects of individual characteristics on the path from environmental conditions to one's psychopathology, DS tends to highlight the differences in the role of individual genes in one's social-emotional behavioral outcomes in interaction with environmental conditions.

In other words, the DM explains how the individual develops psychopathology by the interaction between individual diathesis (genetic components) and stress levels, and compares different vulnerabilities by the level of individual diathesis when drawing moderation plots of high diathesis and low diathesis associations between stress levels and mental health problems (Ingram & Luxton, 2005; Monroe & Simons, 1991). The DS hypothesis strongly recommends interpreting a sensitive allele as a greater sensitivity rather than a greater vulnerability toward environmental stimuli. Thus, proponents of DS want to highlight the aspect that children with sensitive genes can develop better than the average if they receive enough care and supports from their environments (Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007; Ellis & Boyce, 2011).

At first glance, the DS and DM approaches appear conceptually distinct, as argued by proponents of the DS hypothesis (i.e., Bakermans-Kranenburg & van IJzendoorn, 2007; Roisman et al., 2012; van IJzendoorn, Belsky, & Bakermans-Kranenburg, 2012). However, these approaches are not essentially different. There are two reasons that I can say this. First, proponents of the DS perspective argue that their approach sheds light on the "bright side" of having sensitive genes, whereas the DM approach does not. However, when we look at the empirical evidence showing gene and environment interaction based on the DS hypothesis, few of these studies have actually

examined children's positive behavioral outcomes. Instead, many used internalizing or externalizing behavior problems as their outcome variables, and interpreted the absence of problems as an indicator of positive outcomes (i.e., Bakermans-Kranenburg, van IJzendoorn, Pijlman, Mesman, & Juffer, 2008; van Zeijl et al., 2007). Many research based on DS also examined the effects of the environments with/without adversity instead of the effects of the environments with/without positive supports (Belsky & Pluess, 2009). However, having positive environments means more than having non-negative environments (Ellis et al., 2011). Research analyzing the effects of non-negative environments on non-negative outcomes blurs the differences between DS and DM. In those studies, DS could be viewed as an extension of the DM rather than a distinct, competing model.

In addition, like the DS hypothesis, the DM also rests on the concept of different levels in individual diathesis in responding to environmental stress. Previous research suggesting DS has tended to treat the DM as a single perspective, especially with respect to the assumption about genes and children's negative behavioral outcomes (Bakermans-Kranenburg & van IJzendoorn, 2007; Belsky et al., 2007; Roisman et al., 2012; van IJzendoorn et al., 2012). However, when we look at the DM in detail, it is not difficult to find that DM consists of several sub-models such as the additive model, interactive model, or vulnerability-resilience model. As shown in Figure 1, the interpretation of DM by DS applies to interactive model, additive model or vulnerability-resilience model assumes better developmental outcomes (i.e., lower occurrence of mental health

problems) by different levels of individual diathesis under better environmental conditions (i.e., under no stress).

Gene and Environment Interaction by Serotonin Transporter Genes

Generally, previous research has tested gene and environment interaction in relation to anxiety disorders, depression, suicidal behaviors (Mandelli & Serretti, 2013; Nugent, Tyrka, Carpenter, & Price, 2011), treatment effects of antidepressant prescription (Smits et al., 2008), or externalizing behavior problems (Byrd & Manuck, 2014; Cheung, Harden, & Tucker-Drob, 2014). Nugent et al. (2011) reviewed gene and environment interaction studies and showed that frequently investigated genes were serotonin transporter genes (i.e., 5-HTTLPR or STin2). The biological phenotype of Serotonin transporter genes would be the increase of responses on the hypothalamic pituitary adrenal (HPA) axis under stressful situation and the behavioral phenotypes of serotonin transporter genes are the increase of depression or the increase of anxiety-related disorders after stressful experiences (Caspi et al., 2010). In fact, Caspi et al. (2003) is a classic example showing that 5-HTTLPR can moderate the influence of life stress on the etiology of depression.

However, those gene and environment interaction findings are mixed. A meta-analysis (Risch et al., 2009) cast doubt on the moderation by 5-HTTLPR after reviewing studies about the interaction between serotonin transporter gene and stressful life events on the development of depression. In that meta-analysis, the effects of stressful life events were obviously shown to predict later depressive symptoms, while the interaction between 5-HTTLPR and stressful events did not. Thus, Risch et al. (2009) concluded that gene and environment findings related to 5-HTTLPR in previous research appeared by

chance. Contrary to this, another review (Nugent et al., 2011), including more genes in addition to 5-HTTLPR, showed supporting evidence from more empirical studies about a similar topic – the effects of gene and environment interaction and early life stress on anxiety and depressive disorders. Duncan and Keller (2011) reviewed previous research about candidate gene and environment interaction in psychiatry during the first 10 years and showed that 95% of gene and environment interaction studies reported significant findings and 27% of them tried to replicate the results. This review ascribed the gap between the rates to publication bias which made gene and environment interaction findings more robust than the reality, so the authors recommended future research with greater statistical power and directly replicated results.

We call 5-HTT gene-linked polymorphic region as 5-HTTLPR and its intron 2 as STin2, and both of them seem to be associated with the risks of depression and suicide attempts (de Lara et al., 2006). However, while 5-HTTLPR is often used as a main predictor of psychological disorders (i.e., Caspi et al., 2010), STin2 is rarely used as a sole predictor. Instead, STin2 is often included with 5-HTTLPR (i.e., de Lara et al., 2006; Fan & Sklar, 2005; Smits et al., 2004). Since STin2 has been understudied, the findings of 5-HTTLPR and STin2 in health or diseases are incomplete. For example, Smits et al. (2004) showed that European-Americans having two short alleles on the 5-HTTLPR and Asians having 10/12 genotype on the STin2 were less likely to respond to treatment with selective serotonin reuptake inhibitors in reducing depressive symptoms. Fan and Sklar (2005) showed that 5-HTTLPR was not significantly associated with schizophrenia, but STin2 VNTR polymorphism significantly increased the risk of schizophrenia. Fan and

Sklar (2005) used the number 12 allele on the STin2 when investigating the association between serotonin genes and schizophrenia, but de Lara et al. (2006) focused on the STin2 number 10 allele. Generally, STin2 genotypes can be categorized like 12/12, 12/10, 10/10, and other (Florez et al., 2008). In de Lara et al. (2006), having a 10 allele on the STin2 increased the risk of poorer emotional regulation under stress and the risk of suicide attempts.

Gene and environment interaction studies whose outcome variable was positive social-emotional behavior are few. Hankin et al. (2011) tested the interaction between 5-HTTLPR and positive parenting to predict positive affect in adolescents and supported differential susceptibility. They examined the interaction between serotonin gene and parenting three times with different measurements of supportive parenting. To be specific, the first study used parent-report measurement for supportive parenting, the second study used observational measurement for parent-child interaction, and the last study used child-report measurement for parental warmth. All three studies supported that children with two short alleles on the 5-HTTLPR received greater effects from supportive parenting on children's positive affect (Hankin et al., 2011). There are few studies of G X E in young children's positive social-emotional development, but when considering that early childhood is a critical period to develop one's personality and to experience social relationships for the first time outside of one's home, it is worthwhile to examine children's positive social-emotional developmental outcomes and to test the moderation of serotonin transporter genes including 5-HTTLPR as well as STin2.

Predictors of Children's Social-Emotional Behavioral Outcomes

Attachment and Its Interaction with Genetic Sensitivity

In a conceptual framework to explain children's positive social-emotional development, attachment is one of the key aspects of social-emotional development that researchers focus on. In attachment theory, children who established secure attachment with their parents in infancy can develop positive social-emotional behaviors more easily. This is because secure attachment can provide children a safe haven psychologically so they can explore outer worlds more actively and positively (Bowlby, 1988). Bohlin, Hagekull, and Rydell (2000) supported the assumption that infant attachment can be used as a predictor of children's social functioning in school age. They classified infant attachment through the Strange Situation when children were 15 months old. And then, they collected social functioning data at school age through surveys and observations from parents, teachers, and children's self-report. As expected, their results showed that children with secure attachment in infancy were more likely to show positive social behaviors and less likely to show initiative-withdrawal behaviors in peer relationships at school in later periods.

There are some empirical studies inferring the presence of the interaction between attachment and genetic sensitivity in predicting emotional regulation. For example, a recent meta-analysis (Woodhouse, Ayers, & Field, 2015) showed that post-traumatic stress symptoms varied by adult attachment style. This means that attachment style might moderate the effects of individual sensitivity toward stress on the development of psychological symptoms. A recent empirical study (Zimmermann & Spangler, 2016) tested serotonin transporter gene and attachment interaction to explain adolescents' emotion dysregulation and aggressive behaviors toward mothers during a computer

game. Interestingly, for securely attached adolescents, there was no significant difference in emotion dysregulation by children's genetic components (having one or more short alleles vs. not having any short allele on the 5-HTTLPR). However, insecurely attached adolescents showed a significant increase in emotion dysregulation and aggressive behaviors when they had one or more short alleles. This suggests that having secure attachment might reduce the risk of expressing an impulsive genetic disposition. Considering these results, it would be interesting to see how early attachment styles interact with children's genetic sensitivity in predicting their later social competence. For young children, this question has been rarely investigated.

Child Temperament and Its Association with Genetic Sensitivity

Child temperament was often used to predict children's negative social-emotional outcomes. Rothbart and Bates (2007) defined child temperament as "constitutionally based individual differences in reactivity and self-regulation, in the domains of affect, activity, and attention" (p.100). Since child temperament refers to inherited individual differences in reactivity, previous research sometimes used it as a phenotype of children's genetic characteristics. For example, Stoltz, Beijers, Smeekens, and Dekovic (2017) tested the interaction between early parental behaviors and child temperament in predicting children's later internalizing and externalizing behavior problems. The results showed that the interaction between mothers' parenting and child temperament was significant to predict children's externalizing behavior problems. However, additional analysis (i.e., regions of significance) showed that the significant interaction appeared only when the quality of mothers' parenting was poor. For children's internalizing

behavior problems, having negative affectivity in temperament significantly predicted higher risk of internalizing behavior problems regardless of parental behaviors.

Although previous research used child temperament instead of children's genetic sensitivity as a major predictor of social-emotional outcomes, child temperament might function independently from children's genetic sensitivity. Inspired by the hypothesis in the biosocial theory that human personality consists of temperament and character, Hamer, Greenberg, Sabol, and Murphy (1999) tested how serotonin transporter genes were associated with temperament and character. Contrary to the authors' assumption that serotonin genes were closely related to temperament (i.e., harm avoidance), the results showed that serotonin genes were more related to self-directedness and cooperativeness in the character traits rather than temperament traits. Interestingly, there was little attempt to use positive social-emotional outcomes to reveal the association between child genes and temperament, in relation to differential susceptibility and diathesis-stress models.

Parental Adjustment

Mothers' parental adjustment is also an important factor in children's social-emotional development. For example, parental stress negatively influences child development. According to Deater-Deckard and Panneton (2017), parental stress can function as a direct stressor toward children and indirectly socialize children's capacity to handle stressful situations by influencing mothers' parenting behaviors. This is because mothers' parenting stress depletes their personal resources to respond positively to children's requests. Contextual factors such as restriction to socioeconomic resources can

increase mothers' parenting stress. Therefore, mothers in low-income families are likely to be more stressful than others. Through these multi-dimensional aspects, mothers' stress can be a key factor for children's healthy development and mother-child relationship (Deater-Deckard & Panneton, 2017). Parental mastery is a construct related to self-efficacy (see Harmon & Perry, 2011) or the personal control, as often assessed in child abuse and neglect studies (Guterman, Lee, Taylor, Rathouz, 2009; Kang, 2013). Greater parental mastery/personal control is associated with a higher quality of parenting.

The Present Study

In this study, I focused on early childhood because children's social competence in this period predicts their future wellbeing in adolescence and young adulthood (Jones et al., 2015). Child attachment was measured at age 3 because child attachment in this age, showing the security of children's ties to their mothers, is developed by parental sensitivity (Bowlby, 1982). Child temperament was measured at age 1 because it shows individual innate variations in reactivity and self-regulation before training by parental discipline (Rothbart & Bates, 2007). Parenting is a significant factor associated with children's social-emotional development across waves, but I chose parental mastery and stress measured at age 3 because I wanted to observe their associations with children's later social competence at age 5.

Here, I investigated whether and how children's serotonin transporter genes (5-HTTLPR, STin2) moderate the effects of children's individual characteristics and mothers' parental adjustment on children's later social-emotional development by using positive outcomes. So far, there are some studies investigating gene and environment interaction using serotonin transporter genes. In those studies, it appears that there is an

on-going debate between DS and DM. However, as we can see above, many of the gene and environment interaction studies supporting DS also tested the association between children's serotonin transporter genes and negative psychological functioning like depression or anxiety-related diseases as their outcome variables. Therefore, I think those findings are still relevant to DM perspective.

This study used children's positive social behaviors as the outcome variable instead of children's internalizing or externalizing behavior problems, which sets the current study apart from previous research. In addition, I tested how children's serotonin transporter genes, child temperament, child attachment, mothers' parental adjustment predicted children's later social competence. It is well known that serotonin transporter genes are associated with children's emotional dysregulation, but it is not revealed how serotonin transporter genes function in relation to children's social competence. Also, I tested the interaction between children's serotonin transporter genes and each predictor (one by one) to predict children's later social competence. By doing this, I wanted to test whether the results are consistent only with DS, only with DM, or with both types of models. Lastly, considering previous research testing the differences by gender and race/ethnicities, I conducted model comparisons by child gender and race/ethnicities. I anticipated that these attempts can broaden our perspectives on the moderation effect by children's serotonin transporter genes on child development.

Research Questions and Hypotheses

1. How do children's serotonin transporter genes (5-HTTLPR, STin2 VNTR), temperament, attachment, and mothers' parenting adjustment predict children's later social competence in early childhood?

2. Do serotonin transporter genes moderate the effects of other predictors on the development of children's social competence? If they do, which factors interact with children's serotonin transporter genes?

3. Do the results support DS and DM as distinct or related perspectives on gene-environment interaction in children's social-emotional development?

I anticipated that children with major alleles on the 5-HTTLPR or STin2 VNTR, showing less difficult temperament in infancy, having secure attachment to their parents, and having mothers with successful adjustment (i.e., greater parenting mastery and lower parenting stress) would have higher subsequent levels of social competence. For the moderation by serotonin transporter genes, I tested all interactions by serotonin transporter genes with each predictor but did not propose specific hypotheses regarding the interaction effects to predict children's later social competence. Lastly, I expected that the association between predictors and children's social competence would support DS as well as DM, consistent with the notion that DS and DM have more in common than not.

Method

Data and Analytic Sample

This study analyzed the data from 1,529 families who participated in the core parental surveys, in-home interview, and the saliva sample collection across four waves from the Fragile Families and Child Wellbeing study (FFCWS). The original FFCWS study recruited 4,898 couples from hospitals at childbirth and followed up when the focal children were one, three, and five years old. Mothers reported child gender at birth and child temperament at child age one through core parental surveys. The FFCWS added in-home assessment from child age three to five. During in-home assessment, an observer

visited focal children's houses and asked the primary caregivers about parenting and child developmental outcomes. Considering the majority of the primary care givers (> 90%) were biological mothers, this study restricted the sample to the families whose primary care givers were biological mothers to reduce the reporters' or gender bias in ratings. In in-home assessment at children's age three, mothers reported child attachment using the Toddler Attachment Q-Sort. When focal children were five years old, children were invited to participate in saliva sample collection for genetic assays. If children's primary caregivers consented, children's saliva samples were collected in home visitation at child age 5. At each wave, mothers answered questions about family socioeconomic conditions and demographic characteristics (i.e., resident status with the focal child's biological father, the number of children, etc.) in a core parental survey. Based on their answers from the core parental surveys, the FFCWS constructed the variable of family poverty ratio for each household at each wave by applying its definition provided by the U.S. Census Bureau. I included these as control variables in the initial model of this study but removed them after seeing none of these was significantly associated with outcome variables.

Measures

Child social competence. During an in-home assessment when the focal child was five years old, his or her biological mother answered 13 items of the Adaptive Social Behavior Inventory (ASBI; Hogan, Scott, & Bauer, 1992), which measured children's social competence at age five. Items like "(Child) can easily get other children to pay attention to him/her", "(Child) is interested in many and different things", and "(Child)

will join a group of children playing" were included among 13 items. All items were scored from *0 not true* to *2 very true or often true*. Only one item – "In social activity, (child) tends to just watch others" – was reverse coded because it measured children's social competence in the opposite direction. After reverse coding the item, I constructed a latent variable using all 13 items. The reliability of 13 items was .80.

Children's genes. Considering previous research reporting the association between two serotonin transporter genes and social-emotional outcomes, children's 5-HTTLPR and STin2 were used as in the current study. In a preliminary analysis, I tested three different models in coding children's genes: a dominant model, a recessive model, and an additive model. I finally chose to use the recessive model because its model fit was the best. Therefore, children who had two minor alleles (SS on the 5-HTTLPR or 10/10 on the Stin2) were coded as 1 and others were coded as 0. These variables were treated as two independent observed variables.

Child attachment. The FFCWS used the Everett Waters' Attachment Q-Set to measure toddler attachment in in-home assessment at children's age 3. The original Waters' Attachment Q-Set consisted of 90 items but the FFCWS chose 39 items, considering time limitations and the convenience of the measure for parents. An interviewer asked mothers to sort the 39 items into three groups such as frequently applicable, infrequently applicable, and neither of them based on the focal child's behaviors. Then, mothers divided two subgroups for the frequently applicable group and did same thing for the infrequently applicable group based on the level of their frequency (i.e., often vs. sometimes) so that they could apply a 5-point Likert scale from *1 applies*

mostly to *5 rarely or hardly ever* for sorted groups. Based on this scoring, the FFCWS classified focal children into three categories: insecure-avoidant, insecure-resistant, and secure attachment. In this study, I categorized focal children into two groups (secure vs. insecure), because children who showed avoidant insecure attachment were few, so there was little justification to divide insecure attachment into avoidant (2.07%) versus resistant (22.13%). Finally, children who showed secure attachment were 75.79% and children who showed insecure attachment were 24.20%.

Child temperament. Mothers and fathers reported child temperament in core parental surveys when focal children were one year old. This study used mother-report temperament because mothers were a primary caregiver in most cases. Mothers answered three items of the Emotionality, Activity, and Sociability Temperament Survey for Children (Mathieson & Tambs, 1999). These items were: "(Child) often fusses and cries", "(Child) gets upset easily", and "(Child) reacts intensely when upset". Mothers scored each item from *1 not at all* to *5 very much*. I constructed a latent variable of children's difficult temperament using these three items and the reliability of the items was .60.

Parental adjustment. Parental adjustment consisted of parental mastery and parental stress. For parental mastery, five items borrowed from the Parental Mastery Scale (Pearlin & Schooler, 1978) were asked to mothers in the in-home assessment at children's age three. These five items included "I have little control over the things that happen to me", "There is really no way I can solve some of the problems I have", "There is little I can do to change many of important things in my life", "I often feel helpless in

dealing with problems", and "Sometimes I feel that I'm being pushed around". Each item was rated using a 4-point Likert scale from 1 *strongly agree* to 4 *strongly disagree.* Thus, a higher score indicates a higher confidence in managing difficult situations in parenting. The reliability of five items was .81. I constructed a latent variable using all five items.

With regard to parental stress, in the in-home assessment at children's age three, the FFCWS included 12 items, such as "You feel trapped by your responsibilities as a parent?", "Having (child) has caused more problems than you expected in your relationship with men?", or "You enjoy things less than you used to?" to measure parental stress. Originally, each item was scored from 1 *strongly agree* to 5 *strongly disagree*. For easy interpretation, all items were reverse coded from 1 *strongly disagree* to 5 *strongly agree* so that a higher score meant greater stress. The reliability of 12 items was .87. I constructed a latent variable using all 12 items.

Analytic Plan

I first needed to confirm whether the assessments of children's social competence, child temperament, and parental adjustment measured the corresponding constructs correctly. Social scientists investigate abstract concepts in our minds or human relationships based on theories. We can try to measure the behavioral aspects or the degrees of agreement to specific opinions, but if we do not have appropriate measurements to capture a certain aspect of the theoretical concept, then there is no way to measure it. Thus, there is always the possibility of measurement errors. Structural equation modeling (SEM) has the capability to handle measurement error. Moreover, SEM can test a theoretical model because SEM consists of two parts: a measurement

model that tests the validity and reliability of observed items to represent the abstract concepts (using latent variables), and a structural model which tests the relationships between latent variables or latent variables and observed variables (Schreiber, Nora, Stage, Barlow, & King, 2006). Thus, I conducted SEM in Mplus 8.0 after cleaning the data in STATA 14.2.

In the structural equation modeling of this study, I constructed all the variables as latent variables to handle measurement errors except for two predictors (i.e., children's genes and attachment type) which were coded as binary variables. As model fit indices, chi-square (χ^2), RMSEA, CFI, and SRMR were used (Barrett, 2007; Hooper, Coughlan, & Mullen, 2008). Chi-square exact fit test is frequently used in SEM but it is very sensitive to the discrepancies between estimated model and the ideal model especially in large samples (Barrett, 2007). Alternatively, RMSEA, CFI, and SRMR are used because they not sensitive to the increase of sample sizes. RMSEA less than .06 (Hu & Bentler, 1999) or .08 (MacCallum et al., 1996), CFI greater than .90 (Hu & Bentler, 1999), and SRMR less than .05 (Byrne, 1998) indicate a good model fit. RMSEA shows the fitness of the estimated model to the ideally fitted population covariance matrix (Byrne, 1998). CFI (Comparative Fit Index; Bentler, 1990) indicates the comparison of sample matrix to the model without correlation between latent variables (Hooper et al., 2008). SRMR shows the square root of the difference in residuals of covariance matrix between the estimated model and hypothesized model (Hooper et al., 2008). Maximum likelihood estimation (ML) was used in Mplus 8.0.

Results

Table 1 shows all the items which were used in constructing latent variables in the measurement model. The standardized factor loadings for all the items were greater than .30 and the reliabilities of the items for each latent variable were also good. The covariance matrix for all observed variables is given in Table 2. An additional intercorrelation matrix between observed variables showed no correlation coefficients greater than .50 between two items. When I ran the structural equation modeling with these variables, RMSEA was .039 but CFI was .889 and the modification indices recommended me to correlate the residuals of two maternal parenting stress items "Since having (child), are you almost never able to do things you like to do?" and "Since having (child), have you been unable to do new and different things?" and to correlate the residuals of the items "Do you enjoy things less than you used to?" and "Are you less interested in people than you used to be?". After adding these, the model fit very well ($\chi 2 = 1570.64^{***}$ (df = 573), RMSEA = .034, CFI = .916, SRMR = .039). Standardized path coefficients showed that children whose mothers experienced greater stress at child age 3 were less likely to show social competence at age 5 ($\beta = -.239$, $p = .000$). However, children who showed secure attachment at age 3 demonstrated greater social competence at age 5 ($\beta = .088$, $p = .004$). Children's serotonin transporter genes, early temperament at age 1, and mothers' parenting mastery at age 3 did not significantly predict children's later social competence at age 5.

Next, considering that the interaction between two variables might be significant even when the direct effect of each predictor was not statistically significant to predict the outcome variable (Hayes, 2013), I added the interaction between a serotonin transporter

gene and another predictor among non-genetic predictors one by one. The results showed that children's 5-HTTLPR did not interact with infant temperament, mothers' parental adjustment in predicting children's later social competence. However, the interaction between children's 5-HTTLPR and early attachment at age 3 was marginally beyond the significance level of .05 (β = .108, p = .071; for more information, please see the Model 1 column in Table 3). In this model, as shown in Model 1 in Table 3, only the direct effect of children's early attachment (β = .066, p = .045) and the direct effect of mothers' parenting stress at age 3 (β = -.238, p = .000) were significant. In other words, preschoolers who had secure attachment in infancy or who raised by less stressed mothers in earlier periods were more likely to show social competence later on.

However, children's STin2 significantly moderated the effects of early attachment on children's social competence. As shown in Model 2 in Table 3, children who had the 10/10 genotype were less likely to develop social competence (β = -.200, p = .001) and those children were more likely to receive stronger effects from early attachment in developing their social competence (β = .227, p = .000). Like the model 1, the model 2 also showed that children who were raised by more stressed mothers showed significantly less social competence in later periods (β = -.235, p = .000). This model fit well ($\chi 2 = 1594.20^{***}$ (df = 615), RMSEA = .033, CFI = .917, SRMR = .038).

When I added the two interactions between early attachment and children's serotonin transporter genes (5-HTTLPR or STin2) together, all interactions appeared statistically significant. As shown in Model 3 in Table 3, children with the 10/10 genotype (β = .239, p = .000) on the STin2 or the S/S genotype on the 5-HTTLPR (β =

.129, p = .030) were more likely to receive greater effects of early attachment on positive social-emotional development. Figure 2 shows the plot of the interaction between children's 5-HTTLPR and early attachment and Figure 3 shows the plot of the interaction between children's STin2 and early attachment in this model. As we can see in Table 3, children were raised by more stressed mothers (β = -.234, p = .000) tended to show less social competence at age 5. Also, children with 10/10 genotype on STin2 (β = -.210, p = .001) were less likely to show develop social competence at age 5 but the interaction between children's STin2 and child early attachment was significant (β = .239, p = .000). The interaction showed that if children with 10/10 genotype on STin2 established secure attachment at age 3, they they were more likely to show social competence than others. However, if they showed insecure attachment at age 3, they tended to show lower social competence than others at age 5 (Figure 3). The model fit very well ($\chi 2 = 1625.85^{***}$ (df = 647), RMSEA = .032, CFI = .917, SRMR = .038).

Lastly, I was wondering whether these paths differed by child gender or children's race/ethnicities. Table 4 is the results of model comparison by child gender. Interestingly, when I conducted multi-group analysis, the model when all parameters were free ($\chi 2 = 2437.13$, df = 1323) was not significantly different from the model when I constrained all parameters equal across gender ($\chi 2 = 2486.36$, df = 1366; $\Delta\chi 2 = 49.23$, Δdf = 43). This means that all path coefficients from predictors to children's later social competence, factor loadings to construct latent variables, and two additional covariances between maternal parenting stress items were not significantly different by child gender.

Since the FFCWS oversampled low-income population, I also included poverty-related variables such as family poverty ratio, duration of family poverty, or the minimum age of the focal child when he/she was exposed to family poverty as control variables in the model. However, none of them was significantly related to the outcome variable. In other words, the inclusion of these variables did not bring any additional significant results and worsened the model fit. Thus, I decided not to include them in this study.

In terms of children's race/ethnicities, the original data had four groups: whites (17.54%), blacks (51.52%), Hispanics (22.61%), and others (8.33%). However, due to the small proportion of the last group, its inclusion brought the error message like "the sample covariance of the independent variables in this group was singular" in Mplus. So, I excluded the category of other/mixed races and ran the multi-group analysis for the three major racial/ethnic groups in the U.S.: whites, blacks, and Hispanics. As we can see in Table 5, the multi-group analysis results showed that the model when all parameters were free was not significantly different from the model when I constrained the path coefficients in the structural model across child race/ethnicities. However, this model with equal path coefficients across child race/ethnicities was significantly different from the model in which all parameters were equal across race/ethnicities. These results indicate that the paths from predictors to children's later social competence did not differ by child race/ethnicities although factor loadings were different by racial/ethnic groups.

Discussion

With the development of technology, gene and environment interaction studies have proliferated, and the attention to the debate between DS and DM is growing.

Proponents of the DS perspective distinguish their theory from DM in part by emphasizing the importance of revealing the positive relationship between having sensitive alleles and positive social-emotional outcomes under supportive environmental conditions. However, only a few studies testing the DS hypothesis examined positive behavioral outcomes. In the current study, I examined preschoolers' social competence as the outcome variable, and tested whether and how children's serotonin transporter genes interacted with children's early temperament, attachment, and mothers' parenting adjustment to predict children's later social-emotional development.

The results of this study showed that having a 10/10 genotype on the STin2 was significantly associated with lower social competence in later periods. The effect size and significance of children's STin2 as slightly lower than that of mothers' parenting stress. While having an S/S genotype on the 5-HTTLPR seems to increase women's risks of depression- or anxiety-related disorders and men's risks of externalizing behavior problems (Gressier, Calati, & Serretti, 2016), having an S/S genotype on the 5-HTTLPR was not directly associated with children's positive social-emotional development in this study.

I also found that children who were raised by less stressed mothers were more likely to develop social competence. Children's early attachment was significant to predict positive social-emotional development, but only in the model testing one interaction between 5-HTTLPR and early attachment. This direct effect disappeared when adding another interaction between STin2 and early attachment to the model. Thus,

we can infer that having sensitive alleles on the STin2 may explain some of the variation in children's early attachment predicting later social competence.

In terms of moderation effects of children's serotonin transporter genes, children with the 10/10 genotype on the STin2 or the S/S genotype on the 5-HTTLPR were more likely to receive greater influence of early attachment. This means that children with sensitive alleles on serotonin transporter genes are more likely to develop social competence in early childhood if they are securely attached to parents in infancy. If they experienced insecure attachment in infancy, however, they tended to have greater risk for deficits in social competence at preschool age. This is consistent with Humphreys, Zeanah, Nelson, Fox, and Drury (2015). After classifying child attachment at 42 months using Strange Situation Procedure and conducting semi-structured interview for children's externalizing behavior problems at 54 months using the Preschool Age Psychiatric Assessment, Humphreys et al. (2015) showed that children with the S/S allele on the 5-HTTLPR were more likely to show externalizing behavior problems later if they had insecure attachment. However, if children with the S/S allele established secure attachment with their favorite caregiver, then they showed the lowest level of externalizing behavior problems. This highlights the importance of establishing secure attachment in the very early stages of development, especially for children who have greater genetic sensitivity. Additional multi-group analyses supported that these results were not different by child gender or race/ethnicities. These are consistent with Sen, Burmeister, and Ghosh (2004)'s finding which showed no gender or racial differences in the association between 5-HTLPR and anxiety-related personality traits.

It was interesting to see that there were no other significant interactions between children's serotonin transporter genes and mothers' parental adjustment. It might be partly because environmental factors such as parenting stress had an obvious direct effect on children's social competence, so there was little room for one's genetic components to modify this relation. Another possibility is that other important indicators of the parenting environment are missing from this study. In fact, in FFCWS, there were some items about mothers' psychological aggression and physical assaults administered at the same wave (child age 3), but the reliabilities of those items were low, possibly because mothers rarely reported using psychological aggression and physical assaults (beyond spanking) on their 3-year-old children.

The interactions between children's serotonin transporter genes (5-HTTLPR, STin2) and early attachment seem to support the DS hypothesis. This is because children with two sensitive alleles on the serotonin transporter genes had greater changes in their social competence in later periods depending on the security of their attachment to mothers. However, when we look at the plots in Figures 2 and 3, one interesting finding is that the difference between two plots by children's genotype was greater in insecure group rather than in secure group. It infers that the disadvantage by unsupportive environment would appear more obviously rather than the benefit from supportive environment. This might explain the reason why researchers suggested DM first before thinking of DS. In fact, DM has been strongly supported in medical and clinical fields until today. Interestingly, a case supporting DM is also able to support DS with little conflict. Of course, given the low replication rates in gene and environment interaction

studies, we should consider the possibility that this case might not represent all the cases, especially considering the variation by population or child age. However, what I want to suggest here is that integrating the DS perspective with the DM perspective might make more sense than pitting them against one another. In the current study, the significant interactions can be interpreted using DM or DS approaches. For example, the explanation based on DS highlighting the "bright side" such that children with greater genetic sensitivity were more likely to develop greater social competence when they were securely attached in earlier periods is interchangeable with the explanation based on DM highlighting that children with greater genetic vulnerability were at greater risk for hindered social competence in later periods when they were insecurely attached in earlier periods.

There are several limitations of this study. First, this study used mother-report child temperament and social competence. Of course, mothers are reliable reporters because they take the responsibility of childcare and spend the majority of time to observe their children's behaviors. In addition, the findings of this study were consistent with previous research using observational measures. Nevertheless, considering low replication rates in gene and environment interaction studies, in order to strengthen the findings of this research, it would be better if future research can include other observational measurements for child temperament and child social competence and test the association between them and children's "positive" social-emotional outcomes to see whether this study is replicated or not. Future research may use Strange Situation assessment for child temperament as Humphreys et al. (2015) did, or try to do video

recording or teacher-report surveys for children's social competence to add more novel findings. Second, gene and environment interaction studies tend to show very low replication rates due to publication bias. Reviews about gene and environment interaction (i.e., Duncan & Keller, 2011) strongly recommend that the novel significant findings should be replicated in future research and that those findings should have greater statistical power or effect sizes. So, it would be great if future research attempt to replicate the findings of this study. Third, the findings of this study came from low-income population. Therefore, the findings of this study might not be applicable to different populations. According to MacCallum and Austin (2000), structural equation modeling is "a hypothesis about the structure of relationships among measured variables in a specific population. (p. 212)". Thus, understanding the population is critical to interpreting and to applying the results properly. Since the FFCWS oversampled non-marital births and targeted low-income populations in U.S. cities, the findings of this study might not be applicable to middle-high income populations or rural families. More research testing the same types of associations between similar variables in different populations is recommended.

Despite several limitations, however, this study has made a meaningful contribution to our understanding of the role of gene and environment interactions in children's social-emotional development. First, this study has broadened our knowledge by including STin2, which was often disregarded in previous gene and environment interaction studies, and by including children's social competence at preschool age as the outcome variable. Another strength comes from a relatively sample size. In structural

equation modeling, having a large sample is recommended (MacCallum & Austin, 2000), but it is not easy to collect genetic data from a large sample of a population. Given that the FFCWS collected genetic data from more than 1,000 children and longitudinal survey data from their mothers, it was ideal for testing G X E interactions in relation to children's social-emotional development.

Understanding children's social competence is worthwhile, because it can shed light on how to promote and support children's positive development. However, so far, it has not fully gained the same level of attention from researchers as behavior problems, for example, because it is easy to put the priority on the "emergency room". The focus on problematic behaviors instead of positive social-emotional outcomes is also partly because gene and environment interaction has been studied usually in psychiatry or clinical psychology, where more attention is put on psychological disorders or diseases. Growing interest in and attempts to test moderation effects of genetic characteristics by social scientists can contribute to revealing the mechanisms that underlie children's positive social-emotional development, which may in turn contribute to a better understanding of relations between positive social-emotional development, internalizing or externalizing behavior problems. The resulting more integrated understanding of children's social-emotional development can foster continued efforts to improve the development of prevention and intervention programs to better support the development of children with greater sensitivity to their environments from early ages.

Table 1. Final items and constructed latent variables in measurement model

Item	Description	Factor loading [b]	Latent variable (Reliability)
ASBI01	(he/she) plays games and talks with other children	.64***	Child social competence (α = .80)
ASBI02	(he/she) will join a group of children playing	.60***	
ASBI03	(he/she) is confident with other people	.59***	
ASBI04	(he/she) is interested in many and different things	.56***	
ASBI05	(he/she) asks or wants to go play with other children	.55***	
ASBI06	(he/she) tends to be proud of things (he/she) does	.53***	
ASBI07	(he/she) is open and direct about what (he/she) wants	.49***	
ASBI08	(he/she) can easily get other children to pay attention to (him/her)	.47***	
ASBI09	(he/she) is sympathetic toward children's distress, tries to comfort them	.46***	
ASBI10	(he/she) says 'please' and 'thank you' when reminded	.43***	
ASBI11	(he/she) understands others' feelings, like when they are happy, sad or mad	.40***	
ASBI12	(he/she) enjoys talking to you	.37***	
ASBI13	In social activities, (he/she) tends to just watch others [a]	.32***	
TEMP01	(child) gets upset easily	.74***	Child temperament (α = .60)
TEMP02	(child) reacts strongly when upset	.55***	
TEMP03	(child) often fusses and cries	.48***	
P_M01	I often feel helpless in dealing with problems [a]	.75***	Parental mastery (α = .81)
P_M02	There is little I can do to change many of important things in my life [a]	.69***	
P_M03	There is really no way I can solve some of the problems I have [a]	.67***	
P_M04	I have little control over the things that happen to me [a]	.67***	
P_M05	Sometimes I feel that I'm being pushed around [a]	.59***	
P_ST01	Do you find yourself giving up more of your life to meet child's needs than expected?	.80***	Parental stress (α = .87)
P_ST02	Are there quite a few things that bother you about your life?	.77***	
P_ST03	Do you enjoy things less than you used to?	.71***	
P_ST04	Are you less interested in people than you used to be?	.71***	
P_ST05	Do you often have feeling that you cannot handle things very well?	.70***	
P_ST06	Since having (child), are you almost never able to do things that you like to do?	.68***	
P_ST07	Since having (child), have you been unable to do new and different things?	.64***	
P_ST08	Do you feel trapped by your responsibilities as a parent?	.63***	
P_ST09	Do you feel alone and without friends?	.57***	
P_ST10	Are you unhappy with last purchase of clothing you made for yourself?	.50***	
P_ST11	When you go to a party, do you usually expect to have a bad time?	.45***	
P_ST12	Has having (child) caused more problems than you expected in the relationship with your male partner?	.40***	

Note. $^{*}p$ <.05, $^{**}p$ < .01, $^{***}p$ < .001.

a. Higher scores on this item indicate greater disagreement with the item.

b. These are standardized factor loadings when setting the variance of latent variable equal to 1.

Table 2. Covariance matrix for observed variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1	.209																
2	.114	.287															
3	.087	.106	.308														
4	.071	.078	.086	.205													
5	.097	.093	.085	.069	.263												
6	.055	.057	.073	.067	.059	.163											
7	.062	.075	.095	.060	.058	.050	.248										
8	.088	.112	.109	.073	.092	.047	.092	.420									
9	.066	.078	.097	.074	.070	.066	.077	.079	.369								
10	.066	.061	.056	.037	.068	.048	.045	.056	.058	.215							
11	.042	.046	.051	.052	.046	.047	.061	.036	.080	.041	.203						
12	.037	.033	.037	.034	.032	.036	.019	.031	.028	.031	.022	.104					
13	.057	.073	.074	.042	.051	.023	.041	.056	.052	.029	.030	.014	.336				
14	-.046	-.041	-.027	-.033	-.020	.001	-.019	.015	-.064	-.042	-.027	-.003	-.125	2.188			
15	-.012	.003	.002	-.004	-.030	.031	-.014	.024	-.026	-.001	.009	-.005	-.039	.894	2.154		
16	-.028	-.037	-.060	-.026	-.006	-.025	-.029	-.057	-.062	-.028	-.020	-.003	-.096	.694	.520	1.793	
17	.031	.039	.041	.030	.034	.030	.029	.042	.072	.025	.018	.012	.063	-.147	-.090	-.131	.651
18	.023	.043	.031	.025	.039	.030	.024	.047	.083	.034	.030	.012	.054	-.126	-.058	-.108	.336
19	.005	.029	.021	.011	.015	.028	.008	.020	.055	.025	.024	.010	.062	-.093	-.041	-.070	.332
20	.029	.037	.034	.024	.026	.035	.028	.043	.050	.039	.032	.021	.066	-.120	-.038	-.102	.316
21	.028	.044	.039	.007	.018	.018	.033	.011	.050	.021	.010	.013	.067	-.131	-.089	-.099	.328
22	-.060	-.017	-.064	-.022	-.013	-.047	-.040	-.016	-.099	-.052	-.022	-.028	-.081	.237	.115	.184	-.331
23	-.060	-.049	-.083	-.025	-.024	-.037	-.038	-.046	-.072	-.024	-.023	-.037	-.097	.237	.122	.246	-.422
24	-.047	-.045	-.072	-.038	-.045	-.046	-.041	-.070	-.073	-.038	-.033	-.024	-.086	.197	.100	.220	-.311
25	-.056	-.048	-.076	-.032	-.030	-.028	-.019	-.028	-.064	-.016	-.004	-.021	-.081	.224	.138	.159	-.322
26	-.057	-.061	-.059	-.042	-.052	-.038	-.069	-.058	-.096	-.055	-.036	-.035	-.089	.194	.053	.162	-.400
27	-.043	-.045	-.086	-.039	-.027	-.042	-.038	-.052	-.070	-.042	-.028	-.036	-.063	.146	.089	.083	-.221
28	-.025	-.029	-.045	-.023	-.012	-.026	-.037	-.057	-.080	-.023	-.021	-.022	-.061	.036	.111	.071	-.236
29	-.034	-.053	-.056	-.038	-.032	-.058	-.056	-.062	-.076	-.026	-.035	-.029	-.054	.130	.034	.077	-.290
30	-.057	-.045	-.058	-.035	-.033	-.043	-.024	-.062	-.046	-.033	-.031	-.023	-.053	.111	.034	.115	-.258
31	-.028	-.010	-.054	-.021	-.008	-.022	-.031	-.035	-.035	-.025	-.021	-.041	-.040	.042	.035	.137	-.238
32	-.057	-.066	-.058	-.047	-.048	-.033	-.023	-.078	-.071	-.030	-.034	-.023	-.057	.101	.045	.074	-.179
33	-.034	-.034	-.055	-.023	-.024	-.032	-.040	-.057	-.047	-.023	-.031	-.020	-.024	.091	.016	.066	-.151
34	-.001	.000	-.002	.007	-.002	-.005	.005	.001	-.004	.002	.000	.002	-.014	-.004	-.009	.011	-.012
35	.001	.000	-.002	.005	-.002	.000	.000	-.003	.004	.000	.003	-.001	-.003	-.010	.007	-.004	.002
36	.014	.013	.018	.011	.011	.013	.012	.010	.025	.015	.017	.004	.023	-.075	-.055	-.084	.039

Continued

Note. 1 = ASBI01, 2 = ASBI02, 3 = ASBI03, 4 = ASBI04, 5 = ASBI05, 6 = ASBI06, 7 = ASBI07, 8 = ASBI08, 9 = ASBI09, 10 = ASBI10, 11 = ASBI11, 12 = ASBI12, 13 = ASBI13, 14 = TEMP01, 15 = TEMP02, 16 = TEMP03, 17 = P_M01, 18 = P_M02, 19 = P_M03, 20 = P_M04, 21 = P_M05, 22 = P_ST01, 23 = P_ST02, 24 = P_ST03, 25 = P_ST04, 26 = P_ST05, 27 = P_ST06, 28 = P_ST07, 29 = P_ST08, 30 = P_ST09, 31 = P_ST10, 32 = P_ST11, 33 = P_ST12, 34 = 5-HTTLPR (*1 SS* vs. *0 others*), 35 = STin2 (*1 10/10* vs. *0 others*), 36 = child attachment (*1 secure* vs. *0 insecure*).

Table 2 Continued

18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36
.708																		
.401	.734																	
.344	.347	.710																
.240	.239	.274	.690															
-.312	-.262	-.259	-.268	2.118														
-.294	-.332	-.306	-.336	.619	1.484													
-.250	-.226	-.256	-.254	.575	.577	1.159												
-.226	-.179	-.198	-.305	.568	.600	.797	1.457											
-.254	-.268	-.325	-.300	.642	.620	.435	.426	1.327										
-.201	-.195	-.213	-.209	.645	.497	.507	.450	.458	1.164									
-.221	-.200	-.254	-.213	.699	.430	.445	.383	.462	.715	1.478								
-.182	-.205	-.212	-.215	.541	.462	.406	.369	.534	.508	.462	1.024							
-.194	-.156	-.228	-.221	.370	.400	.416	.432	.351	.381	.342	.337	.729						
-.183	-.174	-.153	-.213	.397	.444	.432	.441	.295	.337	.366	.242	.269	1.285					
-.157	-.114	-.139	-.166	.279	.269	.360	.413	.254	.274	.249	.236	.324	.226	.593				
-.126	-.103	-.154	-.157	.252	.266	.256	.251	.246	.265	.255	.284	.305	.168	.231	.545			
-.006	-.014	-.004	-.006	.010	-.020	.009	-.007	-.004	.009	.013	.011	.015	.005	.004	.003	.123		
-.002	.003	.001	.003	.002	-.014	-.014	-.005	-.009	-.001	-.002	-.008	.004	.000	.004	.000	-.010	.074	
.034	.033	.044	.030	-.043	-.059	-.044	-.041	-.053	-.041	-.032	-.038	-.025	-.018	-.030	-.021	-.005	.001	.184

Table 3. Structural equation modeling of serotonin transporter genes, attachment, temperament, parental adjustment to predict children's later social competence

	Children's social competence		
	Model 1	Model 2	Model 3
5-HTTLPR	-.086 (p = .149)	.008 (p = .787)	-.103 (p = .082)
STin2	-.001 (p = .964)	-.200** (p = .001)	-.210** (p = .001)
Attachment	.066* (p = .045)	.057 (p = .071)	.028 (p = .411)
Attachment x 5-HTTLPR	.108 (p = .071)		.129* (p = .030)
Attachment x STin2		.227*** (p = .000)	.239*** (p = .000)
Temperament	-.031 (p = .422)	-.031 (p = .422)	-.035 (p = .369)
Parental mastery	.032 (p = .521)	.035 (p = .474)	.035 (p = .472)
Parental stress	-.238*** (p = .000)	-.235*** (p = .000)	-.234*** (p = .000)
Model fit indices			
χ^2 (df)	1603.99*** (615)	1594.20*** (615)	1625.85*** (647)
RMSEA	.033 [.031, .035]	.033 [.031, .035]	.032 [.030, .034]
CFI	.916	.917	.917
SRMR	.038	.038	.038

*p <.05, **p < .01, ***p < .001. All are standardized models.

Table 4. Model comparisons across child gender

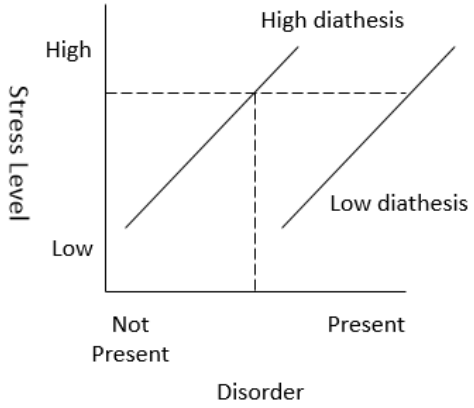
			Comparison to all-free model	
	χ^2	*df*	$\Delta\chi^2$	Δdf
All parameters free	2437.13	1323		
All parameters equal	2486.36	1366	49.23	43

Note. The models included all predictors and two interactions between early attachment and each child serotonin transporter gene (5-HTTLPR or STin2).

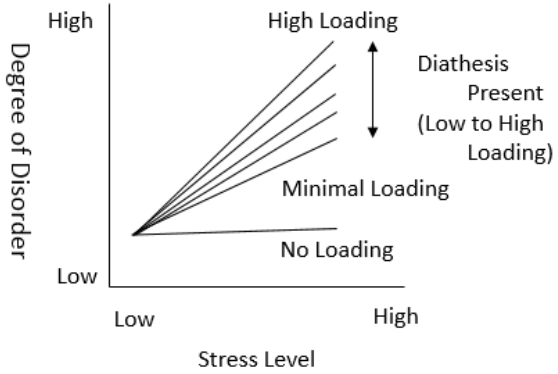
Table 5. Model comparisons across child race/ethnicity

			Comparison to all-free model		Comparison to equal-path-coefficients model	
	χ^2	*df*	$\Delta\chi^2$	Δdf	$\Delta\chi^2$	Δdf
All parameters free	3327.27	1999				
Coefficients only equal	3352.79	2015	25.52	16		
All parameters equal	3506.62	2085	179.35***	86	153.83***	70

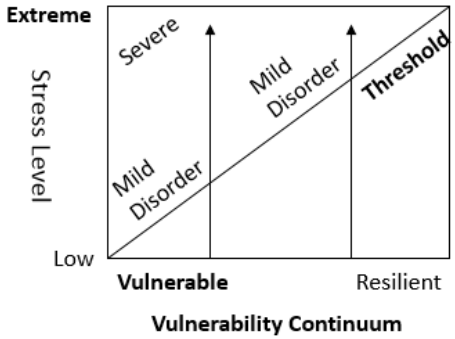
***$p < .001$. The models included all predictors and two interactions between early attachment and each child serotonin transporter gene (5-HTTLPR or STin2).



Additive Model



Interactive Model



Vulnerability-Resilience Model

Figure 1. Theoretical diathesis models

Note. Adapted from "Vulnerability-stress models," by R. E. Ingram, & D. D. Luxton, 2005, In B. L. Hankin & J. R. Z. Abela (Eds.), Development of psychopathology: A vulnerability-stress perspective, pp. 38-39, 41. Copyright 2005 by Sage.

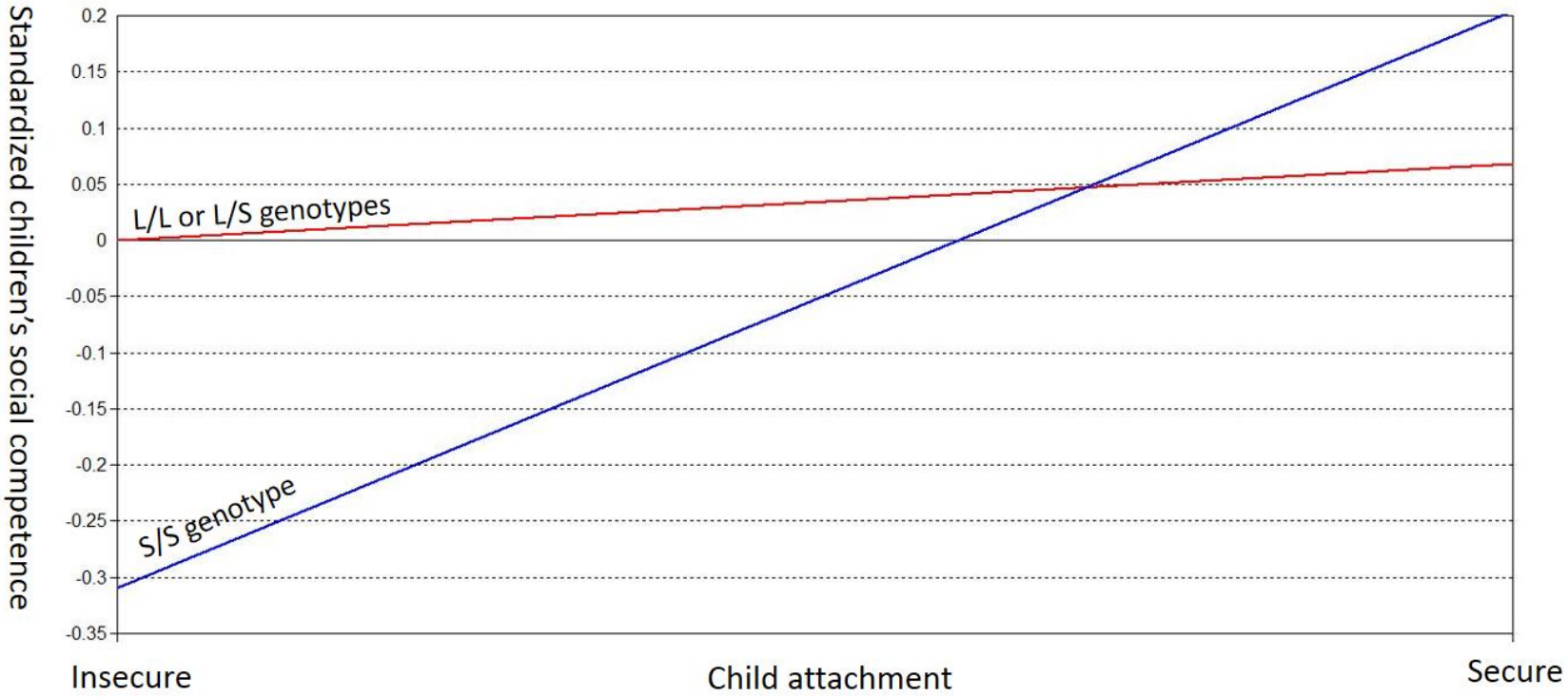


Figure 2. Interaction between children's 5-HTTLPR genotypes and child attachment in predicting later social competence

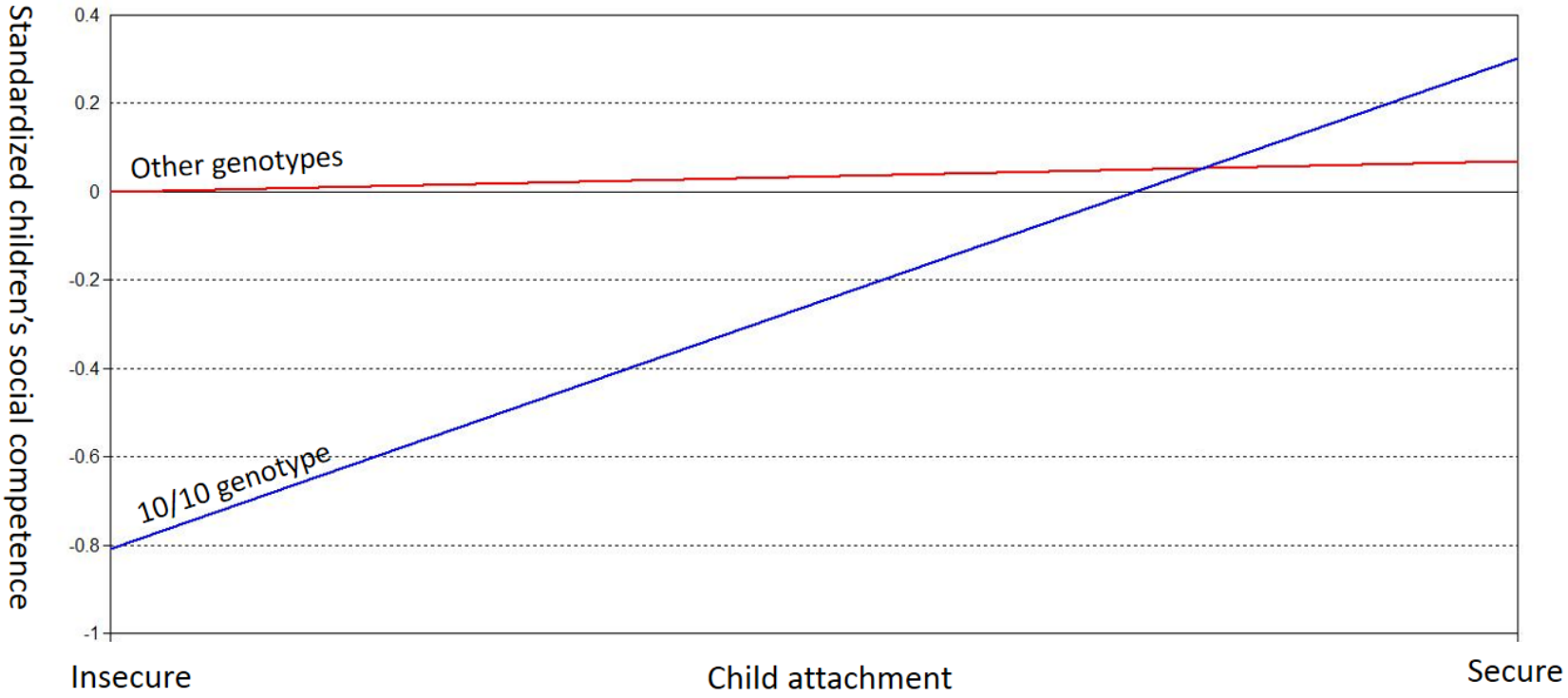


Figure 3. Interaction between children's STin2 genotypes and child attachment in predicting later social competence

Chapter 3. Through gene and environment correlation or interaction? The association between mothers' and children's genes, child temperament, mothers' psychological aggression, and children's internalizing behavior problems

Internalizing behavior problems can be defined as "a core disturbance in intropunitive emotions and moods" (Zahn-Waxler, Klimes-Dougan, & Slattery, 2000; p.443) and it is important to understand the factors which contribute the etiology of internalizing behavior problems. This is because anxiety or depression, typical types of child internalizing behavior problems, can worsen the quality of one's life. Compared to externalizing behavior problems which mean "behaviors that are harmful and disruptive to others" (Zahn-Waxler et al., 2000; p. 443), internalizing behavior problems harm oneself. Internalizing behavior problems in childhood are likely to develop adult mood or anxiety disorders (Pine & Grun, 1999). For example, a longituidnal empirical study (Caspi, 2000) showed that inhibited children at age 3 were more likely to have internalizing behavior problems at age 18, mood disorders at age 21, and suicide attempts by age 21. In childhood or adolescence, the comorbidity rates between internalizing and externalizing behavior problems are also pretty high (Wolff & Ollendick, 2006). Compared to externalizing behavior problems, children's internalizing behavior problems tended to appear later in early childhood (Basten et al., 2016). However, once internalizing behavior problems appeared, these children were more likely to experience internalizing behavior problems over time, and this trend was stronger for girls (Dekker

et al., 2007). Research on which factors contribute children's internalizing behavior problems including neurobiology can help to identify more risky individuals.

Child temperament and parents' parenting behaviors are two major predictors of children's internalizing behavior problems (Karreman, de Haas, van Tuijl, van Aken, & Dekovic, 2010; Lengua, 2006; Oldehinkel Veenstra, Ormel, de Winter, & Verhulst, 2006; Rubin, Burgess, & Hastings, 2002) and effortful control (Cipriano & Stifter, 2010). Inhibited infants were more likely to show social reticence at preschool age but this association was mediated by mothers' negative parenting behaviors. If mothers offered more intrusive controlling behaviors or contemptuous comments, the stability of infants' inhibition to social reticence at preschool age became stronger (Rubin et al., 2002). Young children who showed high fearfulness were also more likely to experience internalizing behavior problems in later periods and this association was strengthened by negative control from their parents (Karreman et al., 2010). It is hard to find longitudinal studies but there are some cross-sectional evidences showing that mothers' low emotional support strengthened the association between high fearfulness or low positive mood in child temperament and depressive symptoms in preadolescence (Betts, Gullone, & Allen, 2009; Oldehinkel et al., 2006). Considering child temperament is defined as individual biologically constructed differences in reactivity and self-regulation to environmental stimuli (Rothbart & Bates, 2007), it can be used to represent a phenotype of children's genetic characteristics. However, the specific roles of mothers' and children's genes on the mechanisms linking child temperament, mothers' parenting, and children's behavioral outcomes have not yet been fully revealed.

The Roles of Genes and Environments

In fact, gene and environment interplay consists of gene and environment interaction (G x E) and gene and environment correlation (rGE). Gene and environment interaction means children are affected differently by their environments depending on their genetic characteristics. For example, children with particular genes (i.e., "sensitive genes") might have better developmental outcomes than the average when they receive supportive parenting from parents, but these children will show more risky behaviors if exposed to negative parenting (Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007). Gene and environment interactions can be tested by adding the interaction term between children's genes and mothers' parenting when predicting children's behavioral outcomes. Feinberg, Button, Neiderhiser, Reiss, and Hetherington (2007) tested the moderation by negative parenting behaviors on the path from children's genotype to adolescent depression and antisocial behavior problems, but, interestingly, the interaction between children's genotype and parents' negative parenting behaviors was significant only for adolescent antisocial behaviors, not for depression.

While gene and environment interaction is useful to explain moderation paths like how children's genetic characteristics influence their sensitivity to environmental factors, gene and environment correlation is helpful to explain the mediation paths –like how mothers' or children's genes are associated with environmental factors that in turn predict child behavioral outcomes. In fact, previous research (Eaves, Sillberg, & Erkanli, 2003) showed that genes related to early anxiety were also associated with children's developmental processes of depression, which supported both gene and environment

interaction and correlation. Gene and environment interaction was supported because children with sensitive genes to early anxiety tended to be more likely to experience negative effects from adverse life events (G x E). Gene and environment correlation was also supported because children who had sensitive genes to early anxiety were more likely to be exposed to more risky environments (rGE). One interesting finding was that if the researchers omitted either gene and environment interaction or gene and environment correlation, the risk of overestimating the influence of environments increased (Eaves et al., 2003). This implies that researchers need to include gene and environment correlation in addition to gene and environment interaction if we want to improve our understanding about the roles of genetic and environmental factors in children's developmental processes.

In terms of gene and environment correlation, there are three types: passive, active, and evocative correlation (Plomin, DeFries, & Loehlin, 1977; Rutter, 2006). Passive gene and environment correlation highlights parent-oriented effects. It assumes mothers who passed on sensitive genes to their children via inheritance are also likely to provide negative parenting behaviors; thus, the children of these mothers are at higher risk for developing internalizing (e.g., anxious or withdrawn) behavior problems. Contrary to passive correlation, active gene and environment correlation focuses on child-driven effects. It assumes children's sensitive genes lead children to experience more behavior problems regardless of parents' genes. For example, children with sensitive genes may be prone to problematic or risky behaviors. For young children in infancy or early childhood, difficult temperament can be a part of active gene and

environment correlation, because difficult temperament is a manifestation of children's tendencies to experience and act on their environments in particular ways. Different from these two types of correlation, evocative gene and environment correlation assumes that children's sensitive genes will elicit negative responses from surrounding people (via their phenotypic manifestation; e.g., difficult temperament) so children with sensitive genes are more likely to develop internalizing behavior problems by eliciting and experiencing negative cycles in their interactions with parents.

Based on these three gene and environment correlation concepts (Plomin et al., 1977), Scarr and McCartney (1983) developed three propositions: (1) The three types of gene and environment correlations can occur simultaneously in children's developmental processes; (2) The relative effects of the three types of gene and environment correlations change over time – for example, the effects of passive gene and environment correlations decrease over time but the effects of active gene and environment correlations will increase as children grow up; and (3) The effects of environmental factors increase over time as children have more chance to choose their own environments as they develop physically, socially, and emotionally.

The Roles of 5-HTTLPR and TPH2

A number of previous studies have attempted to identify candidate genes that underlie the three types of gene-environment correlations in relation to the development of internalizing behavior problems. Although it is still unknown about all the functions of specific genetic variants, the serotonin transporter polymorphism (5-HTTLPR) is the most investigated gene in relation to internalizing pathology like depression (Munafò,

2012). This is because serotonin transporter (5-HTT) in human brain regulates transmission of serotonin into neurons, which affects individual fear- and anxiety-related traits (Becker, El-Faddagh, Schmidt, & Laucht, 2007; Lesch et al., 1994). Compared to people who have long alleles on the 5-HTTLPR, people who have one or more short alleles on the 5-HTTLPR showed the reduction of 5-HTT expression, and therefore are more likely to feel greater fear or anxiety with the increase of amygdala activity (Hariri et al., 2002). In terms of active gene and environment correlation, previous research reported children's 5-HTTLPR was associated with child temperament (Auerbach, Faroy, Ebstein, Kahana, & Levine, 2001), child behavior problems (Jorm et al., 2000), and depression in adulthood (Caspi et al., 2003). However, evidence regarding the directions of the effects of having a sensitive allele in 5-HTTLPR were mixed. Generally, individuals with one or more short alleles were more likely to have depressive symptoms and suicidal ideation or suicide attempts when they experienced stressful life events (Caspi et al., 2003). But this finding was not replicated for adolescent children. Children who were 13-14 or 15-16 years old without any short alleles (L/L genotype) tended to report higher anxiety than other children (Jorm et al., 2000). That might be because a short allele on the 5-HTTLPR is related to children's inhibition in expressing their emotions. In fact, infants with two short alleles (S/S genotype) were significantly less likely to express fearful distress than other children in an experiment using stranger approach, but they were also less likely to express positive emotionality than other children. Children with two short alleles tended to have longer latencies to smile or to express their fear for the first time (Auerbach et al., 2001).

With regard to passive correlation, few studies have tested directly passive gene and environment correlation to explain the development of children's internalizing behavior problems. Several animal studies reported that mother mammalian (Carter, 1998), rodents (Lim & Yong, 2006), or sheep (Keverne & Kendrick, 1992) with higher oxytocin levels were more likely to show nurturing behaviors toward offspring and lower stress levels. Focused on this and and the role of serotonergic system which can buffer the effects of life adversity on depression and might influence the release of oxytocin, Bakermans-Kranenburg and van IJzendoorn (2008) examined how mothers' 5-HTTLPR, oxytocin receptor (OXTR), the interaction between 5-HTTLPR and OXTR were associated with parenting behaviors. Their results showed that mothers who had two short alleles (SS genotype) on 5-HTTLPR tended to provide less sensitive parenting toward their children than mothers who had two long alleles or were heterozygous (LL or LS genotypes). Mothers' oxytocin receptor (OXTR) was also significantly associated with mothers' sensitive parenting behaviors, but the interaction between mothers' 5-HTTLPR and OXTR was not significant.

The role of evocative gene-environment correlations in children's internalizing problems, is more complicated to test, but substantiated both theoretically and empirically. In his classic model of the determinants of parenting, Belsky (1984) proposed child characteristics (e.g., child temperament, influenced by children's genes) as one of three major classes of predictors of parenting behavior in addition to parents' individual characteristics (e.g., personality, influenced by parents' genes) and family relational or contextual factors (e.g., social support or stressful environments). Martini,

Root, and Jenkins (2004) demonstrated that child temperament was significantly associated with mothers' regulation of hostile (i.e., anger) and non-hostile negative emotions (i.e., anxiety or sadness). By comparing monozygotic (MZ) twins and dizygotic (DZ) twins, Forget-Dubois and colleagues (2007) also supported partially the hypothesis that children's heritable characteristics elicit mothers' hostile-reactive behaviors. These studies did not include directly children's genes. However, Klahr and Burt (2014) conducted a meta-analysis of behavioral genetic research and showed that not only mothers' genes but also children's genetic make-ups were significantly associated with mothers' parenting behaviors even if the effect size was smaller than other environmental factors. These studies have supported the existence of evocative gene and environment correlations by examining the paths from children's genetic characteristics to mothers' parenting behaviors.

Compared to 5-HTTLPR, little research has examined the role of the tryptophan hydroxylase-2 (TPH2) gene in the development of children's internalizing behavior problems. Similar to 5-HTTLPR, however, the TPH2 gene is also associated with individual responses in the brain to modulate emotional stimuli (Canli, Congdon, Constable, & Lesch, 2008; Gutknecht et al., 2007). In addition, those who had one or more T alleles on the TPH2, or one or more short alleles on the 5-HTTLPR, were more likely to be fearful than others (Canli et al., 2008). The TPH2 gene has also reported as a predictor of panic disorder (Kim, Lee, Yang, Hwang, & Yoon, 2009). Thus, it is worthwhile to investigate the TPH2 gene in the context of examination of gene-

environment interplay in the development of internalizing behavior problems in childhood.

The Present Study

The central goal of this study was to gain a more integrated understanding of how mothers' and children's genes are associated with child temperament, mothers' parenting, and children's later internalizing behavior problems. As shown in Figure 4, this study first tested gene and environment correlation and interaction models separately. Subsequently, I constructed a final model by incorporating all significant paths and testing specific indirect paths.

This study focused on middle childhood (at 9 years old) because it is possible for children to think logically based on perceived information from this period (Piaget, 1971). In this study, given that the outcome variables were children's internalizing behavior problems at age 9, mothers' psychological aggression at previous wave (age 5) was used as a predictor of mothers' parenting. Children's genes were from saliva samples collected at age 9 but genotypes on the serotonin genes were consistent from children's birth. Child temperament measured at age 1 was used as an indicator of children's selective behaviors to shape their environments, to acknowledge and to respond to environments (Rothbart & Bates, 2007).

To test the existence of gene and environment interaction and passive, active, and evocative gene and environment correlations, I developed four different models. To test passive gene and environment correlation, the first model examined whether mothers' genes predicted children's genes, and whether mothers' genes predicted their parenting

behaviors toward children. To test active gene and environment correlation, the second model examined whether children's genes predicted child difficult temperament and children's later internalizing behavior problems when controlling for mothers' genes. To test evocative gene and environment correlation, the third model tested whether children with sensitive genes were more likely to receive mothers' harsh parenting, and whether this in turn was associated with children's later internalizing behavior problems.

In other words, I estimated gene and environment correlation by investigating (1) whether children's genes directly predicted child difficult temperament and their later internalizing behavior problems when controlling mothers' genes, (2) whether mothers' genes directly predicted their negative parenting behaviors and children's later internalizing behavior problems when controlling children's genes, and (3) whether mothers' parenting was predicted by children's genes, and whether mediation by mothers' parenting explained the path from children's genes to their later internalizing behavior problems. To test gene and environment interaction, the last model examined the interaction between children's genes and mothers' parenting to predict children's later internalizing behavior problems.

For mothers' and children's genes, I tested 5-HTTLPR and TPH2 rs4570625 based on previous research reporting that these genes are associated with individual differences in emotional dysregulation. For mothers' parenting, I tested mothers' psychologically aggressive parenting behaviors because of the prevalence among American parents (Straus & Field, 2003). About 90% of parents in a nationally representative sample reported they utilized psychological aggression toward children by

child age 2 and 98% of them did by child age 5. Although 10-20% of toddlers among them experienced severe psychological aggression, the prevalence rate of overall parental psychological aggression was fairly high and there was no difference in the prevalence of psychological aggression by family characteristics (Straus & Field, 2003). In this study, mothers' and children's serotonin genes (5-HTTLPR, TPH2 rs4570625), child temperament, mothers' psychological aggression, and children's internalizing behavior problems (e.g., anxious or withdrawn) are key variables. In addition to them, child gender, number of children under 18 years old in the household, the duration of family poverty, and the minimum poverty ratio are covariates. Family environment such as family poverty or family instability might work as confounding factors in estimating the effects of child temperament or mothers' parenting behaviors on child internalizing behavior problems. In fact, Lengua (2006) showed that family income was significantly associated with the initial level of child temperament (fear, irritability, and effortful control) and parents' negative parenting behaviors (rejection and inconsistent discipline). And family structure (single-parent status) was related to higher levels of irritability and effortful control in child temperament at the initial wave. Thus, I included poverty-related variables and changes in family structure in the model. Specifically, I used multiple measurements of family poverty including its severity and duration. For family instability, the number of family structure changes after the focal child's birth were assessed. Considering gender difference in the etiology of internalizing behavior problems (Leve, Kim, & Pears, 2005), I also included child gender as another covariate.

Research Questions and Hypotheses

1. Passive rGE: Did mothers with sensitive genes provide more psychologically aggressive parenting, and did it lead to higher risk of children's internalizing behavior problems?

2. Active rGE: Did children with sensitive genes show more difficult temperament, and did it lead to higher risk of internalizing behavior problems regardless of mothers' genes?

3. Evocative rGE: Did children with sensitive genes evoke mothers' more psychologically aggressive parenting via child temperement, and did it lead to higher risk of children's internalizing behavior problems?

4. G x E: Did children with sensitive genes receive different effects of mothers' psychologically aggressive parenting on internalizing behavior problems?

I anticipated that the associations between mothers' and children's genes, mothers' parenting, and children's internalizing behavior problems would support all rGE (passive, active, and evocative) and G x E assumptions. Therefore, I tested four hypotheses. First, I expected that mothers with sensitive genes would provide more psychologically aggressive parenting, and increase children's internalizing behavior problems. Second, I hypothesized that children with sensitive genes would show greater difficult temperament in infancy and subsequently develop more internalizing behavior problems. I expected that this path would be significant even after controlling mothers' psychological aggression. Third, I hypothesized that greater difficult temperament of children with sensitive genes would increase mothers' parenting stress and reduce

parenting mastery, and therefore those children would show greater internalizing behavior problems. Fourth, I expected that if children with sensitive alleles were exposed to mothers' psychological aggression, then they would show greater internalizing behavior problems than other children. However, if children with sensitive alleles did not experience mothers' psychological aggression, I predicted that they would show lower internalizing behavior problems than children without sensitive alleles.

Method

Data and Analytic Sample

This study analyzed the data from 2,646 mothers and their 2,646 children who participated in the saliva sample collection as part of the Fragile Families and Child Wellbeing study (FFCWS). The original FFCWS study recruited 4,898 couples at child birth and followed them up from child birth to 1, 3, 5, and 9 years old. Mothers reported child gender at child birth and temperament at children's age 1. Primary caregiver reported children's behavior problems at home visits at children's age 5 and 9. Considering the majority of primary caregivers were children's biological mothers (about 90% of the whole sample in FFCWS), I limited the sample to children whose primary caregivers were biological mothers to reduce reporters' biases. During the 5-year home visit, an interviewer also asked primary caregivers about their parenting behaviors toward children. At the 9-year home observation, focal children and their biological mothers were asked to participate in the saliva sample collection for genetic analysis. Collected saliva samples were mailed by the interviewers to Westat. Westat shipped specimen containers to the Molecular Biology lab at Princeton university. DNAs were extracted by

the Oragene Laboratory Protocol Manual Purification of DNA®. After adding Oragene® DNA Purifier into the microcentrifuge tube, the samples were incubated to extract clear supernatant. On the supernatant, 95-100% ethanol was added and the samples were left for 10 minutes at room temperature. Then, the samples were put in the centrifuge and DNAs were fully precipitated at 4,000 rpm at room air. After removing supernatant, 70% of ethanol wash was added. After incubating for 1 minute at room temperature, the ethanol was removed to get rehydrated DNAs. After another vortexing and incubating process, the rehydrated DNAs were transferred to microcentrifuge tubes for storage (FFCWS, 2015).

Table 6 shows demographic characteristics of the sample in this study. The average age of mothers was 25.03 years old (std. = 5.92, min. = 15, max. = 43) at focal childbirth and the average age of fathers was 27.58 years old (std. = 7.05, min. = 15, max. = 53). The education levels of mothers and fathers were not high. 32.87% of mothers' education level was lower than high school and 31.39% of mothers had the equivalent level of high school degree. 25.23% of mothers graduated 2-3 years colleges or technical schools and 10.51% of mothers graduated 4-year colleges or graduate schools. For fathers, 37.17% of fathers' education level was equal to high school degree and 32.53% of fathers had lower degree than high school. 20.54% of fathers graduated 2-3 years colleges or technical schools and only 9.76% of fathers had a bachelor's or higher degree. Among 2,645 couples, 24.01% were married, 36.67% were cohabiting, and 39.32% were nonresident at childbirth. However, when the focal children were 9 years old, 30.19% were married, 10.18% were cohabiting, and 59.63% couples did not live together. The

average of mothers' household income was $32,720 at childbirth but increased to $45,321 after 9 years. In terms of race, 47.75% of mothers were African-American, 28.12% of mothers were Hispanic, 20.99% of mothers were European-American, and only 3.15% of mothers were mixed or other races. For fathers, 50.38% were African-American, 27.58% were Hispanic, 18.43% were European-American, and 3.61% were mixed or other races. Among 2,646 children, girls were 51.47% and boys were 48.53%.

Measures

Children's internalizing behavior problems. For children's internalizing behavior problems, I created two mean scores: one represented withdrawn behavior problems and the other one represented anxious behavior problems. During the 9-year home visit, an interviewer asked the primary caregiver about children's behavior problems based on items from the Child Behavior Checklist (CBCL 4-18; Achenbach, 1992). Based on the CBCL 4-18, I created a mean score for children's withdrawn behavior problems using 9 items ($\alpha = .74$): "(child) would rather be alone than with others", "(child) refuses to talk", "(child) is secretive, keeps things to self", "(child) is shy or timid", "(child) stares blankly", "(child) sulks a lot", "(child) is underactive, slow moving, or lacks energy", "(child) is unhappy, sad, or depressed", and "(child) is withdrawn, doesn't get involved with others". With regards to children's anxious behavior problems, another mean score was created based on the CBCL 4-18, using 13 items ($\alpha = .79$): "(child) fears he or she might do something bad", "(child) feels he or she has to be perfect", "(child) feels or complains that no one loves him or her", "(child) feels others are out to get him or her", "(child) feels worthless or inferior", "(child) is nervous,

high strung, or tense", "(child) is too fearful or anxious", "(child) feels too guilty", "(child) is self-conscious or easily embarrassed", "(child) is suspicious", "(child) worries", "(child) complains of loneliness", or "(child) cries a lot". The original scale for each item ranged from *1 not true* to *3 very true or often true.* However, following the FFCWS user guide, I recoded the items from *0* to *2* so that the mean score would be 0 if children did not show any withdrawn or anxious behavior problems.

Child temperament. When the focal children were 1 year old, mothers were asked about child temperament on the telephone interview. Based on the Emotionality sections of the Emotionality, Activity, and Sociability Temperament Survey for Children (Mathiesen & Tambs, 1999), a mean score was created using 3 items ($\alpha = .60$) to represent difficult temperament: "(child) often fusses and cries", "(child) gets upset easily", and "(child) reacts intensely when upset". Each item was scored from *1 not at all* to *5 very much*.

Mothers' and children's genes. In terms of genes, mothers' and children's serotonin transporter (5-HTTLPR) and tryptophan hydroxylase (TPH2 rs4570625) were genotyped. In previous research, 5-HTTLPR was coded according to whether the genotype had one or more S allele(s) or no S allele. In this study, I followed this dominant coding so SS and LS genotypes were coded as 1 (indicating higher sensitivity), and the LL genotype was coded as 0 (indicating lower sensitivity). For TPH2 rs4570625, having at least one or more T allele(s) was coded as 1, whereas those without a T allele were coded as 0.

Mothers' psychological aggression. Mothers reported the frequency of their psychological aggression toward the focal child during the past year to an interviewer at the 5-year home visit. Based on the Parent-Child Conflict Tactics Scales (Straus et al., 1998), a mean score was created using 5 items ($\alpha = .61$): "(How many times in the past year did you) shout, yell, or scream at child", "threaten to spank/hit child-did not actually do", "swear or curse at (him/her)", "call (him/her) dumb or lazy/some other name like", and "say you would send child away/kick out of the house." The choices were (1) once in the past year, (2) twice, (3) 3-5 times, (4) 6-10 times, (5) 11-20 times, (6) more than 20 times, (7) yes, but not in the past year, and (0) this has never happened. Considering that the focus of the questions was on the past year, I recoded (7) as (0).

Covariates

Child gender. At the initial wave, biological mothers reported focal children's gender. It was originally coded as (1) a boy or (2) a girl as a categorical variable but I recoded it as a binary variable (0) a girl or (1) a boy for easy interpretation. Girls were 48.53% and boys were 51.47% of the sample.

Family instability. To account for family instability, I summed the number of changes in the mother's relationship with the child's biological father between waves. For example, if a mother lived together with the focal child's biological father at wave 1, but they did not live together at wave 2, the number of changes became 1. Since there were some couples who repeated separation and re-union, I coded as 1 if there was a change from wave T to wave T + 1. And then, I added the numbers from wave 1 (child birth) to wave T to produce the number of changes in total. This was treated as a time-

varying variable. Therefore, if it was used as a predictor of children's internalizing behavior problems at age 9, this variable meant the total number of changes by children's age 9. But, if it was used as a predictor of mothers' psychologically aggressive parenting behaviors at age 5, this variable meant the total number of changes by children's age 5. If it was used as a predictor of child temperament at age 1, then it meant the number of changes from child birth (wave 1) to child age 1.

The severity and duration of family poverty. Like family instability, the severity and duration of family poverty were also treated as time-varying covariates. Mothers reported the household income in the core telephone interview at the focal child's birth and again when the focal child was 1, 3, 5, and 9 years old. The U.S. Census established the family poverty thresholds considering household size and annual inflation rates. The FFCWS constructed the new variable of income-to-poverty ratio at each wave by dividing mother-reported household income by the corresponding family poverty threshold. Higher income-to-poverty ratio means greater financial resources of the household. Therefore, this study will use the minimum value of the income-to-poverty ratio from childbirth to children's age T as the measure of family poverty severity from infancy to childhood.

In addition to the severity of family poverty, it is also important to consider the duration of family poverty. The duration of family poverty was measured by subtracting the child's age when the income-to-poverty ratio first dropped below 200% from the child's age at the last time point when the income-to-poverty ratio continued to be below 200%. If these variables were used as predictors of children's internalizing behavior

problems at age 9, then these meant the severity and duration of family poverty by age 9. If they were used as predictors for mothers' psychologically aggressive parenting at age 5, then these were indicators of family poverty by age 5. If these were used as predictors of child temperament at age 1, then these indicated the severity and duration of family poverty from child birth (wave 1) to age 1.

Number of children under 18 years old in the household. This variable indicated how many children under 18 years old were living together in the household. This was also treated as a time-varying covariate. Thus, if it was used as a predictor of children's internalizing behavior problems, it indicated the number of children in the household at age 9. If it was a predictor for mothers' psychological aggression at age 5, it meant the number of children at age 5. If it was used as a predictor of child temperament, then it was the number of children at home at age 1.

Analytic Plan

I used STATA 14.2 for data cleaning and Mplus 8.0 for data analysis. To test gene and environment interaction as well as gene and environment correlations, sequential mediator models were tested in Mplus 8.0. For mediation models, I used bootstrapping methods to evaluate the existence of a significant mediation in the model. Also, I used the option "MODEL INDIRECT" with "IND" commands in Mplus 8.0 when testing which specific mediation paths were significant in this multiple sequential mediator model, following Muthén, Muthén, and Asparouhov (2016). For the moderation model of gene and environment interaction, I tested the original model without any interaction to see the association between each predictor and the outcome variable and

then, added three interactions: (1) child 5-HTTLPR x mothers' psychological aggression, (2) child TPH2 x mothers' psychological aggression, and (3) child temperament x mothers' psychological aggression in the model in Mplus 8.0.

To see which path model was acceptable, I used model fit indices such as $\chi 2$ (degree of freedom), RMSEA, CFI, and SRMR (Hooper, Coughlan, & Mullen, 2008). Chi-square shows the sample's discrepancy from fitted covariance matrices (Hu & Bentler,1999) but it is sensitive to sample size. So, nonsignificant chi-square indicates a good overall model fit but it is often violated especially when using a large sample (Hooper et al., 2008). In terms of RMSEA, it shows how much the estimated model is close to an ideally fitted population covariance matrix (Byrne, 1998). It is acceptable if RMSEA is less than .06 (Hu & Bentler, 1999) or .08 (MacCallum et al., 1996). CFI indicates the Comparative Fit Index (Bentler, 1990). Considering its sample size, CFI compares the sample matrix to the null model which assumes no correlation between all latent variables (Hooper et al., 2008). A CFI greater than .90 indicates a good model fit. Lastly, SRMR compares the residuals of the estimated model to the residuals of an ideal model (Hooper et al., 2008). An SRMR lower than .05 indicates a good model fit (Byrne, 1998). When I examined the missing data, the assumption of missing at random (MAR) was not violated. I used Full Information Maximum Likelihood (FIML) estimation.

Results

As shown in Table 7, children's withdrawn behavior problems were correlated with anxious behavior problems (r = .68). Also, the minimum of poverty ratio by age 9 which indicated the severity of family poverty was correlated with the duration of family

poverty by age 9 (r = -.59). Given that the poverty ratio indicated socioeconomic resources of the household, a lower minimum poverty ratio by age 9 indicated more severe poverty. Thus, this is the reason why the correlation between minimum poverty ratio and the duration of family poverty was negative. Except these two correlations, all other correlations were modest ($r < .30$).

Table 8 shows the path analysis results when I tested passive gene and environment correlation in terms of the mechanism of child internalizing behavior problems. This passive rGE model fit well ($\chi^2 = 83.767$, $df = 43$, $p < .001$, RMSEA = .023, CFI = .978, SRMR = .015). The results showed that mothers' 5-HTTLPR as well as TPH2 rs4570625 were inherited by children ($\beta = .461$, $p < .001$ for 5-HTTLPR, $\beta = .394$, $p < .001$ for TPH2 rs4570625). Contrary to the assumption, mothers who had one or more T allele(s) on the TPH2 rs4570625 were less likely to show psychological aggression to their children in early childhood. The effect of mothers' TPH2 genes on psychologically aggressive parenting was greater than that of other predictors such as the number of non-adult children in the household or the frequency of changes in family structure. Unlike TPH2, there was no statistically significant differences in mothers' psychological aggression between mothers who had sensitive allele(s) and those who did not have any sensitive allele(s) on the 5-HTTLPR.

Mothers' psychological aggression in early childhood (age 5) was a highly significant predictor of children's later withdrawn behavior problems ($\beta = .105$, $p < .001$) and anxious behavior problems ($\beta = .097$, $p < .001$) at age 9. Children's genes, temperament, the number of non-adult children in the household, family instability, the

severity of family poverty ratio, and the duration of family poverty were also included to control the effects of these variables on child internalizing behavior problems. Among them, only child temperament and poverty duration were significant predictors of children's withdrawn behavior problems. For children's anxious behavior problems, children's 5-HTTLPR, child temperament, and the number of non-adult children in the household were significant predictors of anxious behavior problems. However, these covariates did not significantly predict child internalizing behavior problems as strongly as did mothers' psychological aggression.

When I tested the child-oriented model based on active gene and environment correlation, the results were like Table 9. This model fit well (χ^2 = 19.583, *df* = 5, *p* <.01, RMSEA = .043, CFI = .985, SRMR = .016). Children's withdrawn behavior problems (β = .011, *p* < .05) as well as anxious behavior problems (β = .014, *p* < .05) at school age (age 9) were predicted by child temperament in infancy (at age 1). Moreover, child temperament was predicted by children's TPH2 rs4570625 (β = .143, *p* < .05). In particular, children who had one or more T alleles were more likely to show difficult temperament in infancy. Children's 5-HTTLPR was not a significant predictor of child temperament (β = -.106, *p* = .059), but it was directly associated with children's anxious behavior problems at school age (β = .028, *p* < .05). Additionally, I also tested the model after including mothers' 5-HTTLPR and TPH2 as predictors of child temperament, but none of mothers' genes were significantly associated with child temperament. Therefore, these results seemed to support active gene and environment correlation, which assumes

that children's genetic characteristics lead children to develop more internalizing behavior problems regardless of the mothers' genetic make-up.

Next, I tested the paths of evocative gene and environment correlation. Considering that child temperament can be used as an indicator of phenotype of children's genetic characteristics, I tested whether the effects of children's genetic characteristics on mothers' psychological aggression in early childhood were mediated by child temperament in infancy, and whether the effects of infant temperament on children's internalizing behavior problems at school age were mediated by mothers' psychological aggression at preschool age. This evocative gene and environment correlation model fit well (χ^2 = 49.457, *df* = 16, *p* <.001, RMSEA = .032, CFI = .974, SRMR = .021). In this model, not only children's TPH2 rs4570625 (β = .131, *p* < .01) but also 5-HTTLPR (β = -.105, *p* < .05) were significant predictors of child temperament in infancy. Interestingly, however, the nature of the associations of these two genes with child temperament were different. Children who had one or more T alleles on the TPH2 gene were more likely to show difficult temperament, but children with one or more short alleles on 5-HTTLPR were less likely to show difficult temperament. As I anticipated, children who showed more difficult temperament in infancy tended to receive greater psychological aggression from mothers in early childhood (β = .073, *p* < .01). In turn, children who received greater psychological aggression from mothers in early childhood were more likely to develop withdrawn (β = .025, *p* < .001) or anxious (β = .023, *p* < .001) behavior problems at school age. Child temperament also directly predicted

children's withdrawn (β = .012, $p < .05$) or anxious (β = .016, $p < .01$) behavior problems.

Lastly, when I tested gene and environment interactions, none of the interaction terms including children's genes and mothers' negative parenting behaviors were significant predictors of children's withdrawn and anxious behavior problems. In this G x E model, the three interaction terms that I tested were the moderations by mothers' psychological aggression from children's genes (5-HTTLPR or TPH2 rs4570625) or child temperament (as a phenotype of children's genetic characteristics) to child internalizing behavior problems at school age. The results of non-significant interaction terms implied that the effects of mothers' psychological aggression on the development of children's withdrawn or anxious behavior problems did not differ by children's 5-HTTLPR, TPH2 rs 4570625, or child temperament. This G x E model was a saturated one in which all possible parameters were estimated so there was no room left to estimate variance (RMSEA = .00, CFI = 1.00, SRMR = .00).

Based on all the results from the passive, active, and evocative rGE models as well as the G x E model, I tested the final model with multiple sequential mediators to obtain more integrated information about how genes work in the developmental processes of children's internalizing behavior problems. Due to the rule of parsimony for the best model, this model included only significant paths from the above results. There was only one exception for family instability. In the model of evocative gene and environment correlation, family instability seemed to predict mothers' psychological aggression significantly. But when I included it in the final model with other significant

paths from different rGE models, family instability became non-significant (β = .045, p = .066). So, I decided to exclude it in the final model. As we can see in Figure 5, the final model included all other significant paths.

Lastly, I tested each indirect path with the option of "IND" under the MODEL INDIRECT command in Mplus to examine which indirect paths were significant and which were not. For passive rGE, the indirect path from mothers' TPH2 rs4570625 to children's withdrawn behavior problems at age 9 mediated by mothers' psychological aggression at age 5 was significant (β = -.009, $p < .05$) and the indirect path to children's anxious behavior problems with the same predictor and the same mediator was also significant (β = -.009, $p < .05$). Since mothers with one or more T alleles were less likely to show psychological aggression when mothers' greater psychological aggression was related to children's greater withdrawn or anxious behavior problems, the indirect paths have negative coefficients. Also, the indirect path mediated by children's 5-HTTLPR from mothers' 5-HTTLPR to children's anxious behavior problems was significant (β = .022, $p < .01$). It means that children who inherited sensitive alleles on 5-HTTLPR from their biological mothers were at greater risk of developing anxious behavior problems at school age. These significant indirect paths supported passive gene and environment correlation in explaining how genes function in the developmental processes of children's internalizing behavior problems.

In terms of active rGE, when I tested specific indirect paths from children's genes to withdrawn or anxious behavior problems via child temperament, the indirect paths from children's genes to anxious behavior problems mediated by child temperament were

significant (β = -.005, $p < .05$ for the path from children's 5-HTTLPR, β = .006, $p < .05$ for the path from children's TPH2 rs4570625). Since children who had one or more short alleles on 5-HTTLPR were less likely to show difficult temperament in infancy, the indirect path from children's 5-HTTLPR to their later anxious behavior problems had a negative coefficient. However, the indirect paths from children's 5-HTTLPR and TPH2 to their withdrawn behavior problems via child temperament were not significant (β = -.003, $p = .076$ for the path from children's 5-HTTLPR, β = .004, $p = .065$ for the path from children's TPH2 rs4570625). Therefore, active gene and environment correlation was supported in part, but only in relation to children's anxious behavior problems.

Interestingly, when I tested indirect paths based on evocative rGE, all separate paths from child genes to child temperament, from child temperament to mothers' psychological aggression, and from mothers' psychological aggression to children's withdrawn or anxious behavior problems were significant, but the sequential mediation paths via child temperament and mothers' psychological aggression from child genes to withdrawn or anxious behavior problems were not significant. To be specific, the indirect path from children's 5-HTTLPR to withdrawn behavior problems mediated by child temperament and mothers' psychological aggression was not significant (β = -.001, $p = .114$), and the indirect path from child TPH2 to withdrawn behavior problems mediated by child temperament and mothers' psychological aggression was not significant (β = .001, $p = .079$). Also, the indirect path from children's 5-HTTLPR to anxious behavior problems through child temperament and mothers' psychological aggression (β = -.001, $p = .111$) as well as the indirect path from children's TPH2 to anxious behavior problems

mediated by child temperament and mothers' psychological aggression (β = .001, *p* = .078) were non-significant. However, if I tested the indirect paths from child temperament (not including children's genes) to withdrawn or anxious behavior problems through mothers' psychological aggression, then those mediated paths were all significant. The indirect effect from child temperament to withdrawn behavior problems mediated by mothers' psychological aggression was significant (β = .009, *p* = < .05) and the indirect effect by mothers' psychological aggression from child temperament to anxious behavior problems was significant (β = .009, *p* < .05).

Discussion

There is growing interest in understanding the roles of children's genetic sensitivity in their social-emotional behavioral outcomes, but there have been fewer studies investigating gene and environment correlations. This study aimed to test three kinds of gene and environment correlations in addition to gene and environment interactions to understand how mothers' and children's genes are associated with environmental factors (mothers' negative parenting behaviors) to predict children's later internalizing behavior problems.

The results of this study showed that not only 5-HTTLPR but also TPH2 rs4570625 might play important roles in explaining how genes are associated with the development of children's internalizing behavior problems. In specific, when I tested indirect paths from mothers' TPH2 to children's withdrawn/anxious behavior problems via mothers' psychological aggression, these mediations of mothers' psychological aggression were significant, which supported passive gene and environment correlation.

5-HTTLPR appeared more related to anxious behavior problems rather than withdrawn behavior problems. There were two significant mediation paths found, and among them, the mediation of children's 5-HTTLPR on the path from mothers' 5-HTTLPR to anxious behavior problems supported passive gene and environment correlation by showing that mothers tended to pass on sensitive genes to children.

The existence of active gene and environment correlation in the developmental processes underlying children's internalizing problems was partially supported. For example, among several indirect paths through child temperament from children's genes to internalizing behavior problems, only the mediation by child temperament from children's 5-HTTLPR to anxious behavior problems was significant. Interestingly, children who had two long alleles were more likely to show difficult temperament, which contradicts the results of Auerbach et al. (1999). In Auerbach et al. (1999), children who had two short alleles showed the highest scores in distress to limitations and negative emotionality than other children with the S/L or L/L genotypes. More research is needed to replicate the nature of the associations between children's 5-HTTLPR and child temperament.

With respect to evocative gene and environment correlation, there was a tendency for children who showed difficult temperament in infancy to receive greater psychological aggression in early childhood from mothers, and these children were in turn more likely to develop internalizing behavior problems. However, the indirect paths from children's genes to internalizing behavior problems through child temperament and mothers' psychological aggression were not statistically significant. That might be

because the paths from children's genes to child temperament were too weak to detect statistical significance. Another possibility is that mothers might have provided care and supports regardless of the levels of child temperament.

Interestingly, none of the gene and environment (mothers' negative parenting behaviors) interaction terms was significant. Except children's TPH2 predicting their later withdrawn behavior problems, neither children's 5-HTTLPR and temperament nor mothers' psychological aggression were significant in this model to predict the development of child internalizing behavior problems. It infers the possibility that child sensitivity toward environments and mothers' psychological aggression are not related to each other at the same time but related in a sequential way. The effects of mothers' parenting were not different by child genetic characteristics and the effects of child individual characteristics were not different by mothers' parenting behaviors. More research need to be shown to make sure whether this result appears only in this specific dataset or it can be applied to other cases.

Of course, when we interpret the results of this study, we should be cautious because of a number of limitations, including little previous research about gene and environment correlations to serve as a guide, small effect sizes of genetic variables, the possibility of missing significant measurements about mothers' parenting or experimental measurement of child temperament, and the possibility of missing covariates. Although we need to consider all the limitations and weaknesses of this study, as an investigation of gene and environment interplay, however, it also had some key strengths such as a large sample size, data from a low-income multi-racial population, longitudinal data from

child birth to age 9, and the inclusion and testing of three gene and environment correlations to explain the role of genes in predicting children's social-emotional developmental outcomes.

For future research, I can suggest several ideas. First, it would be recommended to conduct more research in the near future to examine which findings are replicated and which findings are not by analyzing similar and different samples. Given that the FFCWS oversampled nonmarital births and low-income population, the sample of this study is good to use for examining the patterns among low-income couples but it may not be appropriate to see the patterns in middle or upper class. So, future research can try to investigate gene and environment correlations using a sample representing the general population in U.S. Second, future research can include more integrated measurements about parenting and child temperament. For example, future research might consider the inclusion of observational measurements or qualitative interview to reflect more diverse aspects of mothers' parenting and child temperament. Also, I did not include fathers' parenting because of nonsignificance of the measurements. However, future research might add more integrated measurements for fathers' parenting and draw interesting findings in relation to fathers' parenting. By accumulating the results, I anticipate we can reveal the mechanism of children's internalizing behavior problems and hope those attempts can help to identify children at high risk and to develop more efficient intervention programs to support children's healthy social-emotional development without harming oneself.

Table 6. Demographic characteristics of the sample (N = 2,646)

	Mean (Std.) N (%)	Min.	Max.
Mothers' age at birth	25.03 (5.92)	15	43
Fathers' age at birth	27.58 (7.05)	15	53
Mothers' education at birth	2,644 (100.0)		
Less than high school	869 (32.87)		
High school	830 (31.39)		
2-3yr college	667 (25.23)		
4yr college or higher	278 (10.51)		
Fathers' education at birth	2,561 (100.0)		
Less than high school	833 (32.53)		
High school	952 (37.17)		
2-3yr college	526 (20.54)		
4yr college or higher	250 (9.76)		
Marital status at childbirth	2,645 (100.0)		
Married	635 (24.01)		
Cohabiting	970 (36.67)		
Nonresident	1,040 (39.32)		
Marital status at child age 9	2,623 (100.0)		
Married	792 (30.19)		
Cohabiting	267 (10.18)		
Nonresident	1,564 (59.63)		
Household income at birth	32720.44 (31801.61)	0	133750
Household income at child age 9	45321.30 (50044.67)	0	900000
Mothers' race/ethnicities	2,639 (100.0)		
European-American	554 (20.99)		
African-American	1,260 (47.75)		
Hispanic	742 (28.12)		
Other/mixed	83 (3.15)		
Fathers' race/ethnicities	2,632 (100.0)		
European-American	485 (18.43)		
African-American	1,326 (50.38)		
Hispanic	726 (27.58)		
Other/mixed	95 (3.61)		
Child gender	2,646 (100.0)		
Boy	1,284 (48.53)		
Girl	1,362 (51.47)		

Table 7. Correlation matrix, means, and standard deviations

	1	2	3	4	5	6	7	8	9	10	11	12	13	Mean (SD)
1	1.00													.16 (.22)
2	.673	1.00												.19 (.22)
3	.005	.054	1.00											.59 (.49)
4	.040	.022	.027	1.00										.54 (.50)
5	.011	.065	.446	-.043	1.00									.58 (.49)
6	-.006	.013	.033	.398	-.028	1.00								.54 (.50)
7	.117	.101	-.050	.046	-.013	-.063	1.00							1.75 (1.00)
8	-.002	.012	.024	.055	-.006	.083	.054	1.00						.51 (.50)
9	.076	.077	-.053	.069	.016	.033	.084	.006	1.00					2.82 (1.06)
10	.035	.024	-.068	.042	-.079	.029	.061	.028	.059	1.00				1.10 (.88)
11	-.121	-.070	.057	-.001	.054	-.043	-.025	-.036	-.084	-.195	1.00			1.06 (1.41)
12	.108	.048	-.010	.021	-.063	.057	.038	.027	.067	.136	-.589	1.00		7.00 (3.08)
13	.010	-.045	-.047	.002	-.052	.034	.066	.027	.043	.020	-.212	.260	1.00	2.76 (1.31)

Note. 1 = child withdrawn behavior problems at age 9, 2 = child anxious behavior problems at age 9, 3 = child 5-HTTLPR, 4 = child TPH2 rs4570625, 5 = mother's 5-HTTLPR, 6 = mother's TPH2 rs4570625, 7 = mother's psychological aggression at child age 5, 8 = child gender, 9 = child temperament at age 1, 10 = the frequencies of family structure change from child birth to child age 9, 11 = minimum poverty ratio by age 9, 12 = family poverty duration by age 9, 13 = the number of children at child age 9.

Table 8. Maximum likelihood estimates and selected fit indices for passive gene and environment correlation

	Passive rGE		
	b	SE	B
Child 5-HTTLPR			
← Mother's 5-HTTLPR	.460	.021***	.461
Child TPH2 rs4570625			
← Mother's TPH2 rs4570625	.396	.021***	.394
Mother's psychological aggression			
← Mother's 5-HTTLPR	-.021	.053	-.010
← Mother's TPH2 rs4570625	-.136	.054*	-.067
← Number of children (< 18yrs old) in the household at age 5	.043	.020*	.058
← Frequencies in family structure change by age 5	.071	.033*	.055
← Minimum poverty ratio by age 5	.050	.062	.025
← Poverty duration by age 5	.008	.018	.013
Child withdrawn behavior problems			
← Mother's psychological aggression	.024	.007***	.105
← Child 5-HTTLPR	.008	.011	.017
← Child THP2 rs4570625	.008	.011	.019
← Child gender (1 = boy, 0 = girl)	.003	.011	.006
← Child temperament	.013	.005*	.060
← Number of children (< 18yrs old) in the household at age 9	-.005	.004	-.029
← Frequencies in family structure change by age 9	.000	.007	-.002
← Minimum poverty ratio by age 9	-.024	.013	-.051
← Poverty duration by age 9	.005	.002*	.061
Child anxious behavior problems			
← Mother's psychological aggression	.022	.006***	.097
← Child 5-HTTLPR	.024	.011*	.054
← Child THP2 rs4570625	-.003	.011	-.006
← Child gender (1 = boy, 0 = girl)	.009	.011	.020
← Child temperament	.020	.005***	.095
← Number of children (< 18yrs old) in the household at age 9	-.011	.004**	-.066
← Frequencies in family structure change by age 9	-.004	.006	-.014
← Minimum poverty ratio by age 9	-.015	.013	-.034
← Poverty duration by age 9	.000	.002	.003
Child withdrawn ↔ anxious behavior problems	.033	.004***	.665
Model fit indices			
χ^2 (*df*)	83.767 (43)***		
RMSEA [90% CI]	.023 [.015, .030]		
CFI	.978		
SRMR	.015		

***$p < .001$, **$p < .01$, *$p < .05$.

Table 9. Maximum likelihood estimates and selected fit indices for active gene and environment correlation

	Active rGE		
	b	SE	B
Child temperament			
← Child's 5-HTTLPR	-.106	.056	-.049
← Child's TPH2 rs4570625	.143	.056*	.066
← Child gender (1 = boy, 0 = girl)	.024	.056	.011
Child withdrawn behavior problems			
← Child 5-HTTLPR	.007	.012	.016
← Child THP2 rs4570625	.011	.012	.024
← Child temperament	.011	.006*	.052
← Child gender (1 = boy, 0 = girl)	-.004	.012	-.009
← Mother's psychological aggression	.026	.007***	.112
← Number of children (< 18yrs old) in the household at age 9	-.006	.004	-.034
← Frequencies in family structure change by age 9	-.002	.007	-.009
← Minimum poverty ratio by age 9	-.038	.014**	-.082
← Poverty duration by age 9	.004	.002*	.057
Child anxious behavior problems			
← Child 5-HTTLPR	.028	.012*	.061
← Child THP2 rs4570625	.001	.012	.001
← Child temperament	.014	.005*	.064
← Child gender (1 = boy, 0 = girl)	.001	.012	.003
← Mother's psychological aggression	.025	.006***	.108
← Number of children (< 18yrs old) in the household at age 9	-.015	.004***	-.090
← Frequencies in family structure change by age 9	-.003	.007	-.013
← Minimum poverty ratio by age 9	-.033	.015*	-.072
← Poverty duration by age 9	.002	.002	.029
Child withdrawn ↔ anxious behavior problems	.035	.005***	.675
Model fit indices			
χ^2 (*df*)	19.583 (5) **		
RMSEA [90% CI]	.043 [.024, .064]		
CFI	.985		
SRMR	.016		

***$p < .001$, **$p < .01$, *$p < .05$.

Table 10. Maximum likelihood estimates and selected fit indices for evocative gene and environment correlation

	Evocative rGE		
	b	SE	B
Child temperament			
← Child's 5-HTTLPR	-.105	.049*	-.048
← Child's TPH2 rs4570625	.132	.048**	.061
Mother's psychological aggression			
← Child temperament	.073	.024**	.078
← Child gender (1 = boy, 0 = girl)	.132	.051*	.066
Child withdrawn behavior problems			
← Mother's psychological aggression	.025	.006***	.110
← Child temperament	.012	.005*	.055
← Child gender (1 = boy, 0 = girl)	.002	.010	.004
← Number of children (< 18yrs old) in the household at age 9	-.007	.004	-.040
← Frequencies in family structure change by age 9	-.003	.006	-.012
← Minimum poverty ratio by age 9	-.026	.011*	-.056
← Poverty duration by age 9	.005	.002**	.073
Child anxious behavior problems			
← Mother's psychological aggression	.023	.006***	.103
← Child temperament	.016	.005**	.078
← Child gender (1 = boy, 0 = girl)	.006	.010	.014
← Number of children (< 18yrs old) in the household at age 9	-.013	.004***	-.080
← Frequencies in family structure change by age 9	-.009	.006	-.032
← Minimum poverty ratio by age 9	-.026	.012*	-.057
← Poverty duration by age 9	.001	.002	.019
Child withdrawn ↔ anxious behavior problems	.034	.004***	.663
Model fit indices			
χ^2 (*df*)	42.617 (15)***		
RMSEA [90% CI]	.030 [.019, .040]		
CFI	.978		
SRMR	.020		

***$p < .001$, **$p < .01$, *$p < .05$.

Table 11. Maximum likelihood estimates for gene and environment interactions

	G x E		
	b	SE	B
Child withdrawn behavior problems			
← Child 5-HTTLPR	-.006	.024	-.012
← Child THP2 rs4570625	.048	.024*	.104
← Mother's psychological aggression	.019	.019	.083
← Child 5-HTTLPR x Mother's psychological aggression	.006	.012	.031
← Child TPH2 x Mother's psychological aggression	-.017	.012	-.089
← Child temperament	.005	.011	.021
← Child Temperament x Mother's psychological aggression	.004	.005	.062
← Child gender (1 = boy, 0 = girl)	-.004	.012	-.009
← Number of children (< 18yrs old) in the household at age 9	-.005	.004	-.032
← Frequencies in family structure change by age 9	-.003	.007	-.012
← Minimum poverty ratio by age 9	-.035	.015*	-.077
← Poverty duration by age 9	.005	.002*	.065
Child anxious behavior problems			
← Child 5-HTTLPR	.019	.024	.042
← Child THP2 rs4570625	.041	.024	.090
← Mother's psychological aggression	.036	.019	.158
← Child 5-HTTLPR x Mother's psychological aggression	.003	.012	.016
← Child TPH2 x Mother's psychological aggression	-.019	.012	-.099
← Child temperament	.016	.011	.079
← Child Temperament x Mother's psychological aggression	-.002	.005	-.028
← Child gender (1 = boy, 0 = girl)	.001	.012	.003
← Number of children (< 18yrs old) in the household at age 9	-.013	.004**	-.078
← Frequencies in family structure change by age 9	-.003	.007	-.011
← Minimum poverty ratio by age 9	-.026	.015	-.057
← Poverty duration by age 9	.002	.002	.032
Child withdrawn ↔ anxious behavior problems	.034	.002***	.015
Model fit indices			
RMSEA		.000	
CFI		1.000	
SRMR		.000	

***$p < .001$, **$p < .01$, *$p < .05$.

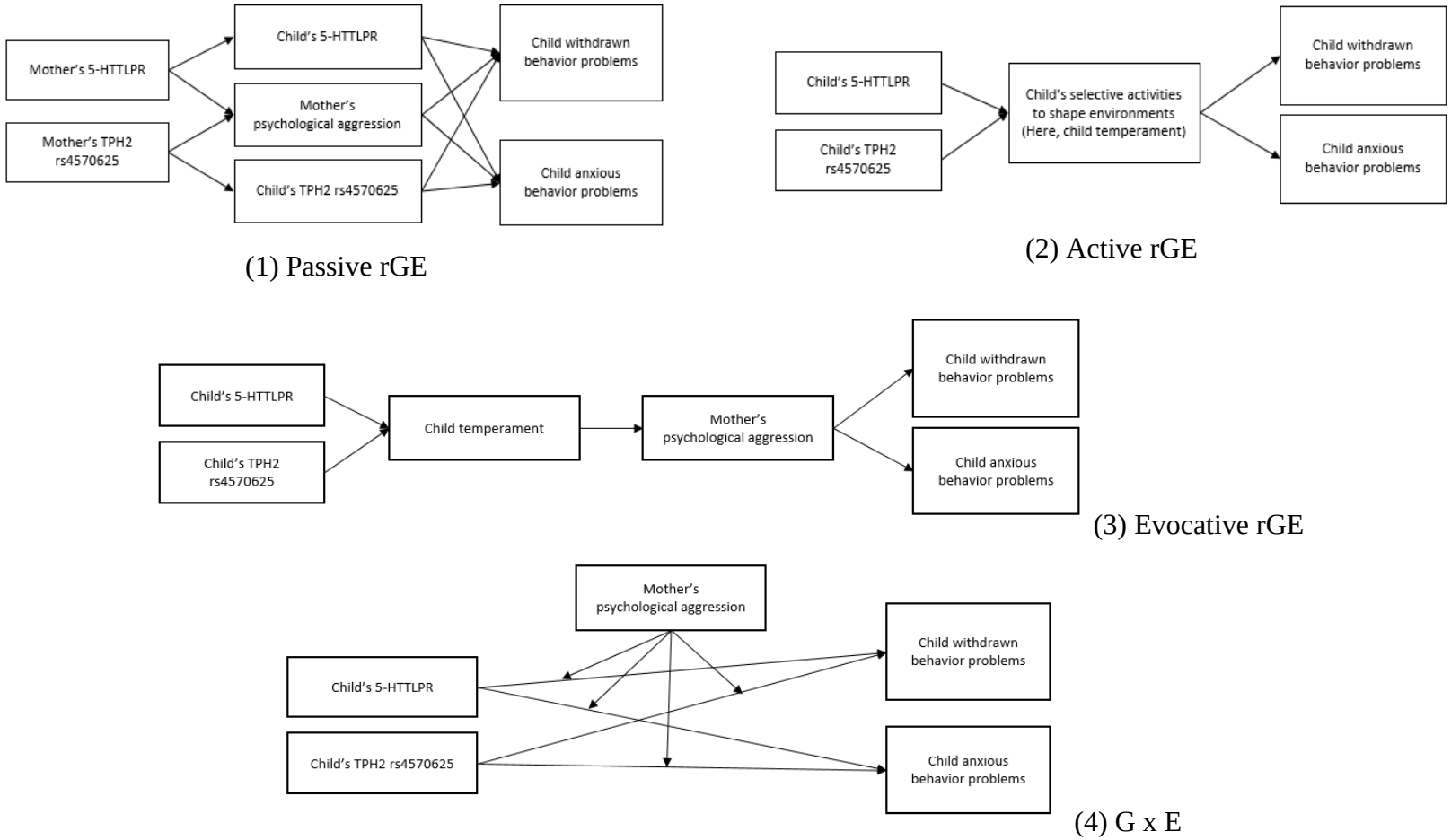


Figure 4. Research models

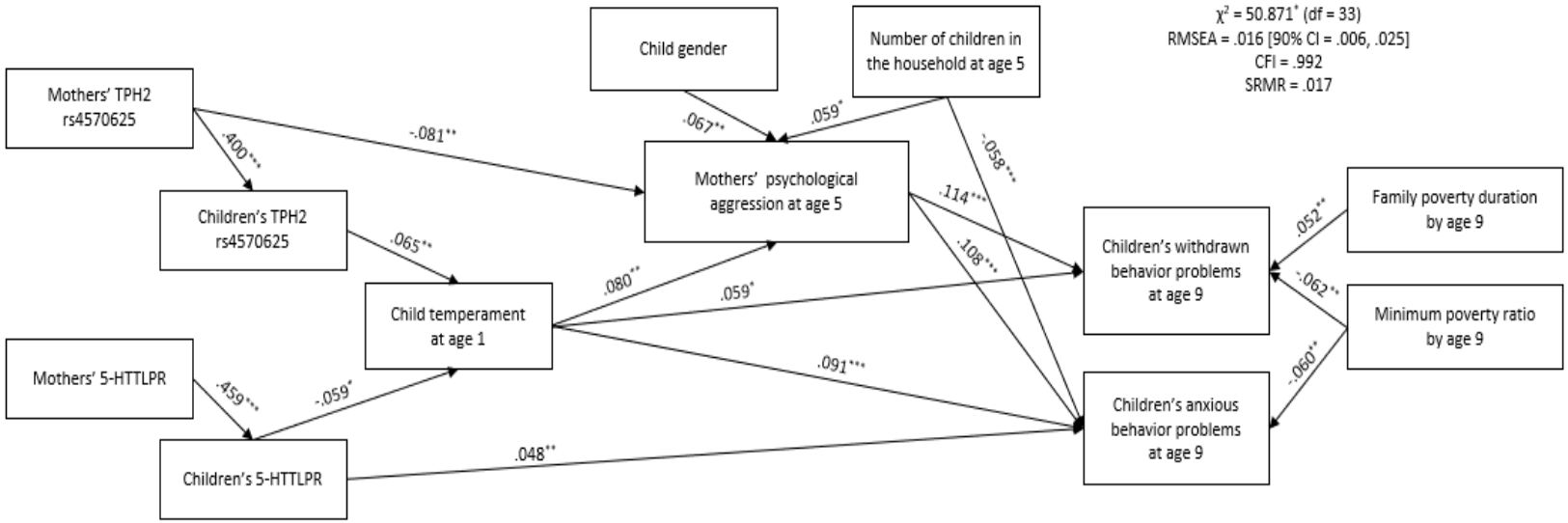


Figure 5. Finalized model

Chapter 4: Mother's genetic sensitivity, father's coparenting, and mother's aggression to children in the context of fathers' marital and residential status

Low-income mothers are more likely to use harsh parenting strategies and mothers' negative parenting can lead their children to have difficulties to adjust at school (Shumow, Vandell, & Posner, 1998). Children who receive physical aggression from parents are also likely to experience psychological aggression and those who receive psychologically aggressive parenting tend to show more negative developmental outcomes (i.e., mental health problems) than other children (Claussen & Crittenden, 1991). In his classic model of the determinants of parenting, Belsky (1984) described mothers' individual psychological resources, child characteristics, and family contexts such as stress or support as significant factors contributing to mothers' parenting behaviors. Building upon this model, diathesis model or vulnerability-stress model (Ingram & Luxton, 2005) can be applied to explain how stressful environments and mothers' genetic sensitivity together increase the risk for mothers' negative parenting behaviors. Another perspective on gene and environment interaction – the differential susceptibility hypothesis (Belsky & Pluess, 2009) – focuses attention on the "bright side" of having greater genetic sensitivity especially in advantageous circumstances. Based on these theories, it is possible to assume that support from fathers can function as an advantageous environment for mothers that may reduce the risk for mothers' negative

parenting behaviors, especially for genetically sensitive mothers. This study focused on testing this assumption.

Risk Factors for Mothers' Negative Parenting

Child temperament. Temperament can be defined as "individuals' deeply rooted predispositions toward emotional reactivity and self-regulation" (Bates & Pettit, 2015, p. 372). Bates and Pettit's review (2015) also suggested that temperament consists of three domains: positive emotionality, negative emotionality, and self-regulation. It is well known that mothers' parenting influences child temperament, but the transactional model also revealed that child temperament can elicit mothers' negative parenting behaviors (Kiff, Lengua, & Zalewski, 2011). The transactional model focuses on constant developmental changes through interaction between children and parents, individuals and environments, or individual environtype, phenotype, and genotype (Sameroff, 2009). Given that the transactional model highlights reciprocal influences and regulation after complex dynamic interactions, it is often used to describe the association between children's individual characteristics and mothers' parenting behaviors (Sameroff, 2009).

A number of studies have demonstrated associations between child temperament and mother's negative parenting behavior, consistent with the transactional model. Clark, Kochanska, and Ready (2000) collected data from mother-report surveys and observations of infants' temperament at 8-10 months postpartum and mothers' parenting behavior at 13-15 months postpartum. The results showed that children's difficult temperament predicted an increase in mothers' power assertion. Also, mothers' personality traits were significantly associated with mothers' later power assertion. After

including the interaction between child temperament and mothers' personality traits, mothers with higher extraversion or low perspective taking personality traits were more likely to provide power assertive parenting behaviors if they raised children with more difficult temperaments (Clark et al., 2000). One explanation might be that mothers who raise children with more difficult temperaments might have a greater need to control their children's behaviors (Kiff et al., 2011). Similarly, Bridgett et al. (2009) showed that an increase in children's negative emotionality from 4 to 12 months of age and a decrease in children's regulatory capacity from 4 to 12 months of age significantly increased the risk of mothers' negative parenting behaviors at 18 months old.

In contrast, the influence of child temperament on mothers' positive parenting behaviors is less clear. For example, in Clark et al. (2000), child temperament was a significant predictor and moderator for mothers' power assertion, but not for mothers' sensitive parenting. That might be because mothers have more responsibility to take care of infants so they would not reduce their engagement with their infants regardless of infants' temperament. McBride, Schoppe, and Rane (2002) showed gender differences between mothers and fathers in parental involvement. Child temperament increased mothers' parenting stress, but mothers' involvement was less likely to be affected by child temperament than fathers' involvement.

Family socioeconomic resources. Another risk factor for mothers' negative parenting is having low socioeconomic resources. The family process model (also called the family stress model) explains that family socioeconomic hardship increases parents' psychological distress, and therefore lessens the quality of parenting (Barnett, 2008;

Conger et al., 2010; McLoyd, 1990; Vernon-Feagans & Cox, 2013). Low-income mothers have fewer resources to cope with challenging situations, so the quality of their parenting is more likely to be compromised as the severity of family poverty increases. Family poverty amplifies family exposure to life stressors and increases mothers' negative parenting behaviors, which in turn worsens children's cognitive and social-emotional functioning (Conger et al., 2010; McLoyd, 1998; Shala & Grajcevci, 2016; Vernon-Feagans & Cox, 2013). Focused on cultural differences by mothers' race/ethnicities, Middlemiss (2003) examined whether parenting stress and parenting behaviors in the context of family poverty differed by mothers' race/ethnicities. The results showed that there were some differences but they were not at the significant level. Rather, the effects of family poverty were greater than the differences by race/ethnicities.

Parenting stress. Mothers' parenting stress is also an important risk factor for negative parenting behavior. Greater parenting stress significantly predicts mothers' aggressive parenting behaviors and this path can be stronger in low-income families (McLoyd et al., 1994; Patterson, 1986). Other research has shown that mothers' stress mediates the influence of family SES on parental perception of children and parental cognitive emotional processes (Pinderhughes, Dodge, Bates, Pettit, & Zelli, 2000). Greater parenting stress also increases the risk of physical maltreatment toward children (Chan, 1994). In this study, I assume mothers' parenting stress can be a risk factor to increase mothter's psychological and physical aggressive behaviors in parenting.

Family instability. Mothers who were not married at childbirth are more likely to experience family instability within 5 years and each transition in relationship increases

mothers' parenting stress and the risk of mothers' harsh parenting toward children (Beck, Cooper, McLanahan, & Brooks-Gunn, 2010). McLanahan and Beck (2010) analyzed interparental relationships in the Fragile Families and Child Wellbeing study sample and ascribed the reasons for high family instability among them to low financial resources, marriage barriers in policies, multiple partner fertility, gender distrust, and poor psychological resources between parents. Experiencing family instability increases hardship in parenting such as poor behavioral control, less warmth/acceptance, and greater psychological control. Children who experience family instability are less likely to have an expectation of their family as a safe haven. The negative effect of family instability on children's appraisal of family security is also mediated by the increase of parenting hardship. So, those children are more likely to show social-emotional behavior problems (Forman & Davies, 2003).

Mothers' Genetic Sensitivity and Parenting Behaviors

Serotonin transporter genes like 5-HTTLPR affect emotional reactivity in stressful situations (Weeland et al., 2015b). Just as children's genetic sensitivity affects children's social-emotional and behavioral outcomes, mothers' genetic sensitivity can play an important role in mothers' psychological functioning and parenting behaviors toward children. Bakermans-Kranenburg and van IJzendoorn (2008) showed that mothers who had two short alleles on 5-HTTLPR and AA/AG alleles on OXTR were less likely to provide sensitive parenting their to infant children at 2 years old. Also, Mileva-Seitz et al. (2011) found that mothers with a short allele on 5-HTTLPR tended to show more sensitive parenting toward their infants at six month postpartum. Cents et al. (2014) also

supported Mileva-Seitz et al. (2011)'s findings that mothers who had one or more short allele(s) were more likely to provide sensitive parenting.

Fathers' Coparenting and Childcare as Protective Factors

Given that raising young children requires a lot of time and energy, fathers' support in childcare and supportive attitudes toward mothers can ease mothers' parenting stress and therefore reduce the risk of mothers engaging in negative parenting behaviors. There are not many studies directly delving into how the amount of father involvement influences the quality of mothers' parenting but there is some previous research reporting that father involvement buffers the negative effects of depressed mothers' parenting on child development (i.e., Mezulis, Hyde, & Clark, 2004). In addition to fathers' support in childcare, which is the most commonly used conceptualization and measurement representing the quantity of father involvement in childrearing, researchers should also consider fathers' coparenting as a measure of the quality of father involvement, because the concept of coparenting includes fathers' respect toward mothers' parenting perspectives and values, which is also an important form of support.

Coparenting can be defined as "the ways that parents and/or parental figures relate to each other in the role of parent (Feinberg, 2003, p.96)". Conceptually, coparenting consists of four major sub-constructs: support/undermining, childrearing agreement, joint family management, and division of labor, and the relations between coparenting and its sub-constructs should be considered as an ensemble (Feinberg, 2003). However, among them, the most frequently investigated aspect is supportive coparenting. Supportive coparenting represents the amount of warmth and cooperation partners provide each other

in their roles as parents (Schoppe-Sullivan, Settle, Lee, & Kamp Dush, 2016). Greater supportive coparenting predicted lower child externalizing behavior problems (Schoppe, Frosch, & Mangelsdorf, 2001), and may be especially helpful for preventing the development of externalizing behavior problems for children with low temperamental effortful control (Schoppe-Sullivan, Weldon, Cook, Davis, & Buckley, 2009). In a meta-analysis, Teubert and Pinquart (2010) analyzed 59 studies about coparenting and child adjustment, and showed that coparenting was significantly and positively associated with children's psychological adjustment. The effect sizes were small but statistically significant even after controlling marital quality and parenting behaviors of individual parents.

Interestingly, compared to the amount of research examining associations between coparenting and children's social-emotional development, relatively few studies have investigated how fathers' supportive coparenting is associated with mothers' parenting behaviors. In Schoppe-Sullivan et al. (2016), fathers' perceptions of supportive coparenting they received from mothers mediated the paths from their perceptions of couple relationship functioning or from their attachment anxiety to parenting stress or to parenting satisfaction. However, mothers' perceptions of supportive coparenting (from fathers) were not significantly associated with mothers' parenting stress or parenting satisfaction in infancy after their first child's birth. That might be because mothers have the main responsibility of taking care of an infant given their unique physical functions (e.g., breastfeeding) during that period, so mothers' variations in parenting stress or

parenting satisfaction that could be explained by fathers' supportive coparenting were not significantly large.

Previous research about fathers' roles as supportive coparents for mothers has focused on the transition to parenthood, so it is less well known how fathers' supportive coparenting predicts mothers' parenting behaviors for mothers raising older children from early to middle childhood. Whereas in infancy fathers are less accessible to children than mothers, and even when both parents are accessible to the child, fathers participate less in basic care but spend similar amounts of time in play or outings (Laflamme, Pomerleau, & Malcuit, 2002). However, fathers' involvement with children increases in toddlerhood and early childhood (Lamb, 2010). Thus, fathers' supportive coparenting when children are older might significantly reduce mothers' parenting stress and contribute to mothers' more positive and less negative parenting behaviors toward their children.

Different Fathers' Participation in Diverse Family Contexts

Harris and Ryan (2004) highlight that family context matters in determining fathers' participation in childcare. They categorized two types of parental strategies as (1) cooperative strategy and (2) compensatory strategy. Which strategy a mother uses influences the amount of fathers' participation, especially for nonresident fathers. They anticipated that two resident biological, adoptive, or surrogate parents are likely to show higher father involvement with both parents adopting cooperative strategies, but anticipated more variation in father involvement and coparenting among nonresident-father families. If a mother has cooperative strategy, then a nonresident father would have more chance to participate in childcare and to show supportive coparenting. Or, if a

mother is less engaged in childcare, then a father might be more likely to participate in childcare to compensate for the lack of mother's investment. In this case, a father might have a lower supportive coparenting attitude toward a mother, but his amount of childcare provision might be higher than the average. However, if a mother has compensatory strategy and is highly involved in childcare, then a nonresident father would have fewer opportunities to take care of their child. This would reduce the chance to develop supportive coparenting because the mother has already developed controlling behaviors toward the father's involvement. According to this theory, the level of fathers' support differs by family context. Based on this, I tested whether mothers' parenting behavior is differentially influenced by fathers' support depending on the family context. Considering the fact that in the FFCWS the couple's marital status at childbirth influences family instability within 5 years (Beck et al., 2010), I considered fathers' residential status as well as marital status to assess family structure in the current study (i.e., married, cohabiting, and nonresident parents).

The Present Study

In this study, I focused on mothers' aggressive behaviors in middle childhood (at age nine). Given that a majority of children showed constantly low or decreasing patterns in aggressive behaviors from age four to nine (Bongers, Koot, van der Ende, & Verhulst, 2004; Campbell, Spieker, Burchinal, Poe, & NICHD ECCRN, 2006), mothers' aggressive behaviors in early childhood (at age five) might be overestimated because of children's behavioral characteristics at that specific developmental stage (i.e., children's impulsive behaviors before attaining self-regulation skills). Fathers' supportive

coparenting as well as childcare provision measured at age three were used as the indicators of fathers' initial support levels. Because during infancy fathers' involvement in childrearing tends to be significantly lower than that of mothers (Yavorsky, Kamp Dush, & Schoppe-Sullivan, 2015), but increases by the preschool years, I selected child age three as more appropriate to measure the amount of fathers' participation and supportive coparenting. Mothers' genes were collected at children's age nine, but their genotypes were assumed to be consistent from mothers' births. Child temperament was measured at age one to indicate children's non-shared inborn characteristics that may elicit mothers' parenting behaviors. In terms of family instability or family poverty, those variables were constructed by the information collected by age five because by that age enough time had passed to most accurately capture levels of family risk.

Considering previous research showing that the effects of child temperament on mothers' positive parenting behaviors were mixed, but those on mothers' negative parenting behaviors were relatively clear, this study test the effects of all the predictors on mothers' negative parenting behaviors. In particular, this study focused on two aspects of mothers' negative parenting behaviors toward school aged children - mothers' psychological and physical aggression – and tested associations between all the predictors (mothers' genetic sensitivity, child temperament, mothers' parenting stress, fathers' supportive coparenting and childcare provision, family poverty), the interactions between mothers' genetic sensitivity and fathers' coparenting/parenting support, and mothers' later psychological and physical aggression toward school aged children. Among them, mothers' psychological and physical aggressive behaviors, fathers'

supportive coparenting, fathers' childcare provision, and mothers' 5-HTTLPR are key variables. Other factors like child temperament, mothers' parenting stress, and family poverty-related variables were covariates that might be associated with mothers' aggressive parenting behaviors, according to the literature review.

Previous research has revealed some evidence that mothers' genetic sensitivity might affect mothers' parenting behaviors. However, there have been insufficient efforts to test interactions between mothers' genetic sensitivity and environmental factors such as fathers' support for mothers' parenting. In terms of fathers' supportive coparenting, there are many studies examining the association between fathers' coparenting and children's developmental outcomes but there has been little research to test the role of fathers' coparenting on mothers' parenting behaviors. This study is a novel attempt to incorporate fathers' supportive coparenting and family environment when examining the association between mothers' genetic sensitivity and mothers' parenting behaviors. By testing whether and how mothers' genetic sensitivity is associated with mothers' parenting behaviors and whether and how fathers' coparenting interacts with mothers' genetic sensitivity to predict mothers' parenting behaviors, this study aims to reveal aspects of the detailed mechanisms underlying mothers' parenting.

Research Questions and Hypotheses

1. How are mothers' 5-HTTLPR, child temperament in infancy, mothers' early parenting stress, fathers' early supportive coparenting, fathers' childcare provision, the severity of family poverty, and family instability associated with mothers' later psychological and physical aggression toward children? Do the effects of fathers'

supportive coparenting and childcare provision on mothers' negative parenting behaviors differ by mothers' 5-HTTLPR?

2. Are there differences in the effects of fathers' supportive coparenting and childcare provision on mothers' later negative parenting behaviors by fathers' marital and residential status?

My hypotheses were that (1) having minor alleles on 5-HTTLPR, raising a focal child who showed more difficult temperament, experiencing higher parenting stress, experiencing more severe family poverty, and having frequent changes in fathers' residential status would increase the risk of mothers' negative parenting. But, I anticipated that (2) greater fathers' supportive coparenting or childcare provision would decrease the risk of mothers' negative parenting. And I also anticipated that (3) the protective effects of fathers' supportive coparenting and childcare provision on mothers' negative parenting would be greater for mothers with minor alleles on 5-HTTLPR. Although the levels of fathers' support might be different by family context, I anticipated that (3) the benefits from fathers' supportive coparenting and childcare provision would appear consistently across the diverse family contexts.

Method

Data and Analytic Sample

This study analyzed data from 1,571 couples who participated in the survey, home visitation, and saliva sample collection at birth, child age 1, 3, 5, and 9 as part of the Fragile Families and Child Wellbeing study (FFCWS). This FFCWS recruited the families at hospitals across the U.S. at the time of the focal child's birth. They

oversampled couples who experienced childbirth while in a nonmarital relationship. When the focal children were one, three, five, and nine years old, their biological mothers and fathers were followed by core phone interviews, and children's primary caregivers were interviewed at home visits. When the focal children were nine years old, interviewers asked biological mothers and children to provide saliva samples through the Oragene® DNA Self-Collection Kit. The collected saliva samples were mailed to Westat by interviewers and from Westat to the Molecular Biology lab at Princeton University. The samples were in an air incubator for at least two hours or in a water incubator for at least one hour. Then, the samples were moved to the microcentrifuge tube to incubate on ice for 10 minutes and centrifuged at room temperature at 4,000 rpm for 10 minutes. TE solution was added as 0.5 to 1.0 ml to rehydrate the DNAs and rehydrated DNAs were transferred to three microcentrifuge tubes for storage (FFCWS, 2015).

Measures

Mothers' psychological aggression. Based on the Parent Child Conflict Tactics Scales (Straus et al., 1998), a mean score of maternal psychological aggression was constructed using mothers' reports on five items: "(I had) shouted, yelled, or screamed at child", "threatened to spank or hit him or her but did not actually do it", "swore or cursed at him or her", "called him or her dumb or lazy or some other name like that" and "said I would send child away or kick child out of the house" ($\alpha = .70$). Each item was scored from 0 *never* to 6 *more than 20 times* during the previous year before child age nine. The original scale also included 7 *yes, but not in past year*, but, considering all the items asked the frequencies during one year, I recoded it to 0.

Mothers' physical aggression. Like mothers' psychological aggression, a mean score of mothers' physical aggression was constructed based on the Parent Child Conflict Tactics Scales (Straus et al., 1998). Mothers' reports on five items were used: "(I had) hit child on the bottom with something like a belt, hairbrush, a stick or some other hard object", "spanked child on bottom with bare hand", "slapped him or her on the hand, arm, or leg", "pinched child", and "shook child" ($\alpha = .70$). Each item was scored from 0 *never* to 6 *more than 20 times* in the previous year before child age nine. The original scale also included 7 *yes, but not in past year*, but, considering all the items asked the frequencies during one year, I recoded it to 0.

Mothers' 5-HTTLPR. If a mother had two short alleles on the serotonin transporter gene (5-HTTLPR), it was coded as 1. Mothers who did not have two minor alleles on 5-HTTLPR were coded as 0, indicating lower genetic sensitivity.

Fathers' supportive coparenting. Mothers reported fathers' supportive coparenting in the core parental interview at children's age three. A mean score was constructed by using six items: "When (father) is with (child), he acts like the father you want for your child", "You can trust (father) to take good care of (child)", "He respects the schedules and rules you make for (child)", "He supports you in the way you want to raise (child)", "You and (father) talk about problems that come up with raising (child)", and "You can count on (father) for help when you need someone to look after (child) for a few hours" ($\alpha = .90$). Each item was scored from *0 never true* to *3 always true*. In this study, fathers' supportive coparenting was used as an indicator of the quality of fathers' support for mothers' parenting.

Fathers' childcare provision. Fathers' childcare provision was assessed based on mothers' reports at children's age three. The original questionnaire included four items: "How often does he look after (child) when you need to do things", "How often does he run errands (for you) like picking things up from the store", "How often does he fix things around your home, paint, or help make it look nicer in other ways", and "How often does he take (child) places (he/she) needs to go, such as to daycare or the doctor". However, the item "How often does he fix things around your home, paint, or help make it look nicer in other ways" worsened the reliability so it was excluded. After excluding this item, a mean score was created using the three remaining items and the reliability was acceptable ($\alpha = .87$). The original scale of each item was ranged from *1 Often* to *4 Never*. However, for easy interpretation, I reverse coded each item from *0 Never* to *3 Often*. In this study, fathers' childcare provision was used as an indicator of the quantity of fathers' parenting support.

Covariates

Child temperament. Based on the subscale of emotionality of the Emotionality, Activity, and Sociability Temperament Survey for Children (Mathieson & Tambs, 1999), a mean score for children's difficult temperament was constructed by using three items: "(Child) often fusses and cries", "gets upset easily", and "reacts intensely when upset" ($\alpha = .60$). Each item was scored from *1 not at all* to *5 very much* by mothers at children's age one.

Mothers' parenting stress. A mean score of mothers' parenting stress was constructed by using 12 items: "You often have the feeling that you cannot handle things

very well", "You find yourself giving up more of your life to meet your child(ren)'s needs than you ever expected", "You feel trapped by your responsibilities as a parent", "Since having (child) you have been unable to do new and different things", "Since having (child) you feel that you are almost never able to do things that you like to do", "There are quite a few things that bother you about your life", "Having (child) has caused more problems than you expected in your relationship with men", "You feel alone and without friends", "When you go to a party, you usually expect to have a bad time", "You are less interested in people than you used to be", "You enjoy things less than you used to", and "You are unhappy with the last purchase of clothing you made for yourself" (α = .87). The original scale for each item ranged from *1 strongly agree* to *5 strongly disagree*. However, for easy interpretation, I recoded them to *1 strongly disagree* to *5 strongly agree* in order to make higher scores indicate higher stress from parenting.

Severity of family poverty. In this study, the severity of family poverty was measured by the lowest family poverty ratio from childbirth to children's age five. The FFCWS provided a constructed variable of family poverty ratio at each wave based on its definition by the U.S. Census Bureau. According to the U.S. Census Bureau, family poverty ratio was calculated by dividing household income by the threshold of poverty that was set differently by the number of family members and reflected annual inflation. Therefore, a lower family poverty ratio means more severe poverty and a higher family poverty ratio indicates greater family economic resources.

Family instability. Considering the differences between resident-father families and non-resident-father families, I created an indicator of family instability by summing

the frequencies in change of residential status with the focal child's biological father from childbirth to child age five. This variable indicates the number of changes in relationships and it was included as an independent predictor in the path model. Since the outcome variable of the research model in this study was mothers' negative parenting behaviors at child age nine, the changes in biological fathers' residential status were counted by the previous wave (children's age five). In addition to this, I also tested multigroup later by family context at age three (i.e., married vs. cohabiting vs. nonresident) when fathers' supportive coparenting and childcare provision were measured.

Analytic Plan

To test the association between mothers' 5-HTTLPR, fathers' supports, and mothers' negative parenting behaviors, I conducted path analysis in Mplus 8.0. Mothers' psychological and physical aggression were correlated. I tested path analysis with all the predictors and the interactions between mothers' 5-HTTLPR and fathers' coparenting or childcare provision to predict mothers' psychological and physical aggression. Then, I conducted multiple-group analysis by fathers' residential status with mothers and children. In the analysis, I used the maximum likelihood estimation with robust standard errors (MLR) option to produce robust results regardless of the normality of the data (Muthén & Muthén, 2007; Satorra & Bentler, 1994).

Results

Preliminary Results

Table 12 shows demographic characteristics of the sample used in this study. On average, mothers and fathers were in their twenties when the focal child was born. Their

education levels were relatively low; only 12.61% of mothers and 11.41% of fathers had a bachelor's degree. When the focal children were born, 40.83% (n = 641) of couples were cohabiting and 32.36% (n = 508) did not live together. Only 26.82% (n = 421) were married. Nine years later, the proportion of cohabiting couples decreased, but that of married or that of nonresident increased. The average of annual household income at focal child's birth was low(n = 1,571 mothers; *M* = $35,435.29, *SD* = 33,414.01). But, mothers' household income increased after nine years from childbirth. So, the average of annual household income of n = 1,510 mothers became $63,666.35 (SD = 64,129.70). This sample includes many African-American and Hispanic couples. Among 1,566 mothers, African-Americans were 47.70% (n = 747) of the sample, Hispanics were 25.03% (n = 392), European-Americans were 24.01% (n = 376), and others were 3.26% (n = 51). Fathers' race/ethnicities were similar to mothers'. African-Americans were 50.38% (n = 789), Hispanics were 25.10% (n = 393), European-Americans were 21.01% (n = 329), and others were 3.51% (n = 55). Lastly, in terms of focal child gender, boys were 50.99% (n = 801) and girls were 49.01% (n = 770) of the sample.

As shown in Table 13, mothers' psychological and physical aggression were correlated with each other. Greater psychological and physical aggression were also correlated with children's more difficult temperament, greater parenting stress, more frequent changes in family structure after childbirth, greater severity in family poverty, and fathers' lower supportive coparenting and lower childcare provision. Having two minor alleles on 5-HTTLPR was correlated with mothers' lower psychological aggression but was not significantly correlated with mothers' physical aggression. Child

difficult temperament was correlated with mothers' higher parenting stress, more severe family poverty, more frequent changes in family structure after childbirth, fathers' less supportive coaprenting, and fathers' less childcare provision. Fathers' less supportive coparenting and less childcare provision were correlated with more severe family poverty and more frequent changes in family structure. Fathers who provided greater supportive coparenting were also likely to provide more childcare.

Path Analyses

Table 14 shows the results of path analysis testing the associations between mothers' psychologically or physically aggressive parenting behaviors and mothers' genetic characteristics (having two minor alleles on 5-HTTLPR), child temperament, parenting stress, and fathers' supportive coparenting and childcare provision. In order to test whether the effects of fathers' supportive coparenting and childcare provision differed by mothers' genetic sensitivity, I added two interactions in the model. One was the interaction between mothers' having two minor alleles on 5-HTTLPR and fathers' supportive coparenting, and the other was the interaction between mothers' having two minor alleles on 5-HTTLPR and fathers' childcare provision.

As anticipated, if mothers experienced greater parenting stress at child age three, they were more likely to show psychological aggression toward their children at five years old (β = .12, p = .000). However, other predictors were not significantly associated with psychological aggression. The interaction between fathers' coparenting and mothers' 5-HTTLPR was not significantly related to mothers' psychological aggression (β = -.246, p = .099) but the interaction between fathers' childcare provision and mothers'

5-HTTLPR significantly predicted mothers' later psychological aggression. Interestingly, if mothers with two short alleles on 5-HTTLPR received greater childcare provision from the focal child's biological fathers, they were more likely to show psychological aggression ($\beta = .232$, $p = .005$).

In contrast to psychological aggression, many of the predictors were significantly associated with mothers' later physical aggression. Mothers who experienced greater parenting stress were more likely to show greater physical aggression subsequently ($\beta = .055$, $p = .036$). If a mother experienced more relationship changes with the focal child's father, then the risk of physical aggression toward the focal child was greater ($\beta = .065$, $p = .044$). However, if a mother had more family socioeconomic resources, the risk of physical aggression was lower ($\beta = -.067$, $p = .004$). For mothers' physical aggression, both interactions were significant but the directions were different. If mothers with two short alleles on 5-HTTLPR received greater supportive coparenting from focal children's biological fathers, the risk of physical aggression was lower ($\beta = -.329$, $p = .047$). However, fathers' greater childcare provision to mothers with two short alleles on 5-HTTLPR was related to an increase in the risk of mothers' physical aggression toward children.

Multigroup Analysis by Fathers' Residential and Marital Status

Originally, I assumed that fathers' greater coparenting and greater childcare provision would be associated with lower risk of mothers' psychological aggression and of mothers' physical aggression. And I anticipated the protective effects of fathers' supportive coparenting and childcare provision would be greater for genetically sensitive

mothers. However, after seeing the above results, I thought the meaning of fathers' supportive coparenting or that of childcare provision might differ by mothers' residential contexts with fathers. This is because the interaction between mothers' 5-HTTLPR and fathers' coparenting was consistent with my hypothesis (see Figure 6), but the interaction between mothers' 5-HTTLPR and fathers' childcare provision betrayed my hypothesis (see Figures 7 and 8). Thus, I conducted multigroup analysis by fathers' residential and marital status with mothers across three groups (1) married, (2) cohabiting, and (3) nonresident groups. For MLR analysis, Mplus does not produce chi-square scores, so it is impossible to use the original chi-square difference test to compare two models. Alternatively, to compare whether the results of multigroup analysis were different from the results of the whole sample, I used the difference testing using the Loglikelihood (Satorra & Bentler, 2010). The formula for this alternative difference testing is below.

$$cd = (p0 * c0 - p1*c1)/(p0 - p1)$$

*cd = the difference test scaling correction

*p0 = the number of free parameters in the nested model

*c0 = scaling correction factor in the nested model

*p1 = the number of free parameters in the comparison model

*c1 = scaling correction factor in the comparison model

$$TRd = -2 * (L0 - L1)/cd$$

*TRd = the alternative chi-square difference test

*L0 = Loglikelihood value for the nested model

*L1 = Loglikelihood value for the comparison model

The chi-square difference test using the Loglikelihood showed that the multigroup analysis results were significantly different from the results using the whole sample (cd = 1.131, TRd = 65.429, $p < .001$). If we look at the details, 572 of 1,571 couples were married, 380 were cohabiting, but 619 couples did not live together. The results showed that the effects of fathers' supportive coparenting and childcare provision on mothers' parenting behaviors were different by fathers' marital and residential status. For married mothers' psychological aggression, mothers' greater parenting stress increased the risk of mothers' later psychological aggression ($\beta = .136$, $p = .003$), but fathers' greater supportive coparenting reduced the risk of mothers' later psychological aggression ($\beta = -.130$, $p = .014$). For married mothers' physical aggression, experiencing more changes in family structure after childbirth increased the risk of mothers' later physical aggression ($\beta = .104$, $p = .038$), and having greater family socioeconomic resources reduced the risk of mothers' later physical aggression ($\beta = -.099$, $p = .009$). However, for cohabiting mothers, only greater parenting stress was associated with their later psychological aggression ($\beta = .144$, $p = .016$). For mothers who did not live together with focal children's fathers, if a focal child had a more difficult temperament in infancy, the risk of mothers' later physical aggression at child age nine increased ($\beta = .091$, $p = .036$). Fathers' supportive coparenting and childcare provision did not have direct effects on mothers' later psychological and physical aggression, but the effects of these predictors were moderated by mothers' 5-HTTLPR. If fathers provided greater supportive coparenting to mothers with two short alleles on 5-HTTLPR, the risk of mothers' later physical aggression was lower ($\beta = -.303$, $p = .044$). If mothers with two alleles on 5-

HTTLPR received greater childcare provision from fathers, they had higher risk of physical (β = .190, p = .023) and psychological (β = .176, p = .044) aggression.

Discussion

Recently, the genetics of parenting has been gaining more attention from academic scholars across fields, but as Mileva-Seitz, Bakermans-Kranenburg, and van IJzendoorn (2016)'s review summarized, little research has investigated how fathers' supports function in predicting mothers' parenting and whether the influence of fathers' supports differs by mothers' genetic sensitivity. Therefore, this study sought to examine direct effects of mothers' 5-HTTLPR, fathers' supportive coparenting and childcare provision, as well as interactions between mothers' 5-HTTLPR and fathers' coparenting or childcare supports on mothers' later psychological and physical aggression toward school aged children. Considering that the meaning of fathers' supports might differ by their residential and marital status with children's mothers, I also tested multigroup analysis by parental relationship status to see whether the effects of interest differed by family context. The major findings of this study are described below.

First, the results suggested that fathers' supportive coparenting and childcare provision might function differently in the mechanisms underlying mothers' parenting. It makes sense that fathers' greater supportive coparenting was associated with lower risk of mothers' psychological and physical aggression. However, the reason why fathers' greater childcare provision significantly predicted mothers' higher psychological or physical aggression needs more consideration. At the very least, these indicate that coparenting and childcare provision measure different aspects of father's support. If we

examine the items composing the measures of fathers' supportive coparenting and childcare provision, the biggest difference is the inclusion of the agreement/respect between parents in the measure of coparenting. For example, the items indicating fathers' supportive coparenting asked whether fathers provided support 'similar to mothers' expectation,' whereas the items indicating fathers' childcare provision did not ask whether fathers provided support as desired by mothers or not. For childcare provision, the items focused on the frequency or quantity of father involvement, and the results of path analyses showed positive association between fathers' greater childcare provision and mothers' higher psychological or higher physical aggression, especially for nonresident couples. Therefore, through the results, we can infer that the quality of fathers' coparenting might be more important than the quantity of fathers' supports to reduce mothers' negative parenting behaviors toward their children.

Of course, there are other possibilities as well. Fathers may have increased their childcare provision to compensate for mothers' poor quality of parenting as we can see in families with depressed mothers (i.e., Chang, Halpern, & Kaufman, 2007; Mezulis et al., 2004). Carlson, McLanahan, and Brooks-Gunn (2008) showed that higher coparenting predicted an increase in father involvement, but the opposite path like higher father involvement to higher coparenting was weak. Even when analyzing data from high-SES two-parent families, Jia and Schoppe-Sullivan (2011) reported that greater father involvement in child care was associated with greater coparenting conflict. It may be that mothers' interpretation of father involvement is critical. Fagan and Lee (2010) showed that mother's postpartum depressive symptoms were predicted by mother's satisfaction

with father involvement rather than mother's acknowledged amount of father involvement. Future research should focus on revealing the motivation for fathers' involvement and how the quantitative aspects of fathers' support may be interpreted differently by mothers.

Second, the significance and the effects of fathers' support appeared to differ by fathers' residential and marital contexts. Before conducting this study, I anticipated that fathers' greater supportive coparenting and greater childcare provision would be associated with lower levels of mothers' psychological and physical aggression later regardless of fathers' marital status. However, as we can see in Table 15, the trends appeared inconsistently between married, cohabiting, and nonresident couples. As described by Harris and Ryan (2004), mothers seem to play an important role in receiving and interpreting fathers' support. In the findings of this study, there was no difference by family context in the role of fathers' supportive coparenting in predicting reduced mothers' negative parenting behaviors. As I explained above, fathers' supportive coparenting seems to reduce the risk of mothers' aggressive parenting, while fathers' childcare provision seems to increase the risk. However, there were slight differences by family context in the significance of direct and interaction effects of fathers' support and mothers' genetic sensitivity.

For married couples, fathers' supportive coparenting predicted mothers' psychological aggression. This path did not differ by mothers' 5-HTTLPR. However, for cohabiting couples, neither fathers' supportive coparenting nor fathers' childcare provision were significantly associated with mothers' psychological or physical

aggression. Interestingly, for nonresident couples, mothers' 5-HTTLPR moderated the effects of fathers' supportive coparenting and childcare provision on mothers' physical aggression.

Why did the roles of fathers' supportive coparenting and childcare provision appear to differ across fathers' marital and residential status? More research is needed, but considering that the frequencies of fathers' coparenting/parenting supports among married couples were greater than those of nonresident father families, the direct effects of fathers' supportive coparenting reducing the risk of mothers' psychologically aggressive parenting might appear more obviously regardless of mothers' genetic sensitivity. In contrast, married mothers might have higher expectations about fathers' childcare support, explaining why the quantity of fathers' childcare provision was not significantly associated with married mothers' later parenting behaviors. However, for mothers who were not coresident with children's biological fathers, fathers' support was not as frequent and its variation is greater than that in other groups. Thus, the moderation by mothers' genetic sensitivity appeared because mothers with two short alleles might be more sensitive to receive fathers' supportive coparenting and to interpret the intention of fathers' childcare provision.

Of course, readers should be cautious to interpret and generalize the results of this study due to a number of reasons. First, the FFCWS oversampled low-income couples who experienced nonmarital births in large U.S. cities. Therefore, it is appropriate to say that the sample of this study represents low-income African-American or Hispanic populations, but it cannot represent two-married-parent families of higher socioeconomic

status or low-income populations living in rural areas of the United States. Second, the FFCWS collected data through different methods such as core parental surveys and home visits, but, due to the breadth of measures included, the FFCWS was not able to include the measurements especially for mothers' positive parenting and the context requiring fathers' childcare support. Lastly, gene and environment interaction studies are known to have low replication rates (i.e., Duncan & Keller, 2011). To strengthen the findings of this research, it would be good if future research tests whether the findings here replicates when analyzing data across different samples.

Despite several cautions, however, this study has made a unique contribution to understanding the mechanisms underlying mothers' parenting behaviors by revealing the paths from mothers' genetic sensitivity, fathers' supportive coparenting and childcare provision, and the interaction between mothers' genetic sensitivity and fathers' coparenting/childcare supports. To enrich the research revealing the mechanisms underlying mothers' parenting behaviors, I would like to suggest the further development of longitudinal measurements for mothers' parenting and fathers' coparenting behaviors. It would be better if researchers can include consistent measurements across time points. Without longitudinal measurement it is challenging to approach questions about causality, and longitudinal measurement at the very least allows us to control the possibility that mothers' poor parenting elicits an increase in fathers' childcare provision (i.e., higher father involvement in depressed-mother families). Also, future research using not only quantitative methods but also qualitative methods such as interviews would be also very helpful to reveal how mothers differently acknowledged fathers' supportive

coparenting and childcare provision and what mothers expect from their partners and how that differs by their residential contexts with fathers and cultures. In particular, nonmarital couples are more likely to experience relationship dissolution and father involvement tends to decrease greatly after relationship dissolution and mothers' multiple partner fertility (Tach, Mincy, & Edin, 2010). If we can understand the roles of fathers' supportive coparenting and childcare supports more deeply, it would help researchers to develop more effective prevention programs to enhance mothers' parenting by making more supportive environments thereby ultimately improving children's healthy social-emotional development as well.

Table 12. Demographic characteristics of the sample (N = 1,571)

	M (*SD*) *n* (%)	Min.	Max.
Mothers' age at birth	25.15 (5.94)	15	43
Fathers' age at birth	27.68 (7.02)	15	53
Mothers' education at birth	1,570 (100.0)		
Less than high school	464 (29.55)		
High school	481 (30.64)		
2-3yr college	427 (27.20)		
4yr college or higher	198 (12.61)		
Fathers' education at birth	1,542 (100.0)		
Less than high school	460 (29.83)		
High school	556 (36.06)		
2-3yr college	350 (22.70)		
4yr college or higher	176 (11.41)		
Marital status at childbirth	1,570 (100.0)		
Married	421 (26.82)		
Cohabiting	641 (40.83)		
Nonresident	508 (32.36)		
Marital status at child age 9	1,520 (100.0)		
Married	493 (32.43)		
Cohabiting	310 (20.39)		
Nonresident	717 (47.17)		
Household income at birth	35435.29 (33414.01)	0	133750
Household income at child age 9	49480.53 (55887.51)	0	900000
Mothers' race/ethnicities	1,566 (100.0)		
European-American	376 (24.01)		
African-American	747 (47.70)		
Hispanic	392 (25.03)		
Other/mixed	51 (3.26)		
Fathers' race/ethnicities	1,566 (100.0)		
European-American	329 (21.01)		
African-American	789 (50.38)		
Hispanic	393 (25.10)		
Other/mixed	55 (3.51)		
Child gender	1,571 (100.0)		
Boy	770 (49.01)		
Girl	801 (50.99)		

Table 13. Correlation matrix, means, and standard deviations

	1	2	3	4	5	6	7	8	9	*M* (*SD*)
1	1.00									1.50 (1.06)
2	-.052*	1.00								.73 (.86)
3	-.056*	-.015	1.00							2.79 (1.04)
4	-.054*	-.013	.159	1.00						2.04 (.69)
5	-.038	-.024	-.016	-.034	1.00					.16 (.37)
6	-.017	.005	-.126***	-.148***	.068**	1.00				2.44 (.75)
7	-.005	-.004	-.110***	-.136***	.053*	.739***	1.00			1.88 (1.07)
8	-.006	.005	.134***	.113***	-.073**	-.456***	-.494***	1.00		.72 (.77)
9	.040	.088*	-.155***	-.215***	.048	.171***	.190***	-.320***	1.00	1.30 (1.61)

Note. 1 = Mothers' psychological aggression at child age 9, 2 = Mothers' physical aggression at child age 9, 3 = Child temperament at age 1, 4 = Mothers' parenting stress at child age 3, 5 = Mothers' 5-HTTLPR, 6 = Fathers' supportive coparenting at child age 3, 7 = Fathers' childcare provision at child age 3, 8 = Family instability by child age 5, 9 = The lowest family poverty ratio by child age 5.

Table 14. Path coefficients for mothers' psychological and physical aggression

	Mothers' psychological aggression at child age 9	Mothers' physical aggression at child age 9
Child temperament at age 1	.040	.051
Mothers' parenting stress at age 3	.119***	.055*
Fathers' supportive coparenting (X1)	-.054	-.031
Fathers' childcare provision (X2)	-.049	-.023
Mothers' 5-HTTLPR (M)	-.023	.123
Family instability by age 5	.007	.065*
The lowest family poverty ratio by age 5	-.014	-.067**
X1 x M	-.246	-.329*
X2 x M	.232**	.218*
R^2	.040***	.035***

Note. N = 1,571. ***$p < .001$, **$p < .01$, *$p < .05$.

Table 15. Multigroup path coefficients for mothers' psychological and physical aggression by fathers' marital and residential status

	Married	Cohabiting	Nonresident
Y1 = Mothers' psychological aggression			
Mothers' 5-HTTLPR (M)	.405	-.342	-.039
Fathers' supportive coparenting (X1)	-.130*	-.053	-.053
Fathers' childcare provision (X2)	-.041	-.094	-.043
X1 x M	-.509	.059	-.193
X2 x M	.081	.242	.176*
(Covariates)			
Child temperament	.062	.023	.024
Mothers' parenting stress	.136**	.144*	.070
Family instability	.013	.013	.004
The lowest family poverty ratio	-.005	.039	.014
Y2 = Mothers' physical aggression			
Mothers' 5-HTTLPR (M)	.696	-.326	.114
Fathers' supportive coparenting (X1)	-.108	-.012	-.035
Fathers' childcare provision (X2)	-.039	-.075	.012
X1 x M	-.866	.402	-.303*
X2 x M	.163	-.017	.190*
(Covariates)			
Child temperament	-.005	.052	.091*
Mothers' parenting stress	.034	.097	.027
Family instability	.104*	.063	-.006
The lowest family poverty ratio	-.099**	-.019	.005
R^2			
Psychological aggression	.071**	.044*	.030*
Physical aggression	.070**	.034	.022*

Note. N = 1,571. ***$p < .001$, **$p < .01$, *$p < .05$.

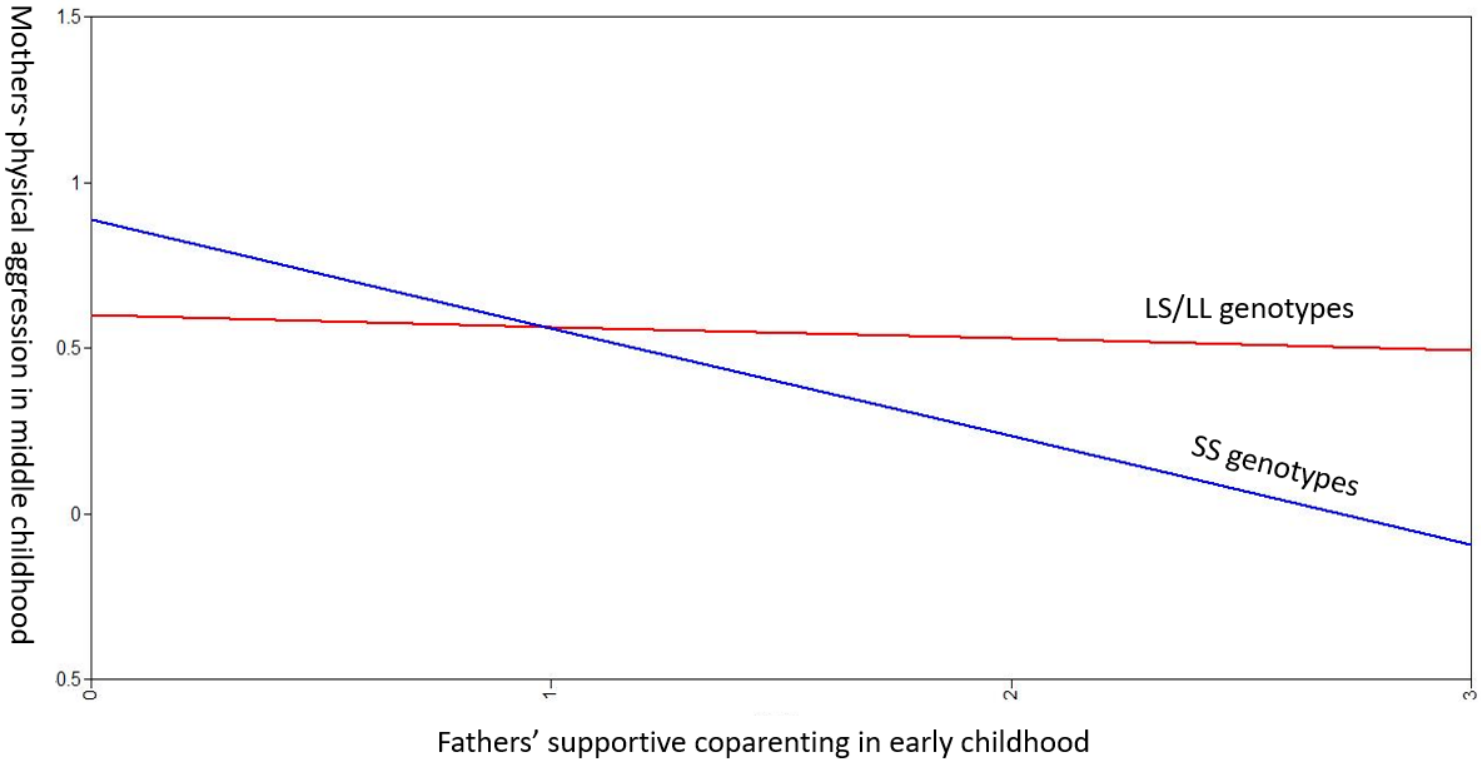


Figure 6. Interaction between mothers' 5-HTTLPR and fathers' coparenting support predicting mothers' physical aggression

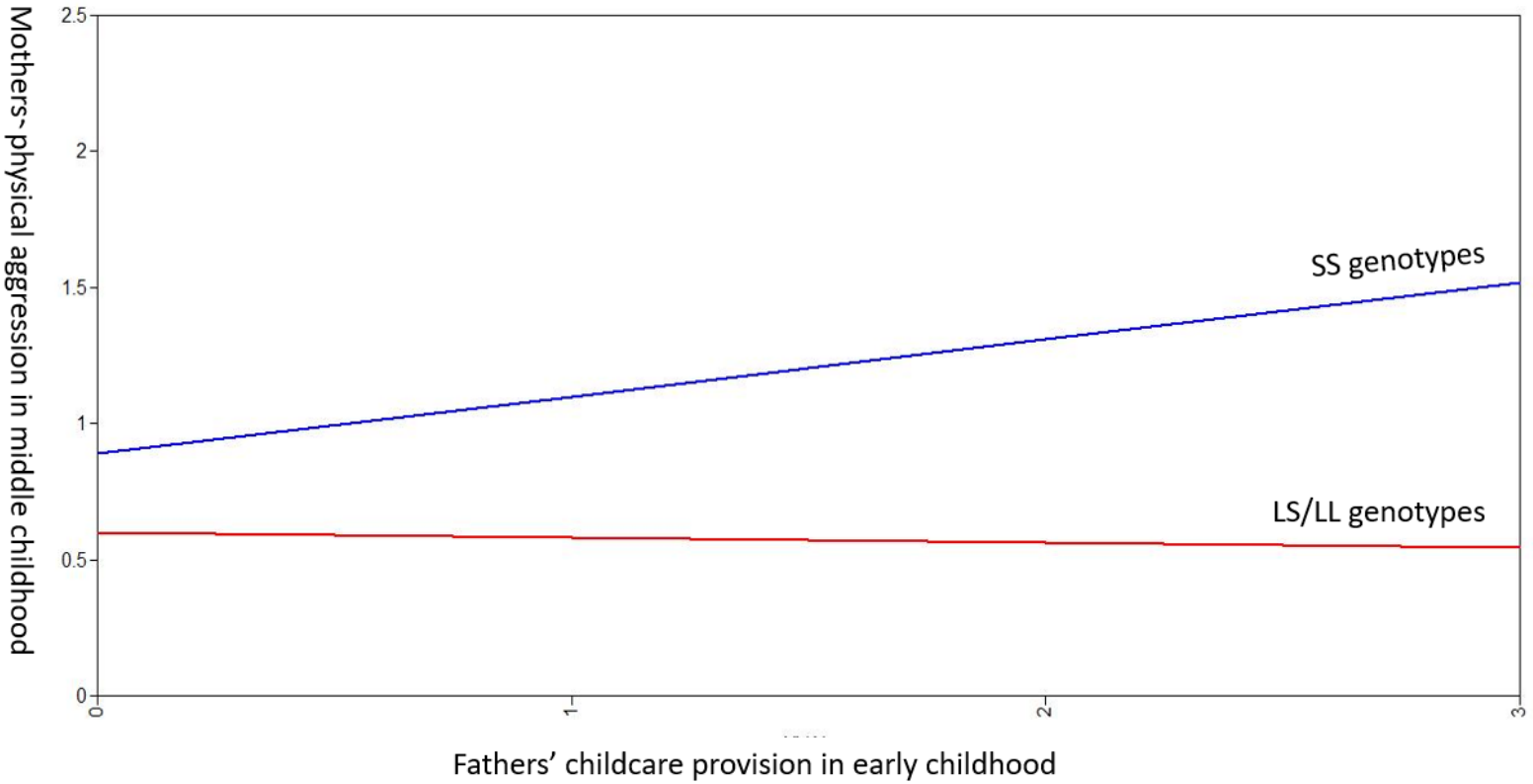


Figure 7. Interaction between mothers' 5-HTTLPR and fathers' childcare provision prediciting mothers' physical aggression

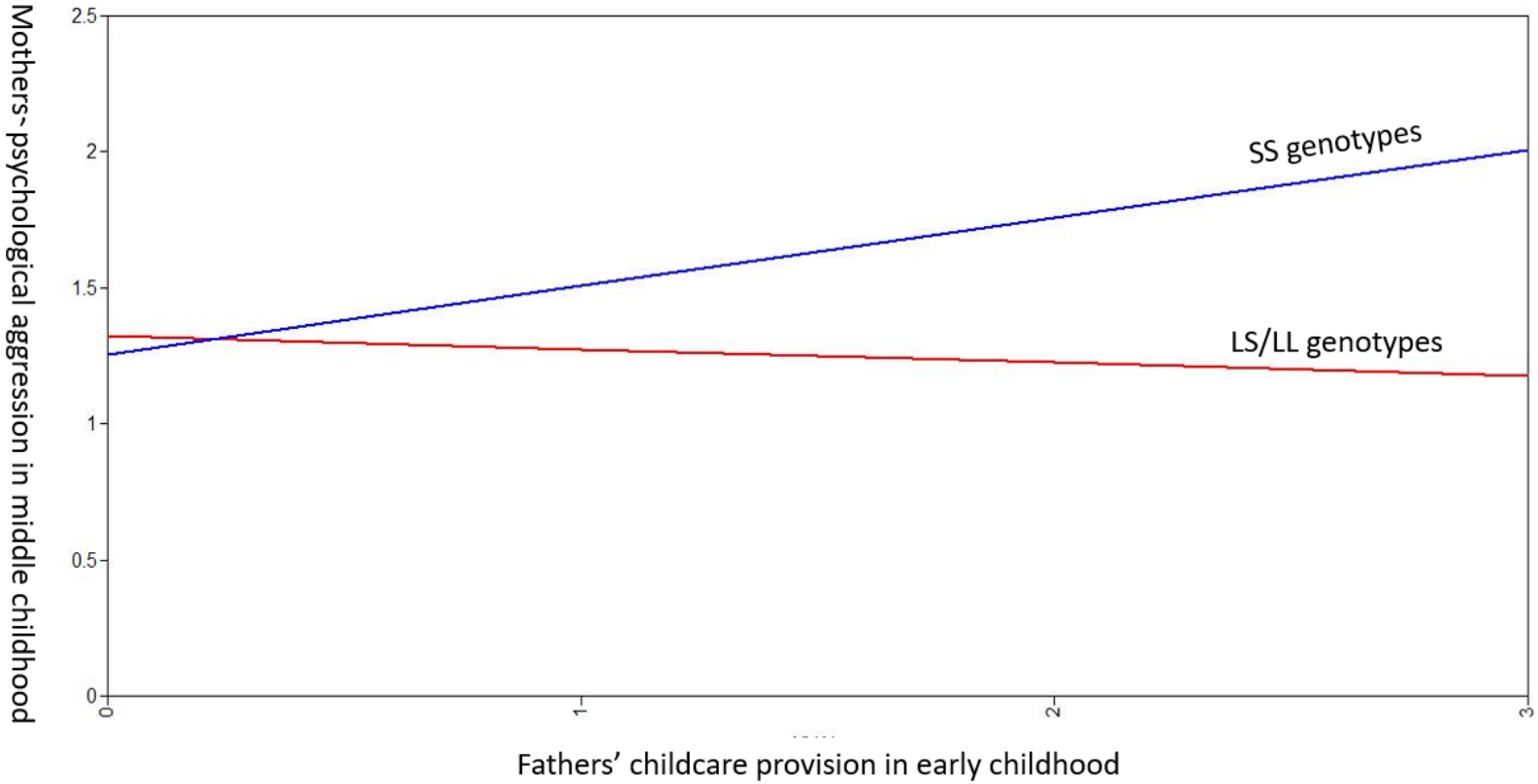


Figure 8. Interaction between mothers' 5-HTTLPR and fathers' childcare provision predicting mothers' psychological aggression

Chapter 5: Final Thoughts

Genetic Sensitivity, Parenting, and Children's Social-Emotional Development

In this dissertation, gene and environment interplay was tested in three different ways. The first study tested how children's serotonin genes are associated with children's social competence, the second study tested how mothers' and children's genetic sensitivity are related to child temperament, mothers' aggressive behaviors, and child internalizing behavior problems, and the third study tested how mothers' serotonin transporter gene interacts with fathers' coparenting and childcare provision to reduce mothers' aggressive behaviors toward children. These three studies have revealed interesting new insights regarding gene-environment interplay in the mechanisms underlying mothers' parenting and children's social-emotional development.

First, including children's and mothers' serotonin-related genes can help enhance our understanding of children's social-emotional development, because these genes appear to play roles in children's social-emotional development and mothers' parenting behaviors toward children. In the first study, children having two minor alleles on the 5-HTTLPR or STin2 VNTR were more sensitive to the effects of attachment security in their development of social competence. This finding supports the diathesis model (Ingram & Luxton, 2005) and differential susceptibility (Ellis & Boyce, 2011) perspective by showing that having two minor alleles on serotonin genes played an

important role to increase a person's vulnerability or sensitivity in social-emotional development. In the second study, the passive gene and environment correlation was strongly supported and the active gene and environment correlation was partially supported. This means that children's genetic sensitivity tends to be inherited from mothers, and that children with greater genetic sensitivity are at greater risk of receiving more aggressive parenting from their genetically sensitive mothers (passive rGE). Children with a minor allele on 5-HTTLPR and TPH2 also tend to show more difficult temperament and to develop internalizing behavior problems (active rGE). Compared to passive and active gene and environment correlation, the evidence for evocative gene and environment correlation was weak in Study 2. In the third study, mothers who had two minor alleles on 5-HTTLPR received greater effects from fathers' supports in relation to their aggressive behaviors toward children. These results suggest that developmental studies including genetic sensitivity of both parents and children can help researchers to better identify at-risk families who might benefit most from intervention or prevention programs.

Second, when individual genetic sensitivity contributes to our understanding of the mechanisms underlying child social-emotional development and mothers' parenting behaviors, the effects of individual genetic sensitivity are more likely to appear as a mediation or a moderation effect rather than a direct effect. Relevant findings of this dissertation are consistent with previous research (see Beauchaine, Hinshaw, & Gatzke-Kopp, 2008, for a review) indicating that individual genotype does not determine a person's phenotype. Rather, individual genotypes raise the amount of the influence from

earlier life experience or environmental stimuli on one's social-emotional behavioral outcomes (Beauchaine et al., 2008).

Related to this, one interesting finding of this dissertation is that the factor interacting with individual genetic sensitivity can be mothers' or children's perceived environment, instead of an objective environmental condition itself. For example, in the first study, while the interactions between children's serotonin-related genes and their early attachment significantly predicted children's later social competence. Considering the rule of parsimony, I did not include the results after adding the interaction between neighborhood/family socioeconomic condition and children's individual genetic sensitivity. But the interactions between family SES and children's genetic sensitivity were not significant to predict their later social competence. In the third study, the effects of fathers' supportive coparenting and childcare provision were different by mothers' genetic sensitivity especially for nonresident-father families, which implies that genetically sensitive mothers tend to have greater effects of fathers' supports and that mothers' perceptions of fathers' supports play an important role.

Sometimes, one's genotype seems to have little power to explain the developmental paths than its phenotype. For example, in this dissertation, the second study shows that individual genetic sensitivity significantly influences one's own social-emotional behaviors (i.e., mothers' genetic sensitivity → more aggressive parenting behaviors, children's genetic sensitivity → more difficult temperament). However, the assumption of evocative gene and environment correlation was not supported. In other words, the indirect path from child temperament to children's internalizing behavior

problems mediated by mothers' aggressive parenting behaviors was significant but the indirect path after adding children's genetic sensitivity at the beginning became nonsignificant. It shows weaker connection between children's genetic sensitivity, child temperament, and mothers' aggressive parenting behaviors. In addition, maternal parenting stress was significantly associated with children's lower social competence (Study 1) and mothers' higher aggressive behaviors (Study 3). There was no significant difference by children's genetic sensitivity in the effects of maternal parenting stress on children's behaviors. And the effects of maternal parenting stress on mothers' aggressive behaviors did not changed by mothers' genetic sensitivity.

By including not only environmental factors but also serotonin genes, this dissertation has made important contributions to reveal the roles of genetic sensitivity in children's social-emotional development from infancy to early and middle childhood. Moreover, this dissertation was among the first to examine the roles of genes and environments in mothers' parenting, while simultaneously considering the roles of fathers. To sum, it is important to include both genetic factors and environmental factors in research on family processes and children's development. Beauchaine et al. (2008) pointed out that gene and environment studies that did not account for the effects of environmental factors appropriately might overestimate the effects of genetic factors. Developmental scientists should remember that integrating individual genetic sensitivity can enlarge our understanding of the mechanisms of children's social-emotional development and mothers' parenting but the effects of genetic factors can be marginal.

Investigating the effects of environmental factors still remains critical to understanding mothers' parenting behaviors and children's social-emotional development.

By analyzing a large dataset representing a high-risk low-income population, this set of studies had many advantages to examine gene and environment interplay. The FFCWS's longitudinal design including data on family demographic characteristics, interparental relationships, parenting behaviors, and child social-emotional behavioral outcomes from child birth to child age 1, 3, 5, and 9 years old also made it possible to test a variety of moderation and mediation paths to understand how earlier phenomena (i.e., temperament or attachment) related to later behaviors.

However, this set of studies also has some important limitations. First, the FFCWS is not a twin study. Given that there is no data about identical or fraternal twins, it is hard to measure the amount of the influence of shared environment and that of non-shared environment. Because of this, the studies in this dissertation are closer to molecular genetic rather than behavioral genetic research, and given that there is no data from siblings in the FFCWS, the studies in this dissertation belong to association studies rather than linkage studies under molecular genetics (see Beauchaine et al., 2008 for more information). If the FFCWS collected saliva samples from focal children's siblings and identified whether siblings had the same biological parents, researchers could better study gene and environment interplay using this dataset.

Second, given that association studies which compare those who have specific alleles and those who do not require stronger theoretical background (Beauchaine & Neuhaus, 2008), it is a weakness in association studies that there are not enough studies

published so far and a lot of roles of candidate genes are not revealed yet. In developmental science, there has been only a short history of including individual genetic sensitivity in research. Therefore, there is not yet enough research to congregate the findings of association studies. This is the reason why I included only serotonin-related genes in this dissertation. Even among serotonin-related genes, there are many studies investigating 5-HTTLPR, but only a few that have included STin2 or TPH2 rs4570625.

Exploratory, data-driven research like that in this dissertation, has the strength of extending our knowledge, but it is a weakness to have little support from previous studies. Considering the benefits of the recent trends toward data-driven approaches (e.g., machine learning, latent class/profile analysis), my dissertation studies might contribute to show novel findings with more accurate estimates from the data but the results from this approach might be applicable only for the specific population studied. More research in association studies would be beneficial to address the criticism of low replication rate. By accumulating relevant studies, we can have more information regarding which genotype is stronger to predict a certain phenotype and how the genotype plays a role in the development of the phenotype.

Third, the studies in this dissertation included one or two genes to predict behavior outcomes, but future research may benefit from using a polygenic approach. I also considered apolygenic approach in these studies, but I chose single gene approaches because the polygenic approach weakened the findings about gene and environment interaction and correlations. I think the major reason why I could not get a better result from a polygenic approach is because the index for which genes is related to the outcome

variable is not fully revealed for children's general social-emotional outcomes or for mothers' parenting behaviors(compared to depression studies). As I mentioned earlier, the FFCWS included 13 genes, but the studies in this dissertation were not able to include all the genes typed by FFCWS because (1) the theoretical background was weak, and (2) the preliminary results identifying latent groups using multiple genes were not significant. Instead, since it was hard to find three-way interactions between two genes and other predictors, I included two genes in a parallel way within one study. For example, the first study tested 5-HTTLPR and Stin2 in the same model and the second study included 5-HTTLPR and TPH2 in the same model. If future research finds interesting results using polygenic approach, it would be great to adopt this approach in future research on gene and environment interplay in mothers' parenting and children's social-emotional development.

Last, since this dissertation targeted early (5 years old) and middle (9 years old) childhood, I focused on children, parents, and their family relationships/environments. However, based on the bioecological model (Bronfenbrenner & Ceci, 1994), future research could extend the examination of environmental factors to include peer groups, romantic relationships, school environments, or social policies. This may be especially important for studies that extend beyond early and middle childhood to adolescence.

Understanding the roles of nature and nurture and how they work together is important for increasing the sophistication of our knowledge about human development. Scholarly interest in understanding and integrating individual genetic sensitivity in developmental research continues to increase. This dissertation started from the question

about whether and how individual genetic sensitivity plays a role in children's social-emotional development and mothers' parenting. The findings of this dissertation support the notion that incorporating genetic sensitivity can help shed light on the detailed mechanisms underlying human development and that the effects of environments can differ by individual genetic sensitivity. One interesting finding from this dissertation is that a person's perception of early life experience or received support can affect the moderation of genetic sensitivity from environment to social-emotional behaviors. The effects of some environmental factors which were not moderated by genetic sensitivity were also apparent. More research strengthening theoretical foundations and including more positive behavioral outcomes is recommended. Considering some evidence about greater effects of environmental factors among genetically sensitive people and theoretical support for the influence of heritability, more research on gene and environment interplay can help to identify which children and families need more support and how they can benefit most from intervention or prevention efforts.

References

Achenbach, T. M. (1982). Assessment and taxonomy of children's behavior disorders. In B. B. Lahey, & A. E. Kazdin (Eds.) *Advances in clinical child psychology* (Vol.5, pp. 1-38). Boston, MA: Springer.

Achenbach, T.M. (1992). *Manual for the Child Behavior Checklist 2-3 and 1992 Profile*. Burlington, VT: University of Vermont Department of Psychiatry.

Asendorpf, J. B., Denissen, J. J. A., & van Aken, M. A. G. (2009). Personality trajectories from early childhood through emerging adulthood. In W. Schneider & M. Bullock (Eds.), Human development from early childhood to early adulthood (pp. 119-144). New York, NY: Psychology Press.

Auerbach, J., Faroy, M., Ebstein, R., Kahana, M., & Levine, J. (2001). The association of the dopamine D4 receptor gene (DRD4) and the serotonin transporter promoter gene (5-HTTLPR) with temperament in 12-month-old infants. *Journal of Child Psychology & Psychiatry*, *42*(6), 777-783. doi: 10.1017/S0021963001007612

Auerbach, J., Geller, V., Lezer, S., Shinwell, E., Belmaker, R. H., Levine, J., & Ebstein, R. P. (1999). Dopamine D4 receptor (D4DR) and serotonin transporter promoter (5-HTTLPR) polymorphisms in the determination of temperament in 2-month-old infants. *Molecular Psychiatry*, *4*, 369-373.

Aviles, A. M., Anderson, T. R., & Davila, E. R. (2006). Child and adolescent social-emotional development within the context of school. *Child and Adolescent Mental Health, 11*(1), 32-39.

Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2007). Research review: Genetic vulnerability or differential susceptibility in child development: The case of attachment. *Journal of Child Psychology & Psychiatry, 48*(12), 1160-1173. doi:10.1111/j.1469-7610.2007.01801.x

Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2008). Oxytocin receptor (OXTR) and serotonin transporter (5-HTT) genes associated with observed parenting. *Social Cognitive & Affective Neuroscience, 3*(2), 128-134. doi:10.1093/scan/nsn004

Bakermans-Kranenburg, M. J., van IJzendoorn, M. H., Pijlman, F. T., Mesman, J., & Juffer, F. (2008). Experimental evidence for differential susceptibility: dopamine D4 receptor polymorphism (DRD4 VNTR) moderates intervention effects on toddlers' externalizing behavior in a randomized controlled trial. *Developmental Psychology, 44*(1), 293-300. doi:10.1037/0012-1649.44.1.293

Barnett, M. A. (2008). Economic disadvantage in complex family systems: Expansion of family stress models. *Clinical Child & Family Psychology Review, 11*(3), 145-161. doi: 10.1007/s10567-008-0034-z

Basten, M., Tiemeier, H., Althoff, R. R., vande Schoot, R., Jaddoe, V. W. V., Hofman, A., Hudziak, J. J., Verhulst, F. C., & van der Ende, J. (2016). The stability of problem behavior across the preschool years: An empirical approach in the

general population. *Journal of Abnormal Child Psychology, 44*, 393-404. doi: 10.1007/sl0802-015-9993-y

Bates, J. E., & Pettit, G. S. (2015). Temperament, parenting, and social development. In J. E. Grusec, P. D. Hastings (Eds.), *Handbook of socialization: Theory and research* (2nd Ed., pp. 372-397). New York, NY: Guilford.

Beauchaine, T. P., Hinshaw, S. P., & Gatzke-Kopp, L. (2008). Genetic and environmental influences on behavior. In T. P. Beauchaine & S. P. Hinshaw (Eds.), *Child and adolescent psychopathology* (pp. 58-90). Hoboken, NJ: John Wiley & Sons.

Beauchaine, T. P., & Neuhaus, E. (2008). Impulsivity and vulnerability to psychopathology. In T. P. Beauchaine & S. P. Hinshaw (Eds.), *Child and adolescent psychopathology* (pp. 129-156), Hoboken, NJ: John Wiley & Sons.

Beck, A. N., Cooper, C. E., McLanahan, S., & Brooks-Gunn, J. (2010). Partnership transitions and maternal parenting. *Journal of Marriage & Family, 72*(2), 219-233. doi:10.1111/j.1741-3737.2010.00695.x

Becker, K., El-Faddagh, M., Schmidt, M. H., & Laucht, M. (2007). Is the serotonin transporter polymorphism (5-HTTLPR) associated with harm avoidance and internalising problems in childhood and adolescence? *Journal of Neural Transmission, 114*(3), 395-402. doi: 10.1007/s00702-006-0577-4

Belsky, J. (1984). The determinants of parenting: A process model. *Child Development, 55*(1), 83-96.

Belsky, J., Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2007). For better and for worse: Differential susceptibility to environmental influences. *Current Directions in Psychological Science, 16*(6), 300-304. doi:10.1111/j.1467-8721.2007.00525.x

Belsky, J., & Pluess, M. (2009). Beyond diathesis stress: Differential susceptibility to environmental influences. *Psychological Bulletin, 135*(6), 885-908. doi:10.1037/a0017376

Berk, L. E., & Meyers, A. B. (2016). *Infants and children: Prenatal through middle childhood*. Boston, MA: Pearson.

Betts, J., Gullone, E., & Allen, J. S. (2009). An examination of emotion regulation, temperament, and parenting style as potential predictors of adolescent depression risk status: A correlational study. *British Journal of Developmental Psychology, 27*, 473-485. doi:10.1348/026151008X314900

Bohlin, G., Hagekull, B., & Rydell, A. M. (2000). Attachment and social functioning: A longitudinal study from infancy to middle childhood. *Social Development, 9*(1), 24-39. doi:10.1111/1467-9507.00109

Bongers, I. L., Koot, H. M., van der Ende, J., & Verhulst, F. C. (2004). Developmental trajectories of externalizing behaviors in childhood and adolescence. *Child Development, 75*(5), 1523-1537. doi: 10.1111/j.1467-8624.2004.00755.x

Bowlby, J. (1988). *A secure base: Parent-child attachment and healthy human development*. New York, NY: Basic Books.

Bowlby, J. (1982). *Attachment* (2nd ed.). New York, NY: Basic Books.

Bridgett, D. J., Gartstein, M. A., Putnam, S. P., McKay, T., Iddins, E., Robertson, C., Ramsay, K., & Rittmueller, A. (2009). Maternal and contextual influences and the effect of temperament development during infancy on parenting in toddlerhood. *Infant Behavior & Development, 32*(1), 103-116. doi:10.1016/j.infbeh.2008.10.007

Bronfenbrenner, U. (1979). *The ecology of human development: Experiments by nature and design*. Cambridge, MA: Harvard University Press.

Bronfenbrenner, U., & Ceci, S. J. (1994). Nature-nuture reconceptualized in developmental perspective: A bioecological model. *Psychological review, 101*(4), 568-586.

Brown, S. M., & Hariri, A. R. (2006). Neuroimaging studies of serotonin gene polymorphisms: Exploring the interplay of genes, brain, and behavior. *Cognitive, Affective, & Behavioral Neuroscience, 6*(1), 44-52.

Byrd, A. L., & Manuck, S. B. (2014). MAOA, childhood maltreatment, and antisocial behavior: Meta-analysis of a gene-environment interaction. *Biological Psychiatry, 75*(1), 9-17. doi: 10.1016/j.biopsych.2013.05.004

Campbell, S. B., Spieker, S., Burchinal, M., Poe, M. D., & NICHD ECCRN. Trajectories of aggression from toddlerhood to age 9 predict academic and social functioning through age 12. *Journal of Child Psychology and Psychiatry, 47*(8), 791-800. doi: 10.1111/j.1469-7610.2006.01636.x

Canli, T., Congdon, E., Constable, R. T., & Lesch, K. P. (2008). Additive effects of serotonin transporter and tryptophan hydroxylase-2 gene variation on neural

correlates of affective processing. *Biological Psychology, 79*, 118-125. doi: 10.1016/j.biopsycho.2008.01.004

Carlson, M. J., McLanahan, S. S., & Brooks-Gunn, J. (2008). Coparenting and nonresident fathers' involvement with young children after a nonmarital birth. *Demography, 45*(2), 461-488.

Caspi, A. (2000). The child is father of the man: Personality continuities from childhood to adulthood. *Journal of Personality and Social Psychology, 78*(1), 158-172.

Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A., Craig, I. W., Harrington, H., McClay, J., Mill, J., Martin, J., Braithwaite, A., & Poulton, R. (2003). Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science, 301*(5631), 386-389. doi: 10.1126/science.1083968

Caspi, A., Hariri, A. R., Holmes, A., Uher, R., & Moffitt, T. E. (2010). Genetic sensitivity to the environment: The case of the serotonin transporter gene and its implications for studying complex disease and traits. *American Journal of Psychiatry, 167*(5), 509-527.

Cents, R. A., Kok, R., Tiemeier, H., Lucassen, N., Székely, E., Bakermans-Kranenburg, M. J., Hofman, A., Jaddoe, V. W. V., van IJzendoorn, M. H., Verhulst, F. C., & Lambregtse-van den Berg, M. P. (2014). Variations in maternal 5-HTTLPR affect observed sensitive parenting. *Journal of Child Psychology & Psychiatry, 55*(9), 1025-1032. doi:10.1111/jcpp.12205

Chan, Y. C. (1994). Parenting stress and social support of mothers who physically abuse their children in Hong Kong. *Child Abuse & Neglect, 18*(3), 261-269.

Chang, J. J., Halpern, C. T., & Kaufman, J. S. (2007). Maternal depressive symptoms, father's involvement, and the trajectories of child problem behaviors in a US national sample. *Archives of Pediatrics & Adolescent Medicine, 161*(7), 697-703.

Cheung, A. K., Harden, K. P., & Tucker-Drob, E. M. (2014). Gene × environment interactions in early externalizing behaviors: Parental emotional support and socioeconomic context as moderators of genetic influences? *Behavior Genetics, 44*(5), 468-486. doi:10.1007/s10519-014-9664-8

Choi, J. K., & Pyun, H. S. (2014). Nonresident fathers' financial support, informal instrumental support, mothers' parenting, and child development in single-mother families with low income. *Journal of Family Issues, 35*(4), 526-546.

Cipriano, E. A., & Stifter, C. A. (2010). Predicting preschool effortful control from toddler temperament and parenting behavior. *Journal of Applied Developmental Psychology, 31*, 221-230. doi: 10.1016/j.appdev.2010.02.004

Clark, L. A., Kochanska, G., & Ready, R. (2000). Mothers' personality and its interaction with child temperament as predictors of parenting behavior. *Journal of Personality and Social Psychology, 79*(2), 274-285. doi:10.1037//0022-3514.79.2.274

Claussen, A. H., & Crittenden, P. M. (1991). Physical and psychological maltreatment: Relations among types of maltreatment. *Child Abuse & Neglect, 15*(1-2), 5-18. doi:10.1016/0145-2134(91)90085-R

Conger, R. D., Conger, K. J., & Martin, M. J. (2010). Socioeconomic status, family processes, and individual development. *Journal of Marriage & Family, 72*(3), 685-704. doi:10.1111/j.1741-3737.2010.00725.x

Cooper, J. L., Masi, R., & Vick, J. (2009). *Social-emotional development in early childhood: What every policymaker should know.* Retrieved from Columbia University Academic Commons website: https://doi.org/10.7916/D83B67VS

de Lara, C. L., Dumais, A., Rouleau, G., Lesage, A., Dumont, M., Chawky, N., Alda, M., Benkelfat, C., & Turecki, G. (2006). STin2 variant and family history of suicide as significant predictors of suicide completion in major depression. *Biological Psychiatry, 59*(2), 114-120. doi:10.1016/j.biopsych.2005.06.021

Deater-Deckard, K., & Panneton, R. (2017). Unearthing the developmental and intergenerational dynamics of stress in parent and child functioning. In K. Deater-Deckard & R. Panneton (Eds), *Parental stress and early child development*. Cham, Switzerland: Springer. doi: 10.1007/978-3-319-55376-4_1

Dekker, M. C., Ferdinand, R. F., van Lang, N. D., Bongers, I. L., van der Ende, J., & Verhulst, F. C. (2007). Developmental trajectories of depressive symptoms from early childhood to late adolescence: gender differences and adult outcome. *Journal of Child Psychology & Psychiatry, 48*(7), 657-666. doi: 10.1111/j.1469-7610.2007.01742.x

Denham, S. A. (2006). Social-emotional competence as support for school readiness: What is it and how do we assess it? *Early Education & Development, 17*(1), 57-89.

Dick, D. M., Agrawal, A., Keller, M. C., Adkins, A., Aliev, F., Monroe, S., Hewitt, J. K., Kendler, K. S., & Sher, K. J. (2015). Candidate gene–environment interaction research: Reflections and recommendations. *Perspectives on Psychological Science, 10*(1), 37-59. doi:10.1177/1745691614556682

Duncan, L. E., & Keller, M. C. (2011). A critical review of the first 10 years of candidate gene-by-environment interaction research in psychiatry. *American Journal of Psychiatry, 168*(10), 1041-1049. doi:10.1176/appi.ajp.2011.11020191

Eaves, L., Sillberg, J., Erkanli, A. (2003). Resolving multiple epigenetic pathways to adolescent depression. *Journal of Child Psychology & Psychiatry, 44*(7), 1006-1014.

Ellis, B. J., & Boyce, W. T. (2011). Differential susceptibility to the environment: Toward an understanding of sensitivity to developmental experiences and context. Development & Psychopathology, 23(1), 1-5. doi:10.1017/S095457941000060X

Ellis, B. J., Boyce, W. T., Belsky, J., Bakermans-Kranenburg, M. J., & Van IJzendoorn, M. H. (2011). Differential susceptibility to the environment: An evolutionary-neurodevelopmental theory. *Development & Psychopathology, 23*(1), 7-28.

Erikson, E. H. (1950). *Childhood and society*. New York, NY: W. W. Norton & Company.

Fagan, J., & Lee, Y. (2010). Perceptions and satisfaction with father involvement and adolescent mothers' postpartum depressive symptoms. *Journal of Youth and Adolescence, 39*(9), 1109-1121. doi:10.1007/s10964-009-9444-6

Fan, J. B., & Sklar, P. (2005). Meta-analysis reveals association between serotonin transporter gene STin2 VNTR polymorphism and schizophrenia. *Molecular Psychiatry, 10*, 928-938. doi:10.1038/sj.mp.4001690

Feinberg, M. E. (2003). The internal structure and ecological context of coparenting: A framework for research and intervention. *Parenting: Science and Practice, 3*(2), 95-131.

Feinberg, M. E., Button, T. M. M., Neiderhiser, J. M., Reiss, D., & Hetherington, E. M. (2007). Parenting and adolescent antisocial behavior and depression: Evidence of genotype x parenting environment interaction. *Archives of general psychiatry, 64*(4), 457-465.

Florez, G., Saiz, P., Garcia-Portilla, P., Álvarez, S., Nogueíras, L., Morales, B., Alvarez, V., Coto, E., & Bobes, J. (2008). Association between the Stin2 VNTR polymorphism of the serotonin transporter gene and treatment outcome in alcohol-dependent patients. *Alcohol & Alcoholism, 43*(5), 516-522. doi:10.1093/alcalc/agn048

Forget-Dubois, N., Boivin, M., Dionne, G., Pierce, T., Tremblay, R. E., & Pérusse, D. (2007). A longitudinal twin study of the genetic and environmental etiology of maternal hostile-reactive behavior during infancy and toddlerhood. *Infant Behavior & Development, 30*(3), 453-465.

Forman, E. M., & Davies, P. T. (2003). Family instability and young adolescent maladjustment: The mediating effects of parenting quality and adolescent

appraisals of family security. *Journal of Clinical Child & Adolescent Psychology, 32*(1), 94-105.

Fragile Families and Child Welling Study (2008). *Introduction to the Fragile Families public use data: Baseline, one-year, three-year, and five-year core telephone data*. Retrieved from https://fragilefamilies.princeton.edu/sites/fragilefamilies/files/ff_public_guide_0to5.pdf

Fragile Families and Child Wellbeing Study (2013). *Scales documentation and question sources for the nine-year wave of the Fragile Families and Child Wellbeing Study*. Retrieved from https://fragilefamilies.princeton.edu/sites/fragilefamilies/files/ff_scales9.pdf

Fragile Families and Child Wellbeing Study (2015). *Fragile Families genetic component/DNA restricted use data appendage: 9-year follow-up wave*. Retrieved from https://fragilefamilies.princeton.edu/sites/fragilefamilies/files/ff_gen_9y_res2_2018.01.31.pdf

Furlong, M. J., Morrison, G. M., & Jimerson, S. R. (2004). Externalizing behaviors of aggression and violence and the school context. In R. B. Rutherford, M. M. Quinn, & S. R. Mathur, (Eds.), *Handbook of research in behavioral disorders* (pp.243-261). New York, NY: Guilford.

Gresham, F. M., & Kern, L. (2004). Internalizing behavior problems in children and adolescents. In R. B. Rutherford, M. M. Quinn, & S. R. Mathur, (Eds.), Handbook of research in behavioral disorders (pp.262-281). New York, NY: Guilford.

Gressier, F., Calati, R., & Serretti, A. (2016). 5-HTTLPR and gender differences in affective disorders: A systematic review. *Journal of Affective Disorders, 190*, 193-207. doi:10.1016/j.jad.2015.09.027

Guterman, N. B., Lee, S. J., Taylor, C. A., & Rathouz, P. J. (2009). Parental perceptions of neighborhood processes, stress, personal control, and risk for physical child abuse and neglect. *Child Abuse & Neglect, 33*, 897-906. doi: 10.1016/j.chiabu.2009.09.008

Gutknecht, L., Jacob, C., Strobel, A., Kriegebaum, C., Müller, J., Zeng, Y., Markert, C., Escher, A., Wendland, J., Reif, A., Mössner, R., Gross, C., Brocke, B., & Lesch, K. (2007). Tryptophan hydroxylase-2 gene variation influences personality traits and disorders related to emotional dysregulation. *International Journal of Neuropsychopharmacology, 10*(3), 309-320.

Hamer, D. H., Greenberg, B. D., Sabol, S. Z., & Murphy, D. L. (1999). Role of the serotonin transporter gene in temperament and character. *Journal of Personality Disorders, 13*(4), 312-328. doi:10.1521/pedi.1999.13.4.312

Hankin, B. L., Nederhof, E., Oppenheimer, C. W., Jenness, J., Young, J. F., Abela, J. R. Z., Smolen, A., Ormel, J., & Oldehinkel, A. J. (2011). Differential susceptibility in youth: Evidence that 5-HTTLPR × positive parenting is associated with

positive affect 'for better and worse'. *Translational Psychiatry, 1,* e44. doi:10.1038/tp.2011.44

Hariri, A. R., Mattay, V. S., Tessitore, A., Kolachana, B., Fera, F., Goldman, D., Egan, M. F., & Weinberger, D. R. (2002). Serotonin transporter genetic variation and the response of the human amygdala. *Science, 297*(5580), 400-403. doi:10.1126/science.1071829

Harmon, D. K., & Perry, A. R. (2011). Fathers' unaccounted contributions: Paternal involvement and maternal stress, *Families in Society, 92*(2), 176-182. doi: 10.1606/1044-3894.4101

Harris, K. M., & Ryan, S. (2004). Father involvement and the diversity of family context. In R. D. Day & M. E. Lamb (Eds.), *Conceptualizing and measuring father involvement* (pp. 293-319). Mahwah, NJ: Lawrence Erlbaum.

Hogan, A. E., Scott, K. G., & Bauer, C. R. (1992). The Adaptive Social Behavior Inventory (ASBI): A new assessment of social competence in high-risk three-year-olds. *Journal of Psychoeducational Assessment, 10*(3), 230-239. doi:10.1177/073428299201000303

Humphreys, K. L., Zeanah, C. H., Nelson III, C. A., Fox, N. A., & Drury, S. S. (2015). Serotonin transporter genotype (5HTTLPR) moderates the longitudinal impact of atypical attachment on externalizing behavior. *Journal of Developmental & Behavioral Pediatrics, 36*(6), 409-416. doi:10.1097/DBP.0000000000000171

Ingram, R. E., & Luxton, D. D. (2005). Vulnerability-stress models. In B. L. Hankin, & J. R. Z. Abela (Eds.), *Development of psychopathology: A vulnerability-stress perspective* (pp. 32-46). Thousand Oaks, CA: Sage.

Jia, R., & Schoppe-Sullivan, S. J. (2011). Relations between coparenting and father involvement in families with preschool-age children. *Developmental Psycholoty, 47*(1), 106-118. doi: 10.1037/a0020802

Jones, D., Greenberg, M., & Crowley, M. (2015). Early social-emotional functioning and public health: The relationship between kindergarten social competence and future wellness. *American Journal of Public Health, 105*(11), 2283-2290. doi: 10.2105/AJPH.2015.302630

Jorm, A. F., Prior, M., Sanson, A., Smart, D., Zhang, Y. & Easteal, S. (2000). Association of a functional polymorphism of the serotonin transporter gene with anxiety-related temperament and behavior problems in children: A longitudinal study from infancy to the mid-teens. *Molecular Psychiatry, 5*, 542-547.

Kang, J. (2013). Instrumental social support, material hardship, personal control and neglectful parenting, *Children & Youth Services Review, 35*, 1366-1373. doi: 10.1016/j.childyouth.2013.05.009

Karreman, A., de Haas, S., van Tuijl, C., van Aken, M. A., & Deković, M. (2010). Relations among temperament, parenting and problem behavior in young children. *Infant Behavior & Development, 33*(1), 39-49.

Kiff, C. J., Lengua, L. J., & Zalewski, M. (2011). Nature and nurturing: Parenting in the context of child temperament. *Clinical Child & Family Psychology Review, 14*(3), 251-301. doi: 10.1007/s10567-011-0093-4

Kim, Y., Lee, H., Yang, J., Hwang, J., & Yoon, H. (2009). A tryptophan hydroxylase 2 gene polymorphism is associated with panic disorder. *Behavior Genetics, 39*(2), 170-175.

Klahr, A. M., & Burt, S. A. (2014). Elucidating the etiology of individual differences in parenting: A meta-analysis of behavioral genetic research. *Psychological Bulletin, 140*(2), 544-586.

Laflamme, D., Pomerleau, A., & Malcuit, G. (2002). A comparison of fathers' and mothers' involvement in childcare and stimulation behaviors during free-play with their infants at 9 and 15 months. *Sex Roles, 47*(11-12), 507-518. doi:10.1023/A:1022069720776

Lamb, M. E. (2010). *The role of the father in child development*. Hoboken, NJ: Wiley.

Leidy, M. S., Schofield, T. J., & Parke, R. D. (2013). Fathers' contributions to children's social development. In Cabrera, N. J., & Tamis-LeMonda, C. S. (Eds.), *Handbook of father involvement: Multidisciplinary perspectives* (Vol. 2, pp. 151-167). New York, NY: Routledge.

Lengua, L. J. (2006). Growth in temperament and parenting as predictors of adjustment during children's transition to adolescence. *Developmental Psychology*, *42*(5), 819-832.

Lesch, K., P., Baling, U., Gross, J., Strauss, K., Wolozin, B. L., Murphy, D. L., & Riederer, P. (1994). Organization of the human serotonin transporter gene. *Journal of Neural Transmission, 95*(2), 157-162. doi:10.1007/bf01276434

Leve, L. D., Kim, H. K., & Pears, K. C. (2005). Childhood temperament and family environment as predictors of internalizing and externalizing trajectories from ages 5 to 17. *Journal of Abnormal Child Psychology, 33*(5), 505-520. doi:10.1007/s10802-005-6734-7

Lundberg, S, Pollak, R. A., & Stearns, J. (2016). Family inequality: Diverging patterns in marriage, cohabitation, and childbearing. *Journal of Economic Perspectives, 30*(2), 79-102.

MacCallum, R. C., & Austin, J. T. (2000). Applications of structural equation modeling in psychological research. *Annual Review of Psychology, 51*(1), 201-226. doi:10.1146/annurev.psych.51.1.201

Mandelli, L., & Serretti, A. (2013). Gene environment interaction studies in depression and suicidal behavior: an update. *Neuroscience & Biobehavioral Reviews, 37*(10), 2375-2397. doi:10.1016/j.neubiorev.2013.07.011

Martini, T. S., Root, C. A., & Jenkins, J. M. (2004). Low and middle income mothers' regulation of negative emotion: Effects of children's temperament and situational emotional responses. *Social Development, 13*(4), 515-530.

Mathieson, K. S., & Tambs, K. (1999). The EAS temperament questionnaire - Factor structure, age trends, reliability, and stability in a Norwegian sample. *Journal of*

Child Psychology & Psychiatry, 40(3), 431-439. doi:10.1017/s0021963098003680

Mayer, S. (1997). *What money can't buy: Family income and children's life chances*. Cambridge, MA: Harvard University Press.

McBride, B. A., Schoppe, S. J., & Rane, T. R. (2002). Child characteristics, parenting stress, and parental involvement: Fathers versus mothers. *Journal of Marriage & Family, 64*(4), 998-1011. doi: 10.1111/j.1741-3737.2002.00998.x

McLanahan, S., & Beck, A. N. (2010). Parental relationships in fragile families. *Future Child, 20*(2), 17-37.

McLoyd, V. C. (1990). The impact of economic hardship on Black families and children: Psychological distress, parenting, and socioemotional development. *Child Development, 61*(2), 311-346.

McLoyd, V. C. (1998). Socioeconomic disadvantage and child development, *American Psychologist, 53*(2), 185-204.

McLoyd, V. C., Jayaratne, T. E., Ceballo, R., & Borquez, J. (1994). Unemployment and work interruption among African American single mothers: Effects on parenting and adolescent socioemotional functioning. *Child Development, 65*(2), 562-589.

Mezulis, A. H., Hyde, J. S., & Clark, R. (2004). Father involvement moderates the effect of maternal depression during a child's infancy on child behavior problems in kindergarten. *Journal of Family Psychology, 18*(4), 575-588. doi:10.1037/0893-3200.18.4.575

Middlemiss, W. (2003). Brief report: Poverty, stress, and support: Patterns of parenting behavior among lower income Black and lower income White mothers. *Infant and Child Development, 12*, 293-300.

Mileva-Seitz, V., Kennedy, J., Atkinson, L., Steiner, M., Levitan, R., Matthews, S. G., Meaney, M. J., Sokolowski, M. B., & Fleming, A. S. (2011). Serotonin transporter allelic variation in mothers predicts maternal sensitivity, behavior and attitudes toward 6-month-old infants. *Genes, Brain & Behavior, 10*, 325-333. doi:10.1111/j.1601-183X.2010.00671.x

Mileva-Seitz, V. R., Bakermans-Kranenburg, B. J., & van IJzendoorn, M. H. (2016). Genetic mechanisms of parenting. *Hormones & Behavior, 77*, 211-223. doi:10.1016/j.yhbeh.2015.06.003

Mitchell, C., McLanahan, S., Hobcraft, J., Brooks-Gunn, J., Garfinkel, I., & Notterman, D. (2015). Family structure instability, genetic sensitivity, and child well-being. *American Journal of Sociology, 120*(4), 1195-1225. doi:10.1086/680681

Monroe, S. M., & Simons, A. D. (1991). Diathesis-stress theories in the context of life stress research: Implications for the depressive disorders. *Psychological Bulletin, 110*(3), 406-425. doi:10.1037/0033-2909.110.3.406

Munafò, M. R. (2012). The serotonin transporter gene and depression. *Depression & Anxiety, 29*(11), 915-917. doi:10.1002/da.22009

Murphy, D. L., Andrews, A. M., Wichems, C. H., Li, Q., Tohda, M., & Greenberg, B. (1998). Brain serotonin neurotransmission: An overview and update with an emphasis on serotonin subsystem heterogeneity, multiple receptors, interactions

with other neurotransmitter systems, and consequent implications for understanding the actions of serotonergic drugs. *The Journal of Clinical Psychiatry, 59*(S15), 4-12.

Muthén, B. O., Muthén, L. K., & Asparouhov, T. (2016). *Regression and mediation analysis using Mplus*. Los Angeles, CA: Muthén & Muthén.

Muthén, L. K., & Muthén, B. O. (2007). *Mplus user's guide*. Los Angeles, CA: Muthén & Muthén.

Nugent, N. R., Tyrka, A. R., Carpenter, L. L., & Price, L. H. (2011). Gene-environment interactions: Early life stress and risk for depressive and anxiety disorders. *Psychoparmacology, 214*, 175-196. doi: 10.1007/s00213-010-2151-x

Oldehinkel, A. J., Veenstra, R., Ormel, J., de Winter, A. F., & Verhulst, F. C. (2006). Temperament, parenting, and depressive symptoms in a population sample of preadolescents. *Journal of Child Psychology & Psychiatry, 47*(7), 684-695. doi:10.1111/j.1469-7610.2005.01535.x

Patterson, G. (1986). Performance models for antisocial boys. *American Psychologist, 41*, 432-444.

Pearlin, L. I., & Schooler, C. (1978). The structure of coping. *Journal of Health & Social Behavior, 19*(1), 2-21. doi:10.2307/2136319

Piaget, J. (1971). *Biology and knowledge*. Chicago, IL: University of Chicago.

Pinderhughes, E. E., Dodge, K. A., Bates, J. E., Pettit, G. S., & Zelli, A. (2000). Discipline responses: Influences of parents' socioeconomic status, ethnicity,

beliefs about parenting, stress, and cognitive-emotional processes. *Journal of Family Psychology, 14*(3), 380-400.

Pine, D. S., & Grun, J. (1999). Childhood anxiety: Integrating developmental psychopathology and affective neuroscience. *Journal of Child & Adolescent Psychopharmacology, 9*(1), 1-12. doi:10.1089/cap.1999.9.1

Plomin, R., DeFries, J. C., & Loehlin, J. C. (1977). Genotype-environment interaction and correlation in the analysis of human behavior. *Psychological Bulletin, 84*(2), 309-322.

Qi, C. H., & Kaiser, A. P. (2003). Behavior problems of preschool children from low-income families: Review of the literature. *Topics in Early Childhood Special Education, 23*(4), 188-216.

Reichman, N. E., Teitler, J. O., Garfinkel, I., & McLanahan, S. S. (2001). Fragile Families: Sample and design. *Children & Youth Services Review, 23*(4-5), 303-326.

Risch, N., Herrell, R., Lehner, T., Liang, K., Eaves, L., Hoh, J., Griem, A., Kovacs, M., Ott, J., & Merikangas, K. R. (2009). Interaction between the serotonin transporter gene (5-HTTLPR), stressful life events, and risk of depression: A meta-analysis. *Journal of the American Medical Association, 301*(23), 2462-2471.

Roisman, G. I., Newman, D. A., Fraley, R. C., Haltigan, J. D., Groh, A. M., & Haydon, K. C. (2012). Distinguishing differential susceptibility from diathesis–stress: Recommendations for evaluating interaction effects. *Development & Psychopathology, 24*(2), 389-409. doi:10.1017/S0954579412000065

Rothbart, M. K., & Bates, J. E. (2007). Temperament. In W. Damon, R. M. Lerner, & N. Eisenberg (Eds.), *Handbook of child psychology* (Vol. 3, pp.99-166). Hoboken, NJ: John Wiley & Sons. doi: 10.1002/9780470147658.chpsy0303

Rubin, K. H., Burgess, K. B., & Hastings, P. D. (2002). Stability and social–behavioral consequences of toddlers' inhibited temperament and parenting behaviors. *Child Development, 73*(2), 483-495.

Rutter, M. (2006). *Genes and behavior: Nature-nurture interplay explained*. Malden, MA: Blackwell.

Sameroff, A. (2009). The transactional model. In A. Sameroff (Ed.), *The transactional model of development: How children and contexts shape each other* (pp.3-21). Washington, DC: American Psychological Association.

Satorra, C., & Bentler, P. M. (1994). Corrections to test statistics and standard errors in covariance structure analysis. In A. von Eye & C. C. Clogg (Eds.), *Latent variable analysis: Applications for developmental research* (pp. 399–419). Thousand Oaks, CA: Sage.

Satorra, A., & Bentler, P. M. (2010). Ensuring positiveness of the scaled difference chi-square test statistics. *Psychometrika, 75*(2), 243-248. doi:10.1007/S11336-009-9135-Y

Scarr, S., & McCartney, K. (1983). How people make their own environments: A theory of genotype → environment effects. *Child Development, 54*(2), 424-435.

Schoppe, S. J., Mangelsdorf, S. C., & Frosch, C. A. (2001). Coparenting, family process, and family structure: Implications for preschoolers' externalizing behavior problems. *Journal of Family Psychology, 15*(3), 526-545.

Schoppe-Sullivan, S. J., Settle, T., Lee, J., & Kamp Dush, C. M. (2016). Supportive coparenting relationships as a haven of psychological safety at the transition to parenthood. *Research in Human Development, 13*(1), 32-48.

Schoppe- Sullivan, S. J., Weldon, A. H., Claire Cook, J., Davis, E. F., & Buckley, C. K. (2009). Coparenting behavior moderates longitudinal relations between effortful control and preschool children's externalizing behavior. *Journal of Child Psychology & Psychiatry, 50*(6), 698-706. doi:10.1111/j.1469-7610.2008.02009.x

Schreiber, J. B., Nora, A., Stage, F. K., Barlow, E. A., & King, J. (2006). Reporting structural equation modeling and confirmatory factor analysis results: A review. *The Journal of Educational Research, 99*(6), 323-338. doi:10.3200/JOER.99.6.323-338

Sen, S., Burmeister, M., Ghosh, D. (2004). Meta-analysis of the association between serotonin transporter promoter polymorphism (5-HTTLPR) and anxiety-related personality traits. *American Journal of Medical Genetics Part B (Neuropsychiatric Genetics), 127*(B), 85-89.

Shala, A., & Grajcevci, A. (2016). The relation between socio-economic status (SES) and early development: empirical findings and theoretical perspectives. *Socioeconomica, 5*(10), 309-329. doi:dx.doi.org/10.12803/SJSECO.51011

Shaw, D. S., Keenan, K., Vondra, J. I., Delliquardi, E., & Giovannelli, J. (1997). Antecedents of preschool children's internalizing problems: A longitudinal study of low-income families. *Journal of the American Academy of Child & Adolescent Psychiatry, 36*(12), 1760-1767.

Shaw, D. S., Vondra, J. I., Hommerding, K. D., Keenan, K., & Dunn, M. (1994). Chronic family adversity and early child behavior problems: A longitudinal study of low income families. *Journal of Child Psychology and Psychiatry, 35*(6), 1109-1122.

Shumow, L., Vandell, D. L., & Posner, J. K. (1998). Harsh, firm, and permissive parenting in low-income families. *Journal of Family Issues, 19*(5), 483-507.

Simons, L. G., Wickrama, K. A. S., Lee, T. K., Landers-Potts, M., Cutrona, C., & Conger, R. D. (2016). Testing family stress and family investment explanations for conduct problems among African American adolescents. *Journal of Marriage & Family, 78*(2), 498-515.

Smits, K. M., Smits, L. J. M., Schouten, J. S. A. G., Stelma, F. F., Nelemans, P., & Prins, M. H. (2004). Influence of SERTPR and STin2 in the serotonin transporter gene on the effect of selective serotonin reuptake inhibitors in depression: A systematic review. *Molecular Psychiatry*, *9*(5), 433-441. doi:10.1038/sj.mp.4001488

Spinath, F. M., & Bleidorn, W. (2017). The new look of behavioral genetics in social inequality: Gene-environment interplay and life chances. *Journal of Personality, 85*(1), 5-9. doi:10.1111/jopy.12268

Stoltz, S., Beijers, R., Smeekens, S., & Deković, M. (2017). Diathesis stress or differential susceptibility? Testing longitudinal associations between parenting,

temperament, and children's problem behavior. *Social Development, 26*(4), 783-796. doi:10.1111/sode.12237

Straus, M. A., & Field, C. J. (2003). Psychological aggression by American parents: National data on prevalence, chronicity, and severity. *Journal of Marriage and Family, 65*(4), 795-808. doi:10.1111/j.1741-3737.2003.00795.x

Straus, M. A., Hamby, S. L., Finkelhor, D., Moore, D.W., & Runyan, D. (1998). Identification of child maltreatment with the parent-child conflict tactics scales: Development and psychometric data for a national sample of American parents. Child Abuse & Neglect, 22, 249 – 270. doi:10.1016/S0145-2134(97)00174-9

Sulik, M. J., Eisenberg, N., Lemery-Chalfant, K., Spinrad, T. L., Silva, K. M., Eggum, N. D., Betkowski, J. A., Kupfer, A., Smith, C. L., Gaertner, B., Stover, D. A., & Verrelli, B. C. (2012). Interactions between serotonin transporter gene haplotypes and quality of mothers' parenting predict the development of children's noncompliance. *Developmental Psychology, 48*(3), 740-754.

Tach, L., Mincy, R., & Edin, K. (2010). Parenting as a "package deal": Relationships, fertility, and nonresident father involvement among unmarried parents. *Demography, 47*(1), 181-204. doi:10.1353/dem.0.0096

Teubert, D., & Pinquart, M. (2010). The association between coparenting and child adjustment: A meta-analysis. *Parenting: Science & Practice, 10*, 286-307. doi:10.1080/15295192.2010.492040

van IJzendoorn, M. H., Belsky, J., & Bakermans-Kranenburg, M. J. (2012). Serotonin transporter genotype 5HTTLPR as a marker of differential susceptibility? A meta-

analysis of child and adolescent gene-by-environment studies. *Translational Psychiatry, 2*, e147. doi: 10.1038/tp.2012.73

van Zeijl, J., Mesman, J., Stolk, M. N., Alink, L .R. A., van IJzendoorn, M. H., Bakermans-Kranenburg, M. J., & Juffer, F. & Koot, H. M. (2007). Differential Susceptibility to discipline: The moderating effect of child temperament on the association between maternal discipline and early childhood externalizing problems. *Journal of Family Psychology, 21*(4), 626-636. doi: 10.1037/0893-3200.21.4.626

Vernon-Feagans, L., & Cox, M. (2013). Poverty, rurality, parenting, and risk: An introduction. *Monographs of the Society for Research in Child Development, 78*(5), 1-23.

Walther, D. J., Peter, J. U., Bashammakh, S., Hortnagl, H., Voits, M., Fink, H., & Bader, M. (2003). Synthesis of serotonin by a second tryptophan hydroxylase isoform. *Science, 299*(5603), 76.

Weeland, J., Overbeek, G., de Castro, B. O., & Matthys, W. (2015a). Underlying mechanisms of gene-environment interactions in externalizing behavior: A systematic review and search for theoretical mechanisms. *Clinical Child & Family Psychology Review, 18*(4), 413-442. doi:10.1007/s10567-015-0196-4

Weeland, J., Slagt, M., Brummelman, E., Matthys, W., de Castro, B. O., & Overbeek, G. (2015b). 5-HTTLPR expression outside the skin: An experimental test of the emotional reactivity hypothesis in children. *PLoS ONE 10*(11), e0141474. doi:10.1371/journal.pone.0141474

Wolff, J. C., & Ollendick, T. H. (2006). The comorbidity of conduct problems and depression in childhood and adolescence. *Clinical Child and Family Psychology Review, 9*(3/4), 201-220. doi:10.1007/s10567-006-0011-3

Woodhouse, S., Ayers, S., & Field, A. P. (2015). The relationship between adult attachment style and post-traumatic stress symptoms: A meta-analysis. *Journal of Anxiety Disorders, 35*, 103-117. doi:10.1016/j.janxdis.2015.07.002

Yavorsky, J. E., Kamp Dush, C. M., & Schoppe- Sullivan, S. J. (2015). The production of inequality: The gender division of labor across the transition to parenthood. *Journal of Marriage and Family, 77*(3), 662-679. doi: 10.1111/jomf.12189

Zahn-Waxler, C., Klimes-Dougan, B., & Slattery, M. J. (2000). Internalizing problems of childhood and adolescence: Prospects, pitfalls, and progress in understanding the development of anxiety and depression. *Development & Psychopathology, 12*(3), 443-466. doi:10.1017/s0954579400003102

Zimmermann, P., & Spangler, G. (2016). Effects of gene × attachment interaction on adolescents' emotion regulation and aggressive hostile behavior towards their mothers during a computer game. *Frontiers in Human Neuroscience, 10*, 254. doi:10.3389/fnhum.2016.00254