GENETIC MODERATION OF COMMUNITY AND FAMILY LEVEL ENVIRONMENTS:
EXTENDING THE BIOECOLOGICAL MODEL

by

JUNHAN CHO

(Under the Direction of Steven M. Kogan)

ABSTRACT

In the ecological systems perspective, individuals are viewed as developing within a set of embedded social contexts. The transactional concept that is central to this perspective underscores the bidirectional nature of human development, which is a product of ongoing interactions between an individual and the environment. The bioecological model further emphasizes that individual-level biological factors can interact with a broad array of multiple-level contexts to affect human development. Over the past two decades, investigations of how an individual’s genetic status interacts with the environment have transformed studies from the bioecological perspective. To date, these studies have focused on the genetic status of youth or children interacting with aspects of their social ecology, usually the family environment. Despite calls for inclusion of the intricate interplay among multiple levels of social contexts and personal biological characteristics, including genotype, little research has considered the complexity inherent in the bioecological perspective. This dissertation is designed to address this need.

In the first study, I examine a series of youth and parent $G \times E$ effects linking community disadvantage to youth risk behavior. This study tests the hypothesis that parental genotype $\times$ community disadvantage will influence parenting behavior, which, in turn, will
interact with youth genotype to predict youth’s planful future orientation, a proximal predictor of risk behavior. In the second study, I investigate transactional relations between parenting practices and child self-regulation considering the genetic status of both parent and child. This study examines the interaction effect of parenting practices and children’s genotypes on their self-regulation, which, in turn, interacts with parental genotypes to predict changes in parenting behavior.

INDEX WORDS: Bioecology, Gene by environment interaction, Community disadvantage, Parenting practices, Future orientation, Risk behavior, Self-regulation, Dopaminergic genes
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by

JUNHAN CHO
B.S., Yonsei University, Republic of Korea, 2005
M.A. Yonsei University, Republic of Korea, 2010

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by

JUNHAN CHO

Major Professor: Steven M. Kogan

Committee: K. A. S. Wickrama
Chalandra M. Bryant
Leslie G. Simons

Electronic Version Approved:

Suzanne Barbour
Dean of the Graduate School
The University of Georgia
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DEDICATION

To my family
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CHAPTER 1
INTRODUCTION AND LITERATURE REVIEW

Introduction

The ecological systems perspective on human development emphasizes the influence of embedded social contexts on individual development (Bronfenbrenner, 1986) and the importance of transactions between the developing individual and the environment. The bioecological model, a refinement of ecological systems theory, further emphasizes that individual-level biological factors can interact with a broad array of multiple-level contexts to affect human development (Bronfenbrenner & Ceci, 1994). Over the past two decades, research on gene by environment interaction (G × E) processes has investigated how an individual’s genetic status interacts with the environment to influence his or her development. To date, these G × E studies have focused on the genetic status of an individual youth or child interacting with aspects of their social environments, usually the family environment. Although studies have examined the interaction of multiple levels of social contexts and personal biological characteristics including genotypes (Kogan et al., 2014; Simons et al., 2011; Wickrama, O’Neal, & Lee, 2013), little research has considered the complexity inherent in the bioecological perspective; specifically, how multiple levels of context and mutual influence processes between individuals and the environment affect youth development. This dissertation is designed to address this need.

This dissertation comprises two distinct but related studies. In the first study, I examine a series of G × E effects of parents and youth linking community disadvantage to youth risk behavior through protective parenting and youth’s planful future orientation. In the second study,
I investigate transactional relations between parenting practices and child self-regulation considering the genetic status of both parent and child. In the remainder of this chapter, the theoretical framework guiding and connecting two studies is presented. This is followed by a discussion of the unique contributions of the dissertation studies to the current literature, and a brief overview of the studies is provided.

**Theoretical Frameworks**

**Ecological systems theory and bioecological theory**

The ecological systems model is an evolving theoretical system for the scientific study of human development over time (Bronfenbrenner, 1986). In the ecological systems perspective, individuals are viewed as developing within a set of embedded social contexts (Bronfenbrenner, 1986). For example, children and youth develop within families, and families are influenced by and to some extent select into community contexts. This model underscores not only the multiple levels of environmental context that affect individual development but the complex transactions between individuals and the environment. Transactional models of child development acknowledge the bidirectional nature of influence between individuals and aspects of their context (Pettit & Arsiwalla, 2008; Sameroff, 2009). For example, children are not passively shaped by family, school, and other socialization processes, but actively influence the environments they develop within and the relationships they have with key socialization agents. This insight has encouraged examinations of bidirectional influence processes between parents and children who are potentially influential environments for each other’s development.

Core to the importance of Bronfenbrenner’s original formulation of the ecological systems theory was the potential for individual-level biological factors to interact with a broad array of environmental factors to affect human development (Bronfenbrenner & Ceci, 1994).
Expanding on this component of human development, Bronfenbrenner and Ceci (1994) articulated a **bioecological** model that emphasizes the functioning of **proximal processes** in child development. Proximal processes are dynamic interactions between individuals’ biological characteristics and their immediate social and physical environments. The bioecological model focuses attention on empirically testable mechanisms through which genetic potentials are actualized in the context of environmental variation. Recent research on **G × E** processes exemplifies this perspective. In modern G × E research from an ecological perspective, data on an individual’s genetic status is hypothesized to affect how environmental variability influences an individual’s development. Multiple studies focused on children and youth have documented the extent to which individuals’ genetic variations interact with environmental factors to predict developmental outcomes (see Bakermans-Kranenburg & van IJzendoorn, 2011; Belsky & Pluess, 2009).

**Differential susceptibility theory**

Human development researchers have long hypothesized that individuals vary in the extent to which their development is affected by environmental factors (Burmeister, McInnis, & Zollner, 2008). Based on G × E studies, a number of candidate genes that affect neurotransmitter activity have been shown to modulate the influence of environmental input on individual behavior. The majority of G × E studies have focused on the extent to which individuals carrying certain “vulnerability genes” or “risk alleles” are relatively more likely to develop poorly when exposed to environmental stressors (see Rende & Plomin, 1992). For example, studies have documented that children with specific “risk” alleles that affect dopaminergic functioning, compared to those without the alleles, are more likely to report conduct problem when exposed to harsh parenting (see Belsky et al., 2009; Popper et al., 2007). These studies are
consistent with a *diathesis-stress* model (Monroe & Simons, 1991; Zuckerman, 1999), in which specific alleles confer vulnerability to individuals that will only be realized in stressful circumstances. In contrast to the diathesis-stress model, Belsky and Pluess (2009) hypothesized that many genotypes thought to act as vulnerability alleles may act to amplify both risk-promoting and development-enhancing environmental conditions. From this *differential susceptibility* perspective, persons vary in their sensitivity to the environment in a “for better and for worse” manner (Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007; Belsky & Pluess, 2009; Ellis et al., 2011).

Informed by the differential susceptibility perspective (Belsky & Pluess, 2009), a number of recent G × E studies have examined how both positive and negative social environments interact with genotypes to predict a range of developmental outcomes. Among several candidate genes from multiple neurotransmitter systems, accumulating evidence suggests the importance of genes that affect the function of the dopaminergic neurotransmitter system (Bakermans-Kranenburg & van IJzendoorn, 2011). For example, genetic moderation effects of dopaminergic genes, for better and for worse, have been detected children’s and youth’s substance use (Beach et al., 2010; Brody et al., 2012, 2014), sexual risk behavior (Kogan et al., 2014), and conduct problems (Bakermans-Kranenburg & van IJzendoorn, 2006; 2011). Although there are conflicting findings and many studies fail to test for differential susceptibility (Kahn, Khoury, Nichols, & Lamphear, 2003; Propper et al., 2007), meta-analytic reviews support the association between specific dopamine genes and differential susceptibility (Bakermans-Kranenburg & van IJzendoorn, 2011; Belsky & Pluess, 2009). That is, specific dopaminergic genes may function as *plasticity alleles* that confer susceptibility or amplify environmental effect. With exposure to enhancing environments, plasticity alleles amplify the
benefit of the environment. Based on the literature, this dissertation research focuses on dopaminergic genes.

**Contribution to the Literature of the Present Dissertation**

Although many G × E studies have examined important aspects of the bioecological framework, there are significant limitations to this literature. First, most G × E studies have focused on the interaction of a singular aspect of the rearing environment, usually family processes, and child genotype to predict developmental outcomes. In contrast, bioecological theory suggests that multiple levels of context jointly influence individual behavior. Studies, however, have not examined simultaneously G × E processes at multiple environmental levels. For example, although research documents that parenting effects on child behavior are modulated by child genotype, parents also are influenced by a range of environmental factors (including community contexts and child’s behavior) that may interact with parental genotype to inform parenting behavior (Beach et al., 2012; van IJzendoorn et al., 2008). Examinations of how G × E processes affect multiple members of the family, however, have not been conducted.

A second limitation to the present research base involves the lack of attention to bidirectional influence processes between parents and children. For example, although research based on bioecological and G × E models specifies parents as an environment of children’s development, child behavior is also a potential environment that interacts with parent genotype (Mills-Koonce et al., 2007). To date, little research has focused on the potential genetic moderation of transactional relations between parenting and child behavior. Both these limitations are informed by a focus on genetic variation in a single individual or family member. An ecological systems perspective, however, suggests that the genetic status of multiple family
members will have complex interactions with transactional influences within a family system (Mills-Koonce et al., 2007).

**Overview of Studies**

To address these limitations, this dissertation research is designed to investigate $G \times E$ effects of multiple family members who interact within multilevel, transactional contexts. Two sets of hypotheses were tested using longitudinal data from rural African American youth and their parents participating in two separate studies. In both cases, families were participating in randomized prevention trials. Because intervention effects were not the focus of my hypotheses, assignment to an intervention was statistically controlled in all analyses. In *Study 1*, 361 African American youth and their parents were randomly recruited from lists that public schools provided. Three waves of data spanning 3 years were obtained; youth were age 16 at baseline.

In *Study 2*, study hypotheses were tested with a sample of 291 children and their parents. In the initial wave, children’s mean age was 11, and the data were collected four times; children’s mean age was 15 at the final time point. These data were selected due to the inclusion of multilevel contextual information on child development, parenting practices, as well as information on candidate dopaminergic genes that confer sensitivity for parents and children.

Informed by the bioecological framework, both studies focus on proximal processes through which the genetic status of parents and children interact with environmental factors to influence their behavioral and psychological outcomes. *Study 1*, described in Chapter 2, is titled *Parent and youth dopamine D4 receptor genotypes moderate multilevel contextual effects on rural African American youth's risk behavior*. This study examines multiple $G \times E$ processes linking multilevel contextual influences to predict youth risk behavior. Study hypotheses are summarized in Figure 1.1. I expected community disadvantage to influence youth risk behavior.
via a series of indirect effects involving protective parenting and youth’s psychosocial process when both parents’ and youth’s genotypes are considered. Community disadvantage was hypothesized to interact with parental genotype to predict engagement in protective parenting practices. Protective parenting, in turn, was hypothesized to interact with youth genotype to predict youth psychosocial development, which affected changes in risk behavior during mid- and late-adolescence. In each case, I also determined whether specific G × E interactions were best characterized as differential susceptibility or diathesis-stress models.

Study 2, presented in Chapter 3, is titled *Genetic moderation of transactions between parenting practices and child self-regulation: A focus on dopaminergic genes*. This study investigates transactional relations between parenting practices and child self-regulation considering genetic susceptibility of both parent and child. Hypotheses are summarized in Figure 1.2. I hypothesized that parenting practices would interact with children’s genotypes to forecast changes in child self-regulation. Child self-regulation, in turn, was hypothesized to interact with parental genotypes to predict changes in parenting behavior. Both G × E effects were examined to identify whether each one conform to a differential susceptibility or diathesis-stress model.

The remainder of this dissertation is organized as follows. The ensuing two chapters represent separate manuscripts of the aforementioned Study 1 and Study 2. In chapter 2 and 3, the significance, methods, findings, and discussion of these studies are described. Chapter 4 provides an integrative summary of the two studies, including implications and future directions for research.
Figure 1.1. Conceptual model of study 1
Figure 1.2. Conceptual model of study 2
CHAPTER 2

PARENT AND YOUTH DOPAMINE D4 RECEPTOR GENOTYPES MODERATE MULTILEVEL CONTEXTUAL EFFECTS ON RURAL AFRICAN AMERICAN YOUTH’S RISK BEHAVIOR

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1 Cho, J., & Kogan, S. M. Accepted by Development and Psychopathology. Reprinted here with permission of publisher, 7/20/2015.
Abstract

The present investigation extends research on gene by environment (G × E) interactions and youth risk behavior by linking multilevel contextual factors, such as community disadvantage and protective parenting practices, to both parental and youth dopamine D4 receptor (DRD4) genotypes. I expected community disadvantage to influence youth risk behavior via a series of indirect effects involving protective parenting and youth’s planful future orientation when both parents’ and youth’s DRD4 status was considered. Genetic moderation processes also were tested to determine whether they conformed to a diathesis-stress or a differential susceptibility model. Hypotheses were investigated with data from 361 rural African American youth and their parents assessed 3 times when youth were ages 16 to 19. Community disadvantage interacted with parental DRD4 status to predict low levels of protective parenting. Protective parenting, in turn, interacted with youth DRD4 status to forecast increases in youth’s planful future orientations, a proximal influence on changes in risk behavior. The parental DRD4 × community disadvantage interaction, but not youth DRD4 × protective parenting, conformed to a differential susceptibility model. Indirect effect analyses revealed a significant indirect path linking community disadvantage to youth risk behavior through a series of multilevel G × E interaction processes.

Keywords: gene by environment interaction, community disadvantage, protective parenting, future orientation, risk behavior
Introduction

Adolescents engage in numerous risk behaviors that compromise their health and well-being, including substance use, risky sexual activity, and delinquency. These risk behaviors frequently co-occur (Hair, Park, Ling, & Moore, 2009; Wu, Witkiewitz, McMahon, & Dodge, 2010) and engagement in multiple forms of risk behavior has been found to have an additive influence on poor health and suboptimal development in young adulthood (Hair et al., 2009; Mason et al., 2010). The present study focuses on risk behaviors reported by African American youth living in resource poor communities in the rural South. Historically, rural residence protected youth from engagement in many risk behaviors. Recent data indicate, however, that rural youth engage in risk behavior at rates that equal or exceed those of urban youth (Kogan, Berkel, Chen, Brody, & Murry, 2006; Milhausen et al. 2003). For African Americans, studies reveal that involvement in risk behaviors during adolescence disproportionately affects their education, employment, involvement with the criminal justice system, mental health, and physical health (Brook et al., 2004; Chatterji, 2006; Massoglia, 2008).

In the ecological-systems perspective, adolescents are viewed as developing within a set of embedded social contexts (Bronfenbrenner, 1986). For example, adolescents develop within families, and families select into and are influenced by community contexts. The bioecological model further emphasizes that individual-level biological factors can interact with a broad array of multiple-level contexts to affect human development (Bronfenbrenner & Ceci, 1994). Despite calls for inclusion of the intricate interplay among multiple levels of social contexts and personal biological characteristics, including genotype (Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2011), little research has considered this complexity.
The present study investigates genetic moderation processes in the development of rural African American youth’s risk behavior. It responds to the need for increasing sophistication in the characterization of gene and environment interaction (G × E) processes by investigating pathways linking multiple contextual levels and multiple family members’ genotypes (Mills-Koonce et al., 2007; Rutter, Moffitt, & Caspi, 2006). Consistent with bioecological theory focusing on the interaction of individuals and their proximal contexts (Bronfenbrenner & Morris, 2006), I hypothesized that, among rural African American families, disadvantaged community environments would interact with parental genotype to predict parenting behavior. Parenting behavior was hypothesized to interact with youth genotype to affect youth’s outlook on the future, which in turn would predict risk behavior. I further hypothesized that community disadvantage would influence youth risk behavior indirectly via this series of G × E interactions.

**Literature Review**

**Environmental Influences on Rural African American Youth’s Risk behavior**

Risk behaviors such as substance use, sexual risk, and delinquency pose significant concurrent and downstream threats to adolescents’ psychosocial and physical well-being (D’Amico, Ellickson, Collins, Martino, & Klein, 2005; Hair et al., 2009; Oesterle et al., 2004). These risk behaviors frequently co-occur, particularly in early adolescence (Biglan, Brennan, Foster, & Holder, 2004; Krueger, Markon, Patrick, Benning, & Kramer, 2007); however, substance use, sexual risk, and delinquency remain strongly associated throughout the high school years (Hair et al., 2009; Mustanski et al., 2013; Wu et al., 2010). Moreover, recent research indicates that engagement in multiple forms of risk behavior may have a particularly deleterious effect, forecasting increases in physical and mental health problems as well as poor academic and employment outcomes in young adulthood (Hair et al., 2009).
A substantial body of research documents the influence of community environments on youth’s involvement in substance use (Brooks, Magnusson, Spencer, & Morgan, 2012; Lambert, Brown, Philips, & Ialongo, 2004), risky sex (Browning, Burrington, Leventhal, & Brooks-Gunn, 2008), and delinquency (Leventhal & Brooks-Gunn, 2000; Sampson, Morenoff, & Gannon-Rowley, 2002). African American families disproportionately reside in disadvantaged communities, characterized by racial segregation, an important driver of multiple institutional disadvantages (Williams & Collins, 2001). This is true in the rural South, where many African American communities are characterized by elevated proportions of single-parent households, receipt of public assistance, and low household income (Boatright, 2009; Sampson, 2012; Williams & Collins, 2001; Wilson, 2012). Community disadvantage potentially affects youth risk behavior via a range of pathways including peer group influence, risk behavior opportunities, educational quality, and access to structured, after-school leisure activities (Chuang, Ennett, Bauman, & Foshee, 2005; Wallace & Muroff, 2002).

Studies of rural African American families suggest that a key pathway linking community disadvantage to youth risk behavior involves community effects on parenting practices (Brody et al., 2006; Kogan et al., 2011, 2013; Simons et al., 1996, 2005). For many African American parents, the daily stress associated with living in a disadvantaged community undermines their ability to engage in a set of risk-reducing protective parenting practices that include high levels of parental monitoring, involvement with youth’s academic activities, consistent discipline, and high levels of nurturance (Brody et al., 2001; Kogan et al., 2013; Mrug & Windle, 2009; Simons et al., 2005). This constellation of parenting practices has been linked to African American youth’s avoidance of substance use (Brody et al., 2009, 2014), sexual risk (Kogan et al., 2011), and conduct problems (Brody, Kogan, Chen, & Murry, 2008; Simons et al.,
2002). Conversely, youth who experience low levels of protective parenting are at elevated risk for engagement in multiple risk behaviors (Brody, Chen, & Kogan, 2010).

Direct effects of protective parenting on risk behavior are evident particularly in early- and mid-adolescence (East, Khoo, & Reyes, 2006; Farmer et al., 2004). As youth exhibit greater autonomy in late adolescence, however, attitudes and self-regulatory competencies internalized through interactions with parents become increasingly important (Brook & Brook, 1996; Brook, Brook, Gordon, Whiteman, & Cohen, 1990; Kogan et al., 2011). A planful future orientation is one such intrapersonal mechanism hypothesized to link protective parenting practices to risk behavior among rural African American youth during the high school years. A planful future orientation combines positive expectations about the future with an orientation toward setting goals and persisting in their accomplishment (Aronowitz, 2005; Brody et al., 2004; Kogan, Brody, & Chen, 2011; Wills, Sandy, & Yaeger, 2001). A planful future orientation locates adolescents’ primary set of psychological influences beyond their current circumstances, which renders them more attentive to the lasting consequences of their behaviors, enhancing their goal setting and persistence (Routledge & Arndt, 2005). In contrast, youth who lack a planful future orientation are influenced more by immediate situational factors and are less likely to inhibit emotional responses to negative environments. Investigations of planful future orientations among African American youth in rural and urban contexts reveal negative associations with substance use (Li et al., 2001; Wills et al., 2001), sexual risk behaviors (Bolland, 2003; Kogan et al., 2010; Li et al., 2001), and delinquency (Bolland et al., 2007; Caldwell, Wieber, & Cleveland, 2006; Kerpelman, Eryigit, & Stephens, 2008).

Disadvantaged communities have been hypothesized to affect rural African American youth’s risk behavior by undermining protective parenting, which in turn influences youth’s
development of a planful future orientation (Kogan et al., 2013). Prior research suggests that harsh environmental conditions lead youth to perceive their futures as unpredictable, undermining their development of a planful future orientation. This is a proximal risk factor for risk behavior (see Ellis, Figueredo, Brumbach, & Sclomer, 2009; Hill, Ross, & Low, 1997). Studies indicate that adolescents hold more optimistic views about their futures when they receive parental support (Dubow, Arnett, Smith, & Ippolito, 2001; Kenny, Blustein, Chaves, Grossman, & Gallagher, 2003; McCabe & Barnett, 2000), and that close relationships with parents are positively related to youth future orientation (Nurmi & Pulliainen, 1991; Seginer, Vermulst, & Shoyer, 2004). A planful future orientation may be particularly important for African American youth living in disadvantaged rural environments because they are exposed to few educational and vocational opportunities within their communities (Gore & Aseltine, 2003; Sum et al., 2002).

**Dopamine Receptor 4 Polymorphism and Susceptibility to Environmental Factors**

Recent studies indicate that both parents and youth vary considerably in the extent to which proximal environmental factors such as community disadvantage and protective parenting practices affect their behavior (Belsky & Pluess, 2009). A number of candidate genes that affect neurotransmitter activity are thought to modulate the influence of environmental input on individual behavior. Interactions between specific polymorphisms and family environments have been detected for youth substance use (Brody et al., 2009; Brody et al., 2014), sexual risk behavior (Guo, Tong, & Cai, 2008), and delinquent behavior (Caspi et al., 2002; Simons et al., 2011; Simons et al., 2012). A more modest literature suggests that genetic status may interact with aspects of parents’ proximal environments to affect their parenting behavior (Beach et al., 2012; van IJzendoorn, Bakermans-Kranenburg, & Mesman, 2008).
The majority of G x E studies investigating youth risk behavior conceptualize the interaction effect from a diathesis-stress perspective. This perspective focuses on the extent to which particular alleles confer vulnerability to adverse social conditions. In contrast, Belsky and Pluess (2009) suggested that some alleles may act to amplify both positive and negative environmental input. From this differential susceptibility perspective, persons who are more vulnerable to adverse social environments also benefit more from positive environments (Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007; Belsky & Pluess, 2009; Ellis et al., 2011).

The dopamine receptor 4 polymorphism, DRD4, is associated with neurocognitive systems that regulate risk and reward (Spear, 2011). Evidence is accumulating that supports the possibility that the DRD4 gene confers differential susceptibility to the environment (Bakermans-Kranenburg & van IJzendoorn, 2007, 2011). DRD4 is a variable nucleotide tandem repeat (VNTR) polymorphism composed of 16 amino acid (48-bp) repeat polymorphisms that range from 2 to 11 repeats. The 2, 3, 4, and 7 repeat versions account for about 98% of allelic variability (Lichter et al., 1993). Studies characterize DRD4 alleles as either “short” (s) or “long” (l), with the s category defined as having 6 or fewer repeats and the l category as having 7 or more repeats (Beach et al., 2012; Brody et al., 2012; Eisenberg et al., 2007; Laucht, Becker, Blomeyer, & Schmidt, 2007; McGeary, 2009). DRD4-l alleles appear to function in a way that yields a protein structure that produces less reactive D4 receptors in both in vitro and in vivo tests of responsiveness, resulting in weaker transmission of intracellular signals for those with at least one l allele versus two s alleles (Levitan et al., 2006; Asghari et al., 1995).

Dopamine regulation is a promising focus for understanding youth risk behavior. Dopamine is released in response to rewarding stimuli, including sexual activity and stimulant drugs. Reward signals initiate a phasic burst of mid-brain dopamine neurons, which induce
positive emotional states and organize the learning of cues that predict future rewards (Spear, 2000a). The dopaminergic system is thus engaged in attentional, motivational, and reward mechanisms (Robbins & Everitt, 1999). Reduced dopaminergic signaling impedes learning based on negative feedback (Klein et al., 2007), which in turn may lead to a relative insensitivity to environmental cues and rewarding stimuli (Spear, 2000b). This results in a strong preference for immediate reinforcement from the environment and elevated reactivity to high-intensity environmental cues (Tripp & Wickens, 2008).

Multiple studies support the hypothesis that $DRD4$ alleles confer differential sensitivity to environmental input. For example, Beach et al. (2010) found that African American youth carrying $DRD4$-$l$ were more responsive to the effects of protective parenting practices, which included child management skills and nurturant-involved parenting practices, on diminished substance use than were those with the $s$ allele. Additional evidence is summarized in a recent review and meta-analysis by Bakermans-Kranenburg and van IJzendoorn (2011). The present study hypothesized that $G \times E$ effects involving $DRD4$ status would demonstrate a pattern conforming to differential susceptibility effects.

**Indirect Effects of Community Disadvantage on Risk Behavior via Multilevel $G \times E$ Interactions**

To date, most $G \times E$ research on youth development and risk behavior has focused on the ways in which proximal family environments interact with youth genotype. Far fewer studies, however, have considered the interaction of parental genotype with the environment in affecting the kind of parenting to which youth are exposed in the family system (for exceptions see Beach et al., 2012; Mills-Koonce et al., 2007; van IJzendoorn et al., 2008). In the present investigation, we extended the research base on $G \times E$ interactions by linking multilevel contextual factors,
such as community disadvantage and protective parenting practices, to both parental and youth genotype. Study hypotheses are summarized in Figure 2.1.

I expected community disadvantage to influence youth risk behavior via a series of indirect effects involving protective parenting and youth’s planful future orientations when both parents’ and youth’s DRD4 status were considered. I predicted that community disadvantage would interact with parental DRD4 status to forecast their engagement in protective parenting practices. Protective parenting, in turn, was hypothesized to interact with youth DRD4 status to predict changes in youth’s planful future orientations, which would affect changes in risk behavior. In each case, I also determined whether specific G × E interactions would be best characterized as differential susceptibility or diathesis-stress models.

Methods

Participants

Study hypotheses were tested with 361 African American youth (159 male and 202 female) and their primary caregivers who resided in six counties in Georgia selected on the basis of their rurality (> 50% of census tracts designated rural) and the proportion of the population that was African American (> 20%). The communities are representative of the Southern “Black Belt” (Wimberly & Morris, 1997), a geographic concentration of rural poverty characterized by the nation’s worst economic and child health disparities by race (Hartley, 2004). Families were recruited randomly from lists that public schools provided. Data were collected within the context of a family-based prevention study at intervals timed to evaluate the prevention program. Because the present study hypotheses were not focused on intervention efficacy, random assignment to the intervention program or to an attention control program was controlled. Three waves of data spanning 3 years were obtained in 2006, 2008, and 2009. Youth’s mean ages were
16 years at Time 1 (T1; $SD = .56$), 17.5 years at Time 2 (T2; $SD = .54$), and 18.6 years at Time 3 (T3; $SD = .56$).

The families’ demographic characteristics were representative of the areas in which they lived (Boatright, 2009). Of the primary caregivers, 88.5% were the youth’s biological mothers, 33.8% of whom were married at baseline. The remaining caregivers were grandparents (7%), aunts/uncles (2%), siblings (0.6%), and other adults (1.9%). The caregivers’ mean age was 43 years ($SD = 8.29$) at T1, and their educational backgrounds ranged from less than a high school diploma (25%) to a bachelor’s or graduate degree (6%); the modal education level (69%) was a high school diploma or GED. Mean family income was $1,761 per month.

**Procedures**

Families were contacted and enrolled in the study by African American community liaisons who resided in the counties where the participants lived. The community liaisons were selected on the basis of their social contacts and standing in the community; they worked with the researchers on participant recruitment and retention. At all data collection points, parents gave written consent to minor youth’s participation, and youth gave written assent or consent to their own participation. During each assessment, one home visit lasting 2 hours was made to each family. At the visits, self-report questionnaires were administered to the primary caregiver and target youth via audio computer-assisted self-interviewing (ACASI) technology on laptop computers. Caregivers were paid $100 and youth were paid $50 at each assessment.

**Measures**

*Community disadvantage.* Community disadvantage was assessed using the 2006 census tract data from the American Community Survey. Participants’ residential addresses at T1 were geocoded and matched to census tracts. Census tracts generally include 1,500 to 8,000 residents
and are designed to be as homogenous as possible regarding population characteristics, living conditions, and economic status (U.S. Bureau of the Census, 2000). Tract boundaries reflect significant physical (e.g., roads, rivers, and railroads) and demographic (e.g., ethnic composition) features of neighborhoods, which are delineated with the advice of local community members. A total of 36 census tracts were identified in this study; the number of participants residing in a tract ranged from 1 to 41. Median household income in these census tracts ranged from $13,018 to $60,417; median income across the 36 tracts was $29,918. Consistent with prior research (Leventhal & Brooks-Gunn, 2000; Sampson, Raudenbush, & Earls, 1997; Simons et al., 2005), I developed a community disadvantage index using four indicators: percentage of female-headed households, percentage of households receiving Temporary Assistance for Needy Families (TANF), per capita annual income, and the proportion of African American families residing in the community. On the basis of studies of both physical morbidity and psychological dysfunction that support the basic concept of cumulative SES risk (see Brody et al., 2013, 2014), an index of cumulative community disadvantage was developed. Risk factor thresholds were set at the first quartile for percentages of female-headed households, receipt of TANF, and African American families, and at the fourth quartile for per capita annual income. Each indicator was coded dichotomously (0 if absent, 1 if present). Scores were summed to indicate the extent of community disadvantage, yielding an index that ranged from 0 to 4 ($M = 1.27, SD = 1.38$).

**Protective parenting.** At T1, parents completed a 14-item scale about three aspects of protective parenting practices that have been linked with adolescent adjustment and risk behavior in previous research (Brody et al., 2014; Cleveland et al., 2005). The measure included items about parental monitoring (4 items; e.g., “How often do you know when your teen gets in trouble at school or someplace else away from home?” and “How often do you know who your teen is
with when he/she is away from home?”), nurturant parenting (5 items; e.g., “How often does your teen talk to you about things that bother him/her?” and “How often do you ask your teen what he/she thinks before making decisions that affect him/her?”), and parental academic involvement (5 items; e.g., “How often do you talk about the importance of finishing high school?” and “How often do you discuss going to college with your teen?”). The response set for parental monitoring and nurturant parenting ranged from 1 (never) to 4 (always); the items for parental academic involvement were rated on a scale ranging from 1 (never) to 6 (4 or more times per week). The items were standardized and summed to form the protective parenting scale ($M = .00, SD = 2.18, \alpha = .78$).

*Planful future orientation.* At T1 and T2, youth reported their planful future orientation using the perceived life chances scale and the state hope scale. The perceived life chances scale (Jessor, Donovan, & Costa, 1996) includes 10 items that measure one’s estimation of achieving certain adaptive life goals, on a 5-point scale from 1 (very low) to 5 (really high). Items assessed perceived chances of outcomes such as going to college, having a job that I enjoy doing, having a job that pays well, and having a happy family life. Cronbach’s alpha was .93. The State Hope Scale (Snyder et al., 1996) included 6 items that addressed youth’s beliefs in their capacity to initiate actions and to generate routes for reaching goals. Example items include, “At this time, I am meeting the goals that I have set for myself” and “I can think of many ways to reach my current goals.” The response set ranged from 1 (really false) to 5 (really true); Cronbach’s alpha was .87. The measures were standardized and summed to form the planful future orientation construct ($\alpha = .78$). The mean scores for the planful future orientation variable were .00 ($SD = 1.73$) for T1 and .00 ($SD = 1.74$) for T2.
**Risk behavior.** I assessed youth’s risk behavior at T1 and T3 using self-reports of substance use, delinquent behavior, and sexual risk. A three item scale was used to assess substance use per prior research with African American youth (Brody, Kogan, & Chen, 2012; Brody et al., 2014). Youth indicated the number of times during the past 3 months they had used alcoholic beverages (beer, wine, wine cooler, or other liquor), drunk a lot of alcohol at one time, and used marijuana. The response set ranged from 0 (*none*) to 6 (*more than 30 times*); Cronbach’s alpha was .69. Delinquent behavior was measured using 8 questions from the National Youth Survey (Elliott, Huizinga, & Ageton, 1985). The scale indicated how many times during the past 6 months youth committed acts such as theft, truancy, fighting, and vandalism, and how many times they were suspended from school; Cronbach’s alpha was .69. Youth’s sexual risk behavior was assessed using 3 items for which youth provided specific numbers: “In the past 3 months, how many people have you had sex with?”, “In the past 3 months, how many times have you had sex?”, and “In the past 3 months, out of the times you have had sex, how many times did you have sex while high on alcohol or drugs?” Items were standardized and then summed to produce the sexual risk behavior index. Cronbach’s alpha was .78. Confirmatory factor analysis of the three indicators of risk behavior indicated a single factor with loadings that ranged from .62 for delinquent behavior to .78 for substance use at T1 and T3. I subsequently used the factor score to form a global assessment of youth’s risk behavior.

**Genotyping.** Participants’ DNA was obtained using Oragene™ DNA kits (DNA Genotek, Kanata, Ontario). Parents and youth rinsed their mouths with tap water and then deposited 4 ml of saliva in the Oragene sample vial. The vial was sealed, inverted, and shipped via courier to a central laboratory in Iowa City, Iowa, where samples were prepared according to the manufacturer’s specifications. The genotype at *DRD4* was determined for each participant.
using the primers F-GGCGTTGCGCTCTGAATGC and R-GAGGGACTGAGCTGGACAAACCAC, standard Taq polymerase and buffer, and standard deoxynucleotide triphosphates with the addition of 100 mM 7-deaza GTP and 10% DMSO (Bradley, Dodelzon, Sandhu, & Philibert, 2005). The resulting polymerase chain reaction products were electrophoresed on a 6% nondenaturing polyacrylamide gel, and the products were visualized using silver staining. The genotype was then called by two individuals blind to the study hypotheses and other information about the participants. For tests of the G × E hypotheses, DRD4 status was dummy coded; participants with at least one l allele were assigned a code of 1 (43.8% of parents; 47.4% of target youths), and participants who were homozygous for the s allele were assigned a code of 0 (56.2% of parents; 52.6% of target youths). Using the Hardy-Weinberg equilibrium test, the observed distribution of DRD4 did not differ significantly from that predicted on the basis of simple Mendelian inheritance.

Control variables. Three variables were controlled that might have influenced the relations among study variables. Consistent with our past research (Brody et al., 2014; Kogan et al., 2012), an index of family SES was developed using five dichotomous variables: family poverty based on federal guidelines, caregiver unemployment, receipt of TANF, female-headed households, and caregiver education level less than high school graduation. Each indicator was coded dichotomously (0 if absent, 1 if present), and the scores were summed to form an index that ranged from 0 to 5 (M = 1.83, SD = 1.22). Youth gender (0 = female; 1 = male) was controlled. Finally, in all analyses we controlled for intervention program assignment in the randomized prevention trial. Families were assigned to one of two family-centered, five-session programs. A dichotomous variable was specified (1 = treatment, 0 = attention control).
Analysis plan

The hypotheses for the present study were tested using structural equation modeling (SEM) with complex designs as implemented in Mplus (Muthén & Muthén, 1998-2010). Because respondents were clustered within census tracts, the error terms of regression models were not independent, leading to an underestimation of standard errors. To avoid this problem, I used the complex analysis to adjust parameter standard errors for interdependence in the data. Interaction terms were used to test the G × E pathways. To produce a common scale, standardized regression weights were used in which all study variables were standardized (a mean of 0 and a standard deviation of 1) before the interaction terms were calculated. Benefits of standardized weights in the interaction model include making coefficients easier to interpret, reducing multicollinearity, and making the simple slope easier to test (Dawson & Richter, 2006). I conducted post hoc analyses of significant interaction terms using the Johnson-Neyman (J-N) technique (Hayes & Matthes, 2009). This procedure identifies regions of significance for interactions between continuous (i.e., community disadvantage and protective parenting) and categorical (i.e., genotypes) variables. I tested the conditional indirect effects of community disadvantage on youth’s risk behavior when both parent and youth have DRD4-l using Monte Carlo integration methods (Hayes, 2013; Preacher, Rucker, & Hayes, 2007). This approach can estimate the power of a test for each individual parameter of the model and account for specific data characteristics when a sampling design includes multiple levels (Thoemmes, MacKinnon, & Reiser, 2010).

To assess goodness-of-fit, we used Root Mean Square Error of Approximation (RMSEA; Browne & Cudeck, 1992), comparative fit index (CFI; Bentler, 1990), and chi-square divided by its degrees of freedom (fit ratio). CFI was truncated to range from 0 to 1; values close to 1
indicated a very good fit (Bentler, 1990). An RMSEA smaller than .05 indicates a close fit; an RMSEA between .05 and .08 suggests a reasonable fit (Browne & Cudeck, 1992).

Results

Preliminary analyses

Attrition analyses were conducted to evaluate predictors of non-participation based on project attrition and refusal to provide DNA. The original sample at T1 comprised 502 youth and their parents. At T3, the 464 youth and their parents remaining in the study were asked to provide DNA. Study hypotheses were tested with the 361 youth and their parents who agreed to provide DNA. I investigated differences on all study variables at T1 between retained participants and those who left the study by T3, and between participants at T3 who agreed or refused to provide DNA. No differences were detected. I next checked for evidence of gene-environment correlations, rGE, a non-random distribution of environments among people with different genotypes. Importantly, rGE likely confounds G × E effects (Caspi & Moffitt, 2006). Table 2.1 presents zero-order correlations for the study variables; no associations of parent or youth genotype with the study variables were significant, ruling out potential rGE effects. Finally, we assessed racial admixture using the Structure program version 2.3.4 (Falush, Stephens, & Pritchard, 2007) with a panel of 24 ancestrally informative markers to infer the number of ancestral populations and to estimate an ancestry proportion for each participant. The average proportion of African ancestry among 24 ancestral populations in this sample was 50.2%. Including the racial admixture variable as a covariate did not change any results; we thus report our findings without ancestry controlled.
Multilevel G × E interaction processes predicting changes in risk behavior

Figure 2.2 presents the SEM for hypothesized indirect pathways. In the sections that follow, we first describe the portion of the model representing the interaction of community characteristics and parental genotype on protective parenting. Then, I focus on the interaction effect of protective parenting and youth genotype on a planful future orientation, an outcome proximal to risk behavior. After this, I present the results of indirect effect tests.

Community disadvantage × parent’s DRD4 effects on protective parenting

The results presented in Figure 2.2 indicate that the parameter representing the interaction between community disadvantage and parent’s DRD4 status significantly predicted protective parenting (β = -.19, p < .01). Figure 2.3 (a) depicts the effect of parent’s DRD4 on protective parenting for levels of community disadvantage ranging from -2 to +2 standard deviations from the mean. The slope indexing the effect of community disadvantage on protective parenting for parents carrying DRD4-l was significantly different from zero (b = -.17, p < .05), whereas the slope for parents carrying only DRD4-s was not significantly different from zero (b = .10, p = .18). Thus, the effect of community disadvantage on protective parenting emerged only for parents carrying DRD4-l.

The graph of the interaction depicted in Figure 2.3 (a) demonstrated the crossover pattern that suggests a differential susceptibility effect. The J-N technique (Preacher et al., 2006) was used to assess regions of significant difference attributable to different alleles of DRD4. A differential susceptibility model produces relatively equal regions of significance on both sides of the graph. The shaded areas in Figure 2.3 (a) represent the regions of significant difference for the G × E interaction effect. The J-N technique yields an index that addresses the equality of the regions of significance, called the proportion of the interaction areas (PoI); scores near .50
indicate relatively equal significance on both sides of the interaction. The PoI in this case was .58, which is within the range consistent with differential susceptibility effects (Roisman et al., 2012).

**Protective parenting × youth DRD4 effects on a planful future orientation**

Consistent with our hypotheses, the data presented in Figure 2.2 demonstrated that the interaction term between protective parenting and youth genotype at DRD4 was associated significantly with increases in planful future orientation ($\beta = .15, p < .05$), which, in turn, was associated with youth risk behavior at age 19 ($\beta = -.16, p < .01$). Analyses of the simple slopes in Figure 2.3 (b) indicated that protective parenting enhanced a planful future orientation only for youth with a DRD4-l allele (DRD4-l, $b = .23, p < .01$; DRD4-s, $b = .02, p = .73$). The J-N technique identified a region of significant difference confined to one side of the interaction effect. This indicated that DRD4 conferred vulnerability to low levels of protective parenting when protective parenting was less than -.48 SD below the sample mean; however, high levels of protective parenting for DRD4-l youth did not result in increases in planful future orientation. In this case, the G × E interaction was more consistent with a diathesis-stress model than a differential susceptibility model (PoI = .25).

**Indirect effects linking community disadvantage to youth risk behavior**

Table 2.2 presents the conditional indirect effects linking multilevel G × E processes to youth’s planful future orientation and subsequent changes in youth risk behavior. A significant indirect effect linked community disadvantage to changes in youth’s planful future orientation via protective parenting when both parent and youth carried DRD4-l; $\beta = -.03, 95\%$ CI [-.060, -.008]. A significant indirect effect also linked protective parenting to changes in risk behavior through planful future orientation when youth carried DRD4-l; $\beta = -.03, 95\%$ CI
Finally, the indirect effect of community disadvantage on risk behavior via protective parenting and planful future orientation was significant only when DRD4 status of both parents and youth are considered; $\beta = .01$, 95% CI [.003, .015].

**Discussion**

Drawing on bioecological (Bronfenbrenner & Ceci, 1994) and differential susceptibility (Belsky et al., 2007; Belsky & Pluess, 2009) perspectives, the present study tested a series of multilevel G × E hypotheses predicting African American youth’s risk behavior from ages 16 to 19. Consistent with our hypotheses, the effects of community disadvantage on protective parenting and the effects of protective parenting on youth’s planful future orientation were moderated by parent and youth DRD4 status. Specifically, parents’ exposure to community disadvantage predicted their provision of protective parenting practices only when parents carried DRD4-1. Protective parenting, in turn, predicted youth’s development of a planful future orientation, a proximal predictor of risk behavior, when youth carried DRD4-1. I subsequently tested the hypothesis that the pathway linking community disadvantage to youth risk behavior via protective parenting and planful future orientation would be evident only when both parents’ and youth’s DRD4 status was considered. The indirect effect analysis supported this hypothesis.

Informed by research on differential susceptibility to environmental exposures, I also examined the possibility that parent and youth DRD4 operated to increase environmental sensitivity in a “for better and for worse” manner or conferred vulnerability to negative environments. Results were mixed for parent and youth genotypes. For the interaction effect of community disadvantage and parents’ DRD4 on protective parenting, this association emerged as a differential susceptibility effect. For the effect of protective parenting on increases in youth’s
planful future orientation, however, the evidence was consistent with the diathesis-stress
perspective.

Most research on G × E interaction effects on youth development has focused on a range
of proximal environments interacting with youth genotypes. The current study extended this
literature by examining the ways in which community environments interacted with parental
genotype at $DRD4$ to forecast parenting behavior. I found that exposure to community
disadvantage did not predict parenting behavior among parents who did not carry $DRD4$-$l$. In
contrast, among parents who carried 1 or 2 copies of the $l$ allele, community disadvantage
predicted diminished protective parenting characterized by monitoring, nurturance, and academic
involvement. I am aware of two other studies that have examined parent genotype by
environment interaction effects on parenting behavior. Van IJzendoorn et al. (2008) found that,
among parents of infants, dopamine-related genes moderated the influence of daily hassles on
parenting behavior. Similar to the present study, parents displayed less sensitivity to their
children’s needs when they were stressed by daily hassles, but only in the group of parents who
had the $DRD4$-$l$ and catechol-O-methyltransferase (COMT)-val alleles. A second study also
yielded results consistent with the present study. Beach et al. (2012) found that parents with
$DRD4$-$l$ who were exposed to community stress developed patterns of negative emotionality that
affected their relationships with their children. Taken together, these studies, in which different
sources and methods were used, suggest that $DRD4$ genotype may interact with contextual
circumstances to affect parenting behavior.

Study finding that youth’s $DRD4$ status moderated the influence of protective parenting
on planful future orientation is consistent with a number of studies investigating youth $DRD4$
and family environments. Most of this research, however, has focused on the interaction effects
of youth DRD4 genotypes × family environments on risk behaviors without consideration of intermediate phenotypes such as planful future orientations (Bakermans-Kranenburg & van IJzendoorn, 2006; Bakermans-Kranenburg & van IJzendoorn, 2007; Beach et al., 2010; Propper, Willoughby, Halpern, Carbone, & Cox, 2007). The focus on the intermediate phenotype, planful future orientation, is a response to recent requests for investigations of intermediate mechanisms that account more precisely for G × E interactions. For example, Brody et al. (2012) found the effect of DRD4 × life stress on substance use was mediated by changes in substance use vulnerability cognitions and increases in affiliations with substance-using peers. Findings of this study suggest that a planful future orientation is a plausible intermediate mechanism linking DRD4 × rearing environment to youth risk behavior.

I also sought to determine whether the G × E effects were consistent with the diathesis-stress or the differential susceptibility perspective. The parent genotype × community disadvantage interaction effect on protective parenting was consistent with the differential susceptibility perspective. Protective parenting among parents carrying DRD4-l was undermined in highly disadvantaged community environments but was enhanced in favorable environments. This finding is consistent with Beach et al. (2012) who documented a “for better and for worse” effect for DRD4 × community context in predicting parents’ negative arousal, a proximal determinant of parenting behaviors. Van IJzendoorn et al. (2008) reported that combinations of COMT and DRD4 moderated the influence of daily hassles on maternal sensitivity; however, evidence for differential susceptibility is unclear (Belsky, 2011). Additional research involving parenting behaviors is needed to clarify further the role of DRD4 in conferring differential susceptibility.
The effects of protective parenting on youth’s planful future orientations were moderated by youth DRD4 in a “for worse” manner indicative of a diathesis-stress effect. Youth with the DRD4-l allele who received little protective parenting reported diminished planful future orientations. There was, however, no evidence emerged for a “for better” effect in which heightened protective parenting led to significant enhancement of planful future orientation among youth with the DRD4-l allele. Relatively few studies, however, investigate intermediate phenotypes, and among those we aware of, only two tested for differential sensitivity. Simons et al. (2011) found that a combined assessment of community and family environments interacted with youth’s cumulative gene index to predict youth hostility in a manner consistent with differential susceptibility. Similarly, Belsky and Beaver (2011) found differential susceptibility effects of parenting practices and a cumulative gene index on male adolescents’ self-regulation. Contradictory findings in our study may be the result of prior studies using cumulative gene index rather than a single genotype. However, given the paucity of G × E studies on intermediate phenotypes, additional research is warranted to determine the specific environmental conditions under which various kinds of measures detect differentially sensitive effects for DRD4-l.

To date, little research has considered the joint effects of parent and youth genotypes interacting with environmental factors to forecast youth behavior. Post hoc analyses suggested that community environments affected youth risk behavior indirectly via a series of indirect effects involving protective parenting and youth’s planful future orientations when the DRD4 statuses of both parents and youth are considered. Significant indirect effects were detected across the pathways linking community disadvantage × parental DRD4 status effects on protective parenting to protective parenting × youth DRD4 status effects on planful future
orientation (linking parent G × E processes to youth G × E processes). The indirect effects model, operating through both parental and youth G × E processes, underscores the need to document the pathways through which members of a family system with varying genotypes shape the environment in which others’ development and behavior take place.

Several methodological strengths in the present study are noteworthy. This study used multi-informant and multi-method assessments gathered from the U.S. Census, parents, and youth. Census tract data were used to obtain an objective assessment of community disadvantage. I also used both parent and youth reports and alternated these reports to minimize self-report bias in the links between parenting and planful future orientation. Both planful future orientation and risk behavior were assessed by youth report, producing the potential for self-report bias to inflate the association of these factors. This concern was mitigated somewhat by the evaluation of these variables at different time points and the use of a baseline control. Some limitations of the research also should be noted. Only one genetic polymorphism was examined in this study. A number of genetic variants are under investigation that may alter risk or environmental sensitivity, although \textit{DRD4} is among the best documented and characterized polymorphisms (Bakermans-Kranenburg & van IJzendoorn, 2006; Brody et al., 2012; Propper et al. 2007). It also should be noted that, although we coded \textit{DRD4} alleles as 6 or fewer repeats versus 7 or more repeats, some studies focus on the 4-repeat allele versus the 7-repeat allele (Beach et al., 2010) or on the 7-repeat allele versus all others (Bakermans-Kranenburg & van IJzendoorn, 2006). Also, the findings focused on rural community contexts; thus, the results may not generalize to other settings. These limitations notwithstanding, the present study demonstrates the ways in which community disadvantage, protective parenting, and both parents’ and youth’s \textit{DRD4} status combine to influence youth risk behavior via a planful future
orientation. The results suggest a continued need for studies that integrate transactional developmental perspectives and G × E research. Such research expands the focus of individual differences to include exposure to environmental characteristics in accounting for complex transactions among family members and communities over time.
Table 2.1

Correlations among the research variables (N = 361)

<table>
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<th>Variable</th>
<th>1</th>
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<td>1. Youth gender</td>
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<td>-.19**</td>
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<td>-.09</td>
<td>.07</td>
<td>.06</td>
<td>.42**</td>
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*p < .05. **p < .01.
Table 2.2

*Conditional indirect effects on youth risk behavior in a condition of parent and youth DRD4-I status*

<table>
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<tr>
<th>Predictors</th>
<th>Mediator(s)</th>
<th>Outcomes</th>
<th>Indirect Effect</th>
<th>Effect Size</th>
<th>95% CI</th>
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<td>Protective parenting</td>
<td>Planful future orientation</td>
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<td>.56</td>
<td>[-.060, -.008]</td>
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<td>Planful future orientation</td>
<td>.01</td>
<td>.21</td>
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</tr>
</tbody>
</table>
Figure 2.1. Conceptual model.
Note. $\chi^2 = 54.73$, df = 47, $p = .20$. RMSEA = .03. CFI = .98. $N = 361$. Standardized coefficients are shown. Family SES, target gender, and program intervention effects were controlled. $^*p < .05$. $^**p < .01$.

Figure 2.2. Final model
Figure 2.3. (a) Community disadvantage × parental DRD4 effects on protective parenting; (b) Protective parenting × youth DRD4 effects on a planful future orientation.
CHAPTER 3

GENETIC MODERATION OF TRANSACTIONS BETWEEN PARENTING PRACTICES AND CHILD SELF-REGULATION: A FOCUS ON DOPAMINERGIC GENES ²

² Cho, J., & Kogan, S. M. To be submitted to Child Development.
Abstract

Consistent with a transactional model of human development, empirical evidence indicates that parenting behaviors influence the development of child self-regulation, which in turn is prospectively related to changes in parenting behaviors. Recent research on gene by environment interaction (G × E) also highlights the possibility that both parents and children are differentially affected by their family experiences. Although most G × E research has focused on the interaction effect of parenting and child genotypes on child developmental outcomes such as externalizing behavior, no research has examined how children comprise an environmental influence on parenting behavior within a G × E framework. The present study investigated how parent and child genotypes jointly modulate the transactional relations between parenting practices and child self-regulation. African American children (N = 291) and their parents provided longitudinal data spanning child ages 11 to 15 years and a saliva sample from which dopaminergic genes were genotyped. A cumulative genetic index which aggregated three dopamine-related genes was developed. Genetic status on this index was hypothesized to confer sensitivity to both positive and negative environmental inputs. Results indicated that responsive-supportive parenting significantly affected child self-regulation among children depending on their genetic status. Also, child self-regulation interacted with a parental dopaminergic gene index to predict changes in parenting practices. In both cases, genetic status conformed to a differential susceptibility model in that dopaminergic genes amplified the effect of environmental inputs on parent and youth outcomes.

Keywords: gene by environment interaction, transactional relations, parenting, self-regulation, dopaminergic genes
Introduction

The transactional model of child development underscores the bidirectional nature of human development, which is a product of ongoing interactions between an individual and the environment (Sameroff, 2009; Sameroff & Chandler, 1975). This perspective frequently has been used to understand bidirectional influences between parenting practices and child development. Based on this perspective, researchers have considered models in which children influence parents just as parents influence children within the family system (Pettit & Arsiwalla, 2008).

Consistent with a transactional perspective, empirical evidence indicates that parenting behaviors influence the development of child self-regulation, which in turn is prospectively related to changes in parenting behaviors (Brody & Ge, 2001; Moilanen, Rasmussen, & Padilla-Walker, 2014; Yates et al., 2010). Children who experience warm, supportive and sensitive parenting prove better able to attend to and concentrate on tasks, regulate their emotions under challenging circumstances and engage in goal-directed behavior than those who experience unresponsive, hostile, and neglectful parenting practices (Eisenberg et al., 2005; Maccoby, 2000; Rothbaum & Weisz, 1994). Conversely, children with poor self-regulation elicit harsher, less optimal parenting than do children with high self-regulation (Moilanen et al., 2014; Scaramella & Leve, 2004).

Emerging research suggests that environmental influences may be modulated by parent and child genotype. Ostensibly this would include not only the parent as an environmental influence on the child, but the child as an environmental influence on the parent. Recent research on gene and environment interaction (G × E) highlights the possibility that both parents and children are differentially affected by their family experiences (see Belsky & Pluess, 2009;
van IJzendoorn, Bakermans-Kranenburg, & Masman, 2008). Although most G × E research has focused on the interaction effect of parenting and child genotypes on child developmental outcomes such as externalizing behavior (see Bakermans-Kranenburg & van IJzendoorn, 2011), no research has examined how children comprise an environmental influence on parenting behavior within a G × E framework. Moreover, no study, to date, has examined the joint effects of parent and child genotypes on bidirectional influences within a family system.

To address this research gap, the present study extends the research on transactional relationships between parenting practices and child self-regulation by examining genetic moderation processes considering both parent and child genotype. The present study investigated the interaction effect of parenting practices and child genotype on the development of children’s self-regulation, which, in turn, would interact with parent genotype to predict changes in parenting behavior.

**Literature Review**

**Bidirectional Associations between Parenting Practices and Self-Regulation**

Self-regulation is the ability of an individual to actively or passively monitor, evaluate, modify, and inhibit one’s emotions or behavior to achieve one’s personal goals in agreement with societal standards of behavior (Moilanen, Shaw, & Fitzpatrick, 2010). These skills are vital for the prevention of maladaptive developmental outcomes in adolescence, including sexual risk-taking, delinquency, and substance use (Brody & Ge, 2001; Luyckx et al., 2011; Quinn & Fromme, 2010; Tangney, Baumeister, & Boone, 2004), and the promotion of positive outcomes, such as prosocial behavior and academic achievement (Bowers et al., 2011; Moilanen, 2007). Studies with children ranging in age from middle childhood to adolescence have shown self-
regulation to be negatively related to sexual risk behavior and substance use (Brody & Ge, 2001; Brody et al., 2005; Wills et al., 2000, 2003).

Research documents the influence of parental socialization on child self-regulation as parents explicitly and implicitly communicate to children what is and is not appropriate behavior through behavioral modeling, parenting practices, and the family’s emotional climate (Moilanen et al., 2014). Parental warmth/responsivity increases the likelihood that children will attend to parents’ guidance for developing strategies for self-regulation (Finkenauer, Engels, & Baumeister, 2005; Olson, Bates, & Bayles, 1990), and parental support/involvement significantly influences self-regulation among children from ages 11 to 16 years (Brody & Ge, 2001; Brody et al., 2005).

Although most research on parenting and self-regulation has focused on parenting effects on improvements in children’s self-regulation, empirical evidence documents mutual influence processes between parents and children (Bell, 1968; Grusec & Goodnow, 1994; Moilanen et al., 2014; Padilla-Walker, Carlo, Christensen, & Yorgason, 2012). For example, Brody and Ge (2001) found that children’s high levels of behavioral self-regulation at ages 11 to 12 years predicted decreases in conflicted-harsh parenting over a 1 year period. Other research documenting associations between self-regulation and parenting reported that teen’s self-regulation influenced authoritarian and permissive-indulgent parenting styles (Moilanen et al., 2014). The current study examined bidirectional associations between parenting and child self-regulation during the transition from childhood to early adolescence.

**G × E Interactions of Parents and Children**

G × E studies indicate that variation in specific genotypes interact with environmental factors to shape individuals’ development and behavior. These studies most commonly identify
specific genotypes that confer vulnerability to environmental risk factors, which increases the likelihood of negative behavioral and psychological outcomes. Emerging research, however, suggests that this diathesis-stress perspective may be limited. Some genotypes may act to amplify an individual’s sensitivity to both positive and negative environmental influences, a dynamic called differential susceptibility (Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007; Belsky & Pluess, 2009).

Studies have identified G × E interaction effects in the development of self-regulation as well as parenting practices. For example, Belsky and Beaver (2011) reported parenting behaviors interacted with youth genotypes to predict their self-regulation. The result also supported the differential susceptibility perspective in that adolescents carrying susceptibility genotypes showed more or less self-regulation under, respectively, supportive and unsupportive parenting conditions. Other studies document G × E influences on parenting behaviors. For example, van IJzendoorn et al. (2008) found that, among parents of infants, dopamine-related genes moderated the influence of daily hassles on parenting behavior. Consistent with the differential susceptibility perspective, parents with specific dopaminergic genotypes displayed less sensitivity to their children’s needs when they were stressed by daily hassles and greater sensitivity to their children when stress was not present (van IJzendoorn et al., 2008). No studies, however, have examined how parental genotypes might affect the extent to which parents are influenced by their children’s behavior. Furthermore, no studies of which we are aware have examined how parent and child genotypes jointly moderate the transactional relations between parenting practices and child self-regulation. The present research addresses this gap.

The present study investigated the effects of genotypic variation in parents and children. I focused on candidate genes in the dopaminergic system including dopamine D4 receptor
(DRD4), dopamine D2 receptor (DRD2), and ankyrin repeat and kinase domain containing 1 (ANKK1). These dopaminergic genes, which are engaged in attentional, motivational, and reward mechanisms, have been studied extensively in relation to children’s externalizing behavior, substance use, and nicotine dependence (see Bakermans-Kranenburg & van IJzendoorn, 2011; Belsky & Pluess, 2009). Also, studies indicate that dopaminergic genes interact with environmental input in modulating parents’ sensitive responses to children (Beach et al., 2012; van IJzendoorn et al., 2008).

To explain these relations between dopaminergic genes and individuals’ behaviors, Spear (2000) proposed a dopamine insensitivity model. This perspective indicates that reward signals initiate a phasic burst of midbrain dopamine neurons, which elicit positive emotional states and organize the learning of cues that predict future rewards (Spear, 2000). Individuals who experience a reduced incentive value from stimuli are more likely than those who do not to pursue new appetitive reinforcers. In contrast, individuals who are particularly sensitive to rewards need less of any particular stimulus to initiate a response. This perspective has increased focus on the potential of factors associated with a blunted dopamine response to predict sensation-seeking and low self-regulative behaviors (Spear, 2011). The current study, therefore, adopted multiple dopaminergic genes to investigate genetic moderation effects on the association between parenting behavior and self-regulation.

DRD4 is a variable nucleotide tandem repeat (VNTR) polymorphism composed of 16 amino acid (48-bp) repeat polymorphisms that range from 2 to 11 repeats. Studies characterize DRD4 alleles as either “short” (s) or “long” (l), with the s category defined as having 6 or fewer repeats and the l category as having 7 or more repeats (Brody et al., 2012; Eisenberg et al., 2007; Laucht, Becker, Blomeyer, & Schmidt, 2007; McGeeary, 2009). The long allele is hypothesized
to confer differential sensitivity to environmental input (Belsky & Pluess, 2009). A single nucleotide polymorphism in the DRD2 gene (rs6275) is characterized by a C to T substitution located in a noncoding region of the DRD2 locus. The A1 allele of this locus has been associated with low dopamine density and lower mean relative glucose metabolic rate in dopaminergic regions in the human brain (Noble, Gottschalk, Fallon, Ritchie, & Wu, 1997), high novelty seeking (Suhara et al., 2001), and a number of substance use disorders, particularly alcoholism (Bowirrat & Oscar-Berman, 2005). Finally, ANKK1, a neighboring gene of DRD2, contains a single serine/threonine protein kinase domain, and the C allele of the rs1711539 locus is thought to confer environmental susceptibility (Dick et al., 2007; Munafo, Johnstone, Welsh, & Walton, 2005; Neville, Johnstone, & Walton, 2003).

Recent research suggests that multiple genes may exert a shared polygenic influence (i.e., cumulative genetic influence) on individual behaviors. For example, Belsky and Beaver (2011) created composite measures by summing plasticity alleles of diverse genes and found a significant cumulative influence of variants of multiple genes on the association between parenting practices and child self-regulation. Consistent with the recent research on cumulative genetic plasticity (Belsky & Beaver, 2011; Masarik et al., 2014; Wickrama & O’Neal, 2013), I expect that the plasticity alleles of the specified dopaminergic genes will influence individual behaviors cumulatively.

The Present Study

To date, most G × E research has focused on the way in which parenting practices interact with child genotype to predict children’s behavior. Although children’s behaviors, particularly those related to self-regulation, comprise an environmental influence on parents and their parenting behaviors, no studies to my knowledge have considered the interaction of parental
genotype with child behavior to predict parenting behavior. Also, to my knowledge, there are no studies that investigate how parent and child genotypes jointly modulate the transactional relations between parenting practices and child self-regulation. The present study extends the research on transactional relations between parents and children by examining genetic moderation processes of both parent and child. The hypotheses were summarized in Figure 3.1.

Responsive-supportive parenting was hypothesized to interact with children’s dopaminergic genes to predict increases in child self-regulation. Child self-regulation, in turn, was hypothesized to interact with parental genotype to forecast changes in parenting behavior from child ages 11 to 15 years. I also expected that each G × E interaction effect would support a differential susceptibility rather than a diathesis-stress model.

**Methods**

**Participants**

Study hypotheses were tested with 291 African American children (131 boys and 160 girls) and their primary caregivers—typically the biological mother (93.4%), who resided in eight rural counties in Georgia. Families were recruited randomly from lists that public schools provided. Data were collected within the context of a family-based prevention study at intervals timed to evaluate the prevention program. Because the present study hypotheses were not focused on intervention efficacy, random assignment to the intervention program or to an attention control program will be controlled. Data were obtained at four time points spanning 4 years. Because of the short time interval between the first two time points, data from these two time points was combined; I report on the combined data using mean values and refer it to as Time 1. Children’s mean age were 11.51 years at Time 1 (T1; $SD = .51$), 13.46 years at Time 2 (T2; $SD = .49$), and 15.51 at Time 3 (T3; $SD = .50$).
At T1, 36.6% of the mothers were married and living with their husbands, 2.3% were married but separated, 7.1% were cohabiting with a significant other, 20.1% were in a significant relationship but not cohabiting, and 32.7% were not in a significant relationship. Mean family income was $2,037 ($D = $1,480) per month. Although 74% of the mothers were employed outside the home and worked an average of 39.7 hours per week, 41% of the families lived below federal poverty standards and another 26% lived within 150% of the poverty threshold; they could be described as working poor (Boatright & Bachtel, 1999).

Procedures

Families were contacted and enrolled in the study by African American community liaisons who resided in the counties where the participants lived. The community liaisons were selected on the basis of their social contacts and standing in the community; they worked with the researchers on participant recruitment and retention. The liaisons sent letters to the families and followed up with phone calls to the primary caregivers, during which the community liaisons answered any questions that the caregivers raised. Families who were willing to participate in the project were told that a research staff member would contact them to schedule the administration of the assessment in the families’ homes. Parents gave written consent to their own and their children’s participation, and children gave written assent to their own participation. At the visits, questionnaires were administered in interview format by African American home visitors to the primary caregiver and target child via computer-assisted technology on laptop computers. Each family was paid $100 after each assessment.

Measures

Responsive-supportive parenting. At T1 and T3, parents self-reported their responsive-supportive parenting on a measure developed by Brody and colleagues (Brody et al., 2001, 2005)
that included items assessing child monitoring, child management, and harsh-inconsistent parenting. Five items concerned child monitoring (e.g., “How often do you know where your child is when he or she is away from home?”) with a five-point Likert scale ranging from 1 (Never) to 5 (Always). Ten items assessed general child management (e.g., “I tell my child the specific household rules we have in our family,” and “I give rewards when my child tries extra hard to do what he/she is supposed to do.”). The response set ranged from 0 (Not true) to 2 (Very true or often true). Harsh-inconsistent parenting was assessed by four items (e.g., “How often does your mother spank you when you do something wrong?” and “When you do something wrong, how often does your mother blow up at you?”) with a four-point response set ranging from 1 (Never) to 4 (Always). The harsh-inconsistent parenting items were reverse scored. Total 19 items were standardized and summed to form the responsive-supportive parenting scale. The mean scores for the responsive-supportive parenting variable were .00 (SD = 13.22) for T1 and .00 (SD = 9.37) for T3. Cronbach’s alphas for the scale were .80 at T1 and .84 at T3.

Child self-regulation. At T1 and T2, teachers assessed children’s self-regulation using the children’s Self-Control Scale (Humphrey, 1982). The measure included a total of 12 items rated on a five-point response set ranging from 0 (never) to 4 (almost always). Examples of items included “thinks ahead of time about the consequences of his or her actions,” “plans ahead of time before acting,” and “has trouble keeping promises to improve his/her behavior (reverse scored).” The mean scores of self-regulation were 2.43 (SD = .77) for T1 and 2.50 (SD = .89) for T2. Cronbach’s alphas for children’s self-regulation were .89 at T1 and .85 at T2.

Genotyping. Participants’ DNA was obtained using Oragene™ DNA kits (DNA Genotek, Kanata, Ontario). Parents and children rinsed their mouths with tap water and then
deposited 4 ml of saliva in the Oragene sample vial. The vial was sealed, inverted, and shipped via courier to a central laboratory in Iowa City, Iowa, where samples were prepared according to the manufacturer’s specifications. Genotyping was performed for genes in the dopaminergic systems — \textit{DRD4} 48 base pair VNTR; \textit{DRD2} (rs6275); and \textit{ANKK1} (rs1711539). Genotype distributions for \textit{DRD4}, \textit{DRD2}, and \textit{ANKK1} were summarized in Table 3.1. Each genetic polymorphism was coded dichotomously (1 = at least one plasticity allele; 0 = no copies of the plasticity allele). A sum score was computed with values ranging from 0 to 3. Higher scores indicated the presence of more genetic plasticity alleles. The mean scores of cumulative gene index for parents and children were 1.92 ($SD = .81$) and 2.02 ($SD = .78$), respectively.

\textit{Control variables.} Three variables were controlled that could influence the relations among study variables. Consistent with previous studies (Brody et al., 2014; Kogan et al., 2012), a cumulative index of family SES was developed using five dichotomous variables: family poverty based on federal guidelines, caregiver unemployment, receipt of Temporary Assistance for Needy Families (TANF), female-headed households, and caregiver education level less than high school graduation. Each indicator was coded dichotomously (0 if absent, 1 if present), and the scores were summed to form an index that ranged from 0 to 5 ($M = 1.64$, $SD = 1.18$). Children’s gender (0 = girls; 1 = boys) was controlled. Finally, the intervention program assignment in the randomized prevention trial was controlled in all analyses. Families were assigned to one of two family centered intervention programs. A dichotomous variable was specified (1 = treatment; 0 = control).

\textbf{Analysis plan}

The hypotheses for the present study were tested using structural equation modeling (SEM) as implemented in Mplus (Muthén & Muthén, 1998-2010). To test the multiple $G \times E$
interaction processes, we used the moderated mediation model, which combines traditional moderation and mediation models (Preacher, Rucker, & Hayes, 2007). The mediating effect of child self-regulation on changes in parenting practices was tested including two genetic moderation processes of parents and children. The present study examined the interaction effect of parenting and child genes on child self-regulation, which in turn would interact with parent genes to predict changes in parenting practices across child ages between 11 and 15 years. To produce a common scale, standardized regression weights were used in which all study variables were standardized (a mean of 0 and a standard deviation of 1) before the interaction terms were calculated. Benefits of standardized weights in the interaction model include making coefficients easier to interpret, reducing multicollinearity, and making the simple slope easier to test (Dawson & Richter, 2006).

To investigate the G × E interactions in more detail, I examined separate slopes of each G × E interaction effect across the range of values (0 – 3) for the cumulative gene index. The online tool was used to estimate the magnitude of the simple slopes between predictors and outcomes of G × E interaction effects (Preacher, Curran, & Bauer, 2006). Then, I conducted post hoc analyses of significant interaction terms using the Johnson-Neyman (J-N) technique (Hayes & Matthes, 2009). This procedure identified regions of significance for interactions between continuous (i.e., responsive-supportive parenting and child self-regulation) and categorical (i.e., genotypes) variables. The J-N technique revealed whether each G × E interaction effect would support the differential susceptibility model or the diathesis-stress model.
Results

Preliminary analyses

Attrition analyses were conducted to evaluate predictors of non-participation based on project attrition and refusal to provide DNA. The original sample at T1 comprised 517 children and their parents. At T3, the 450 children and their parents remaining in the study were asked to provide DNA. Study hypotheses were tested with the 291 children and their parents who agreed to provide DNA and had valid information on all three genotypes. I investigated differences on all study variables at T1 between retained participants and those who left the study by T3, and between participants at T3 who agreed or refused to provide DNA. No differences were detected. We next checked for evidence of gene-environment correlation (rGE), a non-random distribution of environments among people with different genotypes. Importantly, rGE likely confounds G × E effects (Caspi & Moffitt, 2006). Table 3.2 presents zero-order correlations for the study variables; no significant associations of parent or child genotype with the study variables were identified, ruling out potential rGE effects. Finally, I assessed racial admixture using the Structure program version 2.3.4 (Falush, Stephens, & Pritchard, 2007) with a panel of 24 ancestrally informative markers to infer the number of ancestral populations and to estimate an ancestry proportion for each participant. Including the racial admixture variable as a covariate in tests of study hypotheses did not change any results; we thus report our findings without ancestry controlled.

Tests of G × E hypotheses

Figure 3.2 presents the SEM for the hypothesized transactional relations between responsive-supportive parenting and child self-regulation. In the sections that follow, I first describe the portion of the model representing the interaction effect of responsive-supportive
parenting and children’s gene index on changes in their self-regulation. Then, I focus on the interaction effect of self-regulation and the parental gene index on changes in parenting practices. Each G × E process was delineated through post hoc analyses examining whether these genetic moderation processes support differential susceptibility or diathesis-stress model.

**Responsive-supportive parenting × child dopaminergic genes effects on self-regulation**

The results presented in Figure 3.2 indicate that the parameter representing the interaction between responsive-supportive parenting and child’s dopaminergic gene index significantly predicted increases in child self-regulation ($\beta = .14, p < .01$). I identified the magnitude of the simple slopes between parenting practices and self-regulation across the range of the cumulative gene index. Unstandardized estimates of each simple slope were -.20 ($SE = .16, p = .21$), -.01 ($SE = .09, p = .86$), .17 ($SE = .06, p = .00$), and .34 ($SE = .10, p = .00$) for children’s cumulative gene index scores ranged 0, 1, 2, and 3, respectively.

Then, the J-N technique was conducted to test differential susceptibility hypothesis by assessing regions of significant difference between groups who had 0 or 1 versus 2 or 3 plasticity alleles. Figure 3.3 (a) depicts the effect of child’s gene index on self-regulation for levels of responsive-supportive parenting ranging from -2 to +2 standard deviations from the mean. The slope indexing the effect of responsive-supportive parenting on self-regulation for children carrying 2 or 3 plasticity alleles of dopaminergic genes was significantly different from zero ($b = .36, p < .01$), whereas the slope for children carrying 0 or 1 plasticity allele was not significantly different from zero ($b = .07, p = .27$). Thus, the effect of parenting practices on child self-regulation emerged only for children with relatively more plasticity alleles.

The graph of the interaction depicted in Figure 3.3 (a) demonstrated the crossover pattern that suggests a differential susceptibility effect. A differential susceptibility model produces
relatively equal regions of significance on both sides of the graph. The shaded areas in Figure 3.3 (a) represent the regions of significant difference for the G × E interaction effect. The J-N technique yields an index that addresses the equality of the regions of significance, called the proportion of the interaction areas (PoI); scores between .40 and .60 indicate relatively equal significance on both sides of the interaction. The PoI in this case was .44, which is within the range consistent with differential susceptibility effects (Roisman et al., 2012).

**Child self-regulation × parent dopaminergic genes effects on parenting practices**

Consistent with our hypotheses, the data presented in Figure 3.2 demonstrates that the interaction term between child self-regulation and parental gene index was associated significantly with increases in responsive-supportive parenting ($\beta = .12, p < .05$). Analyses of the simple slopes indicated the magnitude of the simple slopes between self-regulation and parenting practices across the range of parental cumulative gene index. Unstandardized estimates of each simple slope were -.17 ($SE = .13, p = .20$), -.02 ($SE = .07, p = .76$), .13 ($SE = .05, p = .02$), and .26 ($SE = .09, p = .00$) for parents’ cumulative gene index scores ranged 0, 1, 2, and 3, respectively.

Then, I conducted the J-N technique to assess regions of significant difference between groups who had 0 or 1 versus 2 or 3 plasticity alleles in Figure 3.3 (b). Self-regulation enhanced responsive-supportive parenting only for parents with 2 or 3 plasticity alleles ($b = .28, p < .01$), whereas self-regulation did not significantly influence parenting practices for parents carrying 0 or 1 plasticity allele ($b = -.09, p = .43$). The J-N technique identified a region of significant difference on both sides of the interaction effect when self-regulation was more than .85 SD as well as less than -.73 SD from the sample mean. The G × E interaction was more consistent with a differential susceptibility model than a diathesis-stress model (PoI = .43).
Discussion

Drawing on transactional (Sameroff & Chandler, 1975) and differential susceptibility (Belsky et al., 2007) models, the present study examined genetic moderation effects on the transactional relations between parenting behavior and child self-regulation. Consistent with the hypotheses, bidirectional influences between responsive-supportive parenting and child self-regulation were moderated by both parent and child genotypes. Specifically, I examined the moderating influence of a cumulative genetic plasticity index which aggregated the plasticity alleles of three dopamine-related genes. The influence of responsive-supportive parenting on increases in child self-regulation was moderated by children’s score on the genetic plasticity index. Child self-regulation, in turn, interacted with parental plasticity index to affect changes in parenting practices. The current study subsequently tested whether each G × E effect was consistent with a differential susceptibility model. Both G × E effects were consistent with a differential susceptibility model in that parents’ and children’s genetic plasticity indices operated to increase environmental sensitivity in a “for better and for worse” manner rather than solely conferring vulnerability to negative environments.

Research based on the transactional perspective underscores the bidirectional influence between parenting practices and child self-regulation (Brody & Ge, 2001; Moilanen et al., 2014; Yates et al., 2010). The present study extended this literature by examining the ways in which parent and child genotypes modulate this bidirectional influence process. Accumulating evidence exists for the differential influence of parenting practices on child outcomes based on child genotypes (see Bakermans-Kranenburg & van IJzendoorn, 2011; Belsky & Beaver, 2011). However, the influence of children on parenting behavior as a function of parental genetic status has not been investigated.
Consistent with my hypotheses, when children carried relatively higher number of plasticity alleles in the dopaminergic system, their self-regulation was significantly influenced by responsive-supportive parenting. This G × E effect conformed to a differential susceptibility model: the more plasticity alleles children carried, the more and less self-regulation they manifested under, respectively, supportive and unsupportive parenting conditions. This result was consistent with previous G × E studies testing interaction effects of parenting behaviors and child genotypes on child developmental outcomes (see Bakermans-Kranenburg & van IJzendoorn, 2011; Belsky & Beaver, 2011). For example, using a national dataset and cumulative gene index, Belsky and Beaver (2011) found that adolescents carrying more plasticity alleles were more positively and negatively influenced by, respectively, supportive and unsupportive parenting in terms of their self-regulation.

In addition to the influence of parents on children, the present study also examined the extent to which child effects on parenting practices were moderated by parents’ susceptibility genes. In this study, child self-regulation interacted with a parental dopaminergic gene index to predict changes in parenting practices. When parents carried higher numbers of the specified plasticity alleles, they showed more supportive and unsupportive parenting under high and low levels of child self-regulation, respectively. This result conformed to a differential susceptibility model rather than a diathesis-stress model. Relatively few studies have examined G × E effects on parenting behavior. Extant studies, however, have documented differential susceptibility effects similar to those found in the present study. For example, van IJzendoorn, Bakermans-Kranenburg, and Mesman (2008) found evidence for dopamine-related genes (catechol-o-methyltransferase [COMT] and DRD4) moderating the effect of daily hassles on parenting. Parents with more plasticity alleles of dopamine-related genes proved to be less responsive to
their children when confronted with more than average daily hassles. In the case of fewer than average daily hassles, however, they showed higher levels of responsive parenting. Beach et al. (2012) also documented a differential susceptibility effect for parental $DRD4 \times$ community context in predicting parents’ negative arousal, a proximal determinant of parenting behaviors. Finally, in Study 1 of this dissertation, parents’ $DRD4$ interacted with levels of community disadvantage to predict their protective parenting in a manner consistent with differential susceptibility. These three studies in conjunction with findings of the current study provide consistent support for the differential susceptibility model as well as underscoring the importance of dopaminergic genotypes in understanding parenting behavior.

The present study builds on these studies of parenting behavior by extending the range of environmental inputs beyond various contextual stressors in parents’ lives. The experience of interacting with more or less regulated children is likely a powerful, proximal influence on parenting behavior. Highly regulated children tend to be easier to manage, and studies reveal that parents with highly regulated children not only report less difficulty in parenting but have a greater sense of efficacy and psychological well-being as parents (Brody & Ge, 2001; Moilanen et al., 2014; Pardini, file, & Burke, 2008; Yates et al., 2010). Brody (2004) referred to this process as a “basking” effect: basking is a phenomenon in which one's psychological well-being increases because of positive characteristics and behaviors of persons to whom one is close. The current study suggests that the extent to which parents are affected by well-behaved children may depend on genetic susceptibility to the environment conferred by genes in the dopamine system.

Study findings confirm the importance of dopaminergic genes in understanding differential susceptibility effects. Although evidence for the precise mechanisms of these environmental amplification effects is sparse, a plausible hypothesis has been advanced (Kogan
et al., 2014). The dopaminergic system is engaged in attentional, motivational, and reward mechanisms, and lower dopaminergic signaling impedes learning based on negative feedback; this in turn may lead to a relative insensitivity to environmental cues and rewarding stimuli (Spear, 2011). This results in a strong preference for immediate reinforcement from the environment and elevated reactivity to high-intensity environmental cues. High-intensity environments that clearly reinforce both negative and positive behaviors exert a heightened influence on behavioral development among reward-deficient children and parents. Rather than withdrawing from the environment or carefully processing environmental cues, such individuals are likely to be hyper-reactive to the environment and reflect the positive or negative input from their immediate surroundings (Bakermans-Kranenburg & van IJzendoorn, 2011).

The current study also underscores the importance of considering multiple plasticity alleles. In Study 1 of this dissertation, I used a well-established candidate gene (DRD4) and documented a susceptibility effect in the case of parenting behavior but not in predicting children’s behavior. In the present study, a composite score based on multiple plasticity alleles of dopamine-related genes conferred susceptibility to the environment for both children and parents. Findings of this study using a composite measure of cumulative genetic plasticity support the contention that the more plasticity alleles an individual carries, the more susceptible he or she will prove to be to environmental influences (Belsky & Beaver, 2011; Belsky & Pluess, 2009) and may explain contradictory findings between Study 1 and the present study.

Some strengths and limitations of the research should be noted. The present study examined the joint effects of parent and child genetic susceptibility on transactional relations between parent and child within a family system. To test this model, multi-informant assessments were used. Parenting practices were assessed by parent-report, and child self-
regulation assessed by teacher-report. I alternated these reports at different time points and used a baseline control to examine transactional relations between parenting practices and child self-regulation. This research design mitigated the potential for self-report bias to inflate the association of parenting and self-regulation.

Limitations are also apparent. The findings focused on a sample of African American families. Thus, the results may not generalize to other racial ethnic groups. Also, the present study focused on genes related to the dopaminergic system. Although there is considerable support for these genes’ functioning as plasticity alleles (see Bakermans-Kranenburg et al., 2011; van IJzendoorn et al., 2008), there is evidence of plasticity alleles in several neurotransmitter systems (Belsky & Beaver, 2011; Simons et al., 2011; Wickrama, O’Neal, & Oshri, 2014). Future studies comparing the results of single versus multiple systems indices are needed. These limitations notwithstanding, the present study expands the understanding of the transactional relations between parenting practices and child development, documenting genetic moderation processes of both parents and children in the family system.
Table 3.1

*Descriptive for genotypes (N = 291)*

<table>
<thead>
<tr>
<th>Genotype</th>
<th>Children</th>
<th></th>
<th>Parents</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>no plasticity allele</td>
<td>1 or 2 plasticity alleles</td>
<td></td>
</tr>
<tr>
<td></td>
<td>n (%)</td>
<td>n (%)</td>
<td></td>
</tr>
<tr>
<td>DRD4</td>
<td>156 (53.6)</td>
<td>135 (46.4)</td>
<td>160 (55.0)</td>
</tr>
<tr>
<td>DRD2</td>
<td>117 (40.2)</td>
<td>172 (59.1)</td>
<td>125 (43.0)</td>
</tr>
<tr>
<td>ANKK1</td>
<td>97 (33.3)</td>
<td>194 (66.7)</td>
<td>113 (38.8)</td>
</tr>
</tbody>
</table>
### Table 3.2

*Correlations among the research variables (N = 291)*

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Child gender</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Family SES</td>
<td>-.03</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Intervention</td>
<td>.03</td>
<td>.01</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Self-regulation T1</td>
<td>.17**</td>
<td>-.11*</td>
<td>.10</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Self-regulation T2</td>
<td>.18**</td>
<td>-.05</td>
<td>.05</td>
<td>.46**</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Supportive parenting T1</td>
<td>-.03</td>
<td>-.10</td>
<td>.10</td>
<td>.10</td>
<td>.15*</td>
<td>-</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Supportive parenting T3</td>
<td>.11</td>
<td>-.04</td>
<td>.14*</td>
<td>.10</td>
<td>.19**</td>
<td>.45**</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>8. Gene Index-Child</td>
<td>-.03</td>
<td>-.07</td>
<td>-.08</td>
<td>.07</td>
<td>.02</td>
<td>-.07</td>
<td>-.10</td>
<td>-</td>
</tr>
<tr>
<td>9. Gene Index-Parent</td>
<td>.02</td>
<td>-.07</td>
<td>.06</td>
<td>.07</td>
<td>.06</td>
<td>.08</td>
<td>.08</td>
<td>.38**</td>
</tr>
</tbody>
</table>

*p < .05; **p < .01.*
Figure 3.1. Conceptual model
\[ \chi^2 = 23.36, \text{df} = 15, p = .08. \text{ RMSEA} = .03, \text{CFI} = .96. \] Standardized coefficients are shown. Child self-regulation (T1), child gender, family SES, and program intervention effects were controlled.

\[ * p < .05, \quad ** p < .01. \]

*Figure 3.2. Final model*
Figure 3.3. (a) Responsive-supportive parenting × child gene index effects on self-regulation; (b) Self-regulation × parent gene index effects on responsive-supportive parenting.
CHAPTER 4
DISCUSSION AND CONCLUSION

Core to the importance of Bronfenbrenner’s original formulation of ecological systems theory was the potential for individual-level biological factors to interact with a broad array of environmental factors to affect human development (Bronfenbrenner & Ceci, 1994). Informed by the bioecological perspective (Bronfenbrenner & Ceci, 1994), this dissertation investigated gene by environment interaction (G × E) processes across multiple-level contexts. Based on G × E research from this framework, individuals’ genetic status was hypothesized to affect the extent to which environmental variability would influence developmental outcomes.

The studies in this dissertation addressed key limitations in the G × E literature. Whereas most G × E studies have focused on the interaction of a singular aspect of the rearing environment and child genotype to predict developmental outcomes, I examined G × E processes across multiple environmental levels including community and family contexts. Also, little research to date has focused on the potential genetic moderation of transactional relations between parenting and child behavior. Similar to parenting practice as an environment of children’s development, child behavior is also a potential environment that interacts with parent genotype (Mills-Koonce et al., 2007). Therefore, the dissertation studies investigated multiple G × E effects of parents and children on their transactional relations within a family system.

Summary of results

Study 1 extended G × E research on youth risk behavior by linking multilevel contextual factors, such as community disadvantage and protective parenting practices, to both parental and
youth dopamine D4 receptor (DRD4) genotypes. Using longitudinal data from 361 rural African American youth and their parents across youth ages 16 to 19 years, Study 1 found that community disadvantage interacted with parental DRD4 status to influence protective parenting. Protective parenting, in turn, interacted with youth DRD4 status to predict youth’s planful future orientation, a proximal influence on changes in risk behavior. The parental DRD4 × community disadvantage interaction, but not youth DRD4 × protective parenting, conformed to a differential susceptibility model. Indirect effect analyses revealed a significant indirect path linking community disadvantage to youth risk behavior through a series of multilevel G × E processes.

Study 2 extended the research on transactional relations between parenting practices and child self-regulation by examining genetic modulation of a parent-child mutual influence process. Using a sample of 291 African American children and their parents, this study investigated the moderating influence of a cumulative genetic plasticity index which aggregated the plasticity alleles of three dopamine-related genes. Results indicated that responsive-supportive parenting significantly affected child self-regulation among children with more plasticity alleles. Also, child self-regulation interacted with a parental dopaminergic gene index to predict changes in parenting practices. Both G × E effects conformed to a differential susceptibility model.

General discussion

Study 1 and 2 examined multiple G × E processes that affect both parent and child within a family. Considering both parental and youth genotypes, Study 1 investigated the external community influences on parenting behavior, which, in turn, affected youth developmental outcomes. The results supported the significant indirect path linking community disadvantage to youth risk behavior through parenting practices. However, the transactional relations between parent and child were not considered in this study. To address this limitation, Study 2 integrated
the transactional perspective and G × E interactions. This study examined mutual influences between parenting practices and child self-regulation by considering both parental and child’s genotypes. Through these two studies, the complexity inherent in the bioecological perspective was examined and well-documented.

Both two studies of this dissertation focused on dopaminergic genes. Study 1 focused on *DRD4* whereas study 2 examined *DRD4, DRD2*, and *ANKK1*. These genes were selected based on accumulating research that documents interaction effects between dopaminergic genes and rearing environments on children’s self-regulative behaviors such as externalizing behavior and substance use (see Bakermans-Kranenburg & van IJzendoorn, 2011; Belsky & Pluess, 2009). Also, past studies found moderation effects when considering parental dopaminergic genes, parents’ environment and their joint effects on parenting behaviors (Beach et al., 2012; van IJzendoorn et al., 2008). Focusing on a single candidate gene (*DRD4*) and a cumulative gene index (*DRD4, DRD2*, and *ANKK1*), the present research confirms the importance of dopaminergic genes in understanding variability in the influence of environmental inputs on individual development.

Study 1 and Study 2 are noteworthy in their different operationalization of genetic status. Study 1 used a single, well documented gene whereas Study 2 used a cumulative index of three genes related to the dopaminergic system. Recent research suggests that multiple genes may exert a shared polygenic influence (i.e., cumulative genetic influence) on individual behaviors (Belsky & Beaver, 2011; Masarik et al., 2014; Wickrama & O’Neal, 2013). The greater power of a genetic index may explain the one finding that was inconsistent with expectations in Study 1. I expected youth genotype to interact with parenting in a “for better and worse” manner, but
this differential susceptibility effect did not merge. It may be the case that a cumulative index may better capture environmental susceptibility.

Some strengths and limitations of the research should be noted. To test the complex models based on the bioecological perspective, analyses of moderated mediation were conducted (Preacher et al., 2007). Interaction terms were used to test the $G \times E$ processes, and each $G \times E$ effect were examined through the J-N technique to identify whether it would be consistent with the differential susceptibility model or the diathesis-stress model. Although the present studies were conducted using advanced methods and techniques, the use of candidate genes has recently come under criticism resulting in the development of increasingly restrictive standards for publication. Among the issues described in the policy of *Behavior Genetics* (Hewitt, 2012), attention must be paid to: (1) the reliable and valid measures of all study variables; (2) sample sizes that provide sufficient statistical power based on reasonable estimates of effect sizes; (3) prospective longitudinal designs; and (4) need for replication.

In regard to these standards, the present studies used (a) prospective designs, (b) appropriate measures with solid psychometric properties, (c) examined population stratification, and (d) selected genes with reasonable biological plausibility. In the context of modern genetic research, however, sample size would be a major limitation in the present studies. Also, editors strongly suggest that the findings be replicated in a second data set. Given the modest sample size of the studies and problems with replication of findings in $G \times E$ research in general, replication of these studies in larger datasets is important for future research. This suggests that additional research is needed using a second and hopefully larger dataset. The National Longitudinal Study of Adolescent to Adult Health (Add Health) is one such dataset that may to some extent allow replication of the present findings.
Conclusion

This dissertation demonstrates the ways in the genetic status of children and their parents can jointly affect how multilevel contexts and mutual influence processes affect their developmental outcomes. The results suggest a continued need for studies that integrate transactional developmental perspectives and G × E research. Such research expands the focus of individual differences to include exposure to environmental characteristics in accounting for complex transactions among family members and communities over time. This dissertation also provides support for the influence of genetic status in the dopaminergic system as conferring susceptibility to environmental input in a “for better and for worse” manner.

In the two studies of this dissertation, I tested G × E processes to identify whether they are consistent with a diathesis-stress or different susceptibility model. Distinguishing between the diathesis-stress and differential susceptibility models is important to understand the mechanism of genetic moderation effects on individual development. Understanding these two different models can contribute to the intervention program development. Whereas the diathesis-stress model supports the argument that individuals carrying certain genetic vulnerable alleles are relatively more likely to develop poorly when exposed to environmental stressors, the differential susceptibility model argues that their genetic sensitivity to both negative and positive environmental factors can make them more benefit from diverse intervention programs shaping favorable environmental conditions.

The dissertation studies are noteworthy because they add to knowledge about the G × E process of both parent and child in the family system. Ecology of the family including the relationship between parent and child has been focused as an important context for human development. The dissertation expands this perspective by investigating the influence of
external environments on the functioning of families and the interaction genetics and environment in family processes. The findings of this dissertation can provide new insights through the investigation of multiple family members’ genetic moderation processes in the dynamic human ecological systems.
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