HHS Public Access

Author manuscript

J Fam Psychol. Author manuscript; available in PMC 2017 October 01.

Published in final edited form as:

J Fam Psychol. 2016 October ; 30(7): 780–790. doi:10.1037/fam0000228.

Genetic Moderation of Transactional Relations Between Parenting Practices and Child Self-Regulation

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Abstract

The present study addressed the ways in which parent and child dopamine D4 receptor (*DRD4*) genotypes jointly moderate the transactional relations between parenting practices and child self-regulation. African American children ($N = 309$) and their parents provided longitudinal data spanning child ages 11 to 15 years and a saliva sample from which variation at *DRD4* was genotyped. Based on the differential susceptibility perspective, this study examined moderation effects of *DRD4* status on (a) the extent to which parenting practices affect child self-regulation and (b) the extent to which child self-regulation, as an environmental influence on the parent, affects parenting behavior. Results indicated that responsive-supportive parenting interacted with children's *DRD4* status to influence increases in child self-regulation. Also, child self-regulation interacted with parent's *DRD4* status to predict changes in parenting practices. Both G × E effects conformed to a differential susceptibility model in which parents' and children's *DRD4* genes operated to increase environmental sensitivity in a "for better and for worse" manner.

Keywords

transactional relations; parenting; self-regulation; gene by environment interaction

Transactional models of human development underscore the bidirectional influences between individuals and their environments (Sameroff & MacKenzie, 2003). This perspective underscores the ways in which parents and children both act as environmental influences on each other's behavior. Studies of child self-regulation document that effective parenting promotes child self-regulation, which in turn supports effective parenting behavior (Brody & Ge, 2001; Moilanen, Rasmussen, & Padilla-Walker, 2015; Yates, Obradović, & Egleand, 2010). These mutual influence processes have been documented in families with children of various ages, from infancy through adolescence (Moilanen et al., 2015; Scaramella & Leve, 2004). Recent studies of gene × environment interactions (G × E) suggest that environmental influences on behavior can be moderated by individual genetic variation. These studies include examinations of the ways in which parenting behaviors interact with child genotype to predict child behaviors and, to a limited extent, the ways in which parental genotype may interact with environmental inputs to influence parenting

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behaviors (see Bakermans-Kranenburg & van IJzendoorn, 2011; Van IJzendoorn, Bakermans-Kranenburg, & Masman, 2008). Ostensibly, given the transactional nature of parenting behaviors and child self-regulation, the G × E framework should inform (a) the extent to which parenting practices affect child self-regulation, and (b) the extent to which child self-regulation, as an environmental influence on the parent, affects parenting behavior. No research to date, however, has examined the joint effects of parent and child genotypes on the reciprocal influences between parenting and child self-regulation.

Recent G × E theory suggests that genotypes may confer *differential susceptibility* to environmental input (Belsky & Pluess, 2009). Accordingly, specific genotypes act to amplify environmental input in a “for better or for worse” manner. Thus, individuals who carry *plasticity alleles* will experience heightened benefits from positive environments and intensified detriments from negative environments. We investigated the genetic moderation of transactional relations between parenting practices and child self-regulation from a differential susceptibility perspective, focusing on plasticity alleles related to dopaminergic functioning. Hypotheses were tested with prospective data from 309 African American children who were followed from ages 11 to 15 and the parents who were primarily responsible for their care. Child genotypes were expected to moderate the influence of parenting behavior on subsequent self-regulation, which, in turn, was expected to interact with parent genotypes to predict changes in parenting behavior.

Bidirectional Associations Between Parenting Practices and Child Self-Regulation

Self-regulation is the ability to manage one’s attention, affect, and activity in accordance with internal and external demands (Crockett, Raffaelli, & Shen, 2006). It includes the skills needed to monitor, evaluate, modify, and inhibit one’s emotions or behavior, either actively or passively, to achieve one’s personal goals in accordance with societal standards of behavior (Moilanen, Shaw, & Fitzpatrick, 2010). As an important determinant of children’s psychosocial adjustment, self-regulation is particularly vital for the prevention of maladaptive developmental outcomes in adolescence, including sexual risk-taking, delinquency, and substance use (Crockett et al., 2006; Quinn & Fromme, 2010; Wills, Walker, Mendoza, & Ainette, 2006), and the promotion of positive outcomes, such as prosocial behavior and academic achievement (Bowers et al., 2011; McClelland & Wanless, 2012).

Although self-regulation is relatively stable from early childhood through adulthood (Raffaelli, Crockett, & Shen, 2005), it continues to develop throughout young adulthood (Steinberg et al., 2008). The transition from childhood to adolescence has been identified as a sensitive period for the development of self-regulation, during which parental socialization can affect children’s self-regulatory competencies (McClelland, Geldhof, Cameron, & Wanless, 2015). During late childhood and early adolescence, parents influence their children’s self-regulatory development through behavioral modeling, parenting practices, and the family’s emotional climate (Moilanen et al., 2015). For example, warm and supportive parents help children to modulate their arousal by discussing emotions so that

children can better respond to and learn from parental socialization efforts (Morris, Silk, Steinberg, Myers, & Robinson, 2007). Parental warmth and responsivity, along with avoidance of harsh parenting, also increase the likelihood that children will attend to parents' guidance in developing strategies to regulate their own behavior (Choe, Olson, & Sameroff, 2013).

Most research on the relations between parenting and self-regulation has focused on the effects of parenting on children's self-regulation. However, family systems theory as presented by Bowen (1974) suggests that families are composed of interconnected and interdependent individuals. The individuals within the family system are expected to respond to each other in certain ways according to their roles, and a family member's behavior both influences and is influenced by other family member's behaviors. Several studies informed by the family systems perspective document the influence of child behaviors on parenting practices (see Minuchin, 2002). For example, research conducted with young children revealed that children's poor self-regulation elicited parenting stress and distress, which led to harsher, less optimal parenting practices (Perry, Mackler, Calkins, & Keane, 2014). Bidirectional influence processes persist into late childhood and adolescence (Moilanen et al., 2015; Scaramella & Leve, 2004). Moilanen et al. (2015) reported that self-regulation among children between the ages of 11 and 16 predicted decreases in authoritarian and permissive-indulgent parenting styles.

The present study is part of an ongoing program of research examining risk and resilience among African American families in the rural South. Participants live in small communities in rural Georgia in which poverty rates are among the highest in the nation and unemployment rates are above the national average (Dalaker, 2001). Life in rural areas can be more challenging than in urban areas due to restricted educational and employment opportunities, lack of public transportation, absence of recreational facilities, and difficulties in obtaining physical and mental health care (Brody et al., 2012). Research focusing on African American families living in disadvantaged rural communities demonstrated that parents' caregiving practices can serve as protective factors against child and youth risk behaviors that compromise their development (Brody et al., 2005). Previously, Brody and Ge (2001) found bidirectional influences in a prospective study of rural African American children and their parents. They examined the influence of *responsive-supportive* parenting, defined as high levels of parental support and warmth combined with consistent discipline and monitoring. Children who, at age 11, received responsive-supportive parenting from their mothers evinced increases in self-regulation during late childhood, which in turn predicted increases in responsive-supportive parenting when the children were in early adolescence. Consistent with these studies, we expected responsive-supportive parenting practices to predict changes in child self-regulation, which in turn would influence parents' use of responsive-supportive parenting during late childhood and early adolescence.

Differential Susceptibility Effects on Child Self-Regulation and Parenting Behavior

Advances in molecular genetics have sponsored heightened interest in the ways in which genetic variation may moderate environmental influences on child development and, to a lesser extent, parenting behavior. Numerous G × E studies examined interaction effects of parenting practices and children's genotypes on the development of self-regulatory behaviors (see Bakermans-Kranenburg & van IJzendoorn, 2011; Belsky & Beaver, 2011; Kochanska et al., 2009; Smith et al., 2012). Although most G × E studies have focused on child genotypes interacting with parenting to influence child developmental outcomes, some studies have examined the potential for genetic variation among parents to interact with environmental input to affect parenting behaviors (Beach et al., 2012; Cho & Kogan, 2016; Van IJzendoorn et al., 2008). Children comprise an environmental influence on parents, and genetic variability within parents can moderate the influence of child self-regulation on their behavior.

The majority of G × E studies conceptualize the interaction effect from a *diathesis–stress* perspective, in which specific genotypes were hypothesized to confer vulnerability to negative environmental inputs on the individuals who carried them. Emerging research, however, suggests that the diathesis-stress perspective may be limited. Many of the genotypes investigated as vulnerability factors may actually function as plasticity alleles, amplifying an individual's sensitivity to both positive and negative environmental influences. As mentioned previously, this dynamic has been termed as *differential susceptibility* effects (Belsky & Pluess, 2009).

Dopamine D4 Receptor Gene and Differential Susceptibility

One of the most promising candidate polymorphisms for conferring differential susceptibility to environmental influences is found on the dopamine D4 receptor (*DRD4*) gene. *DRD4* is a variable nucleotide tandem repeat (VNTR) polymorphism composed of 16 amino acid (48 base pairs) 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 or long, with the short category defined as having 6 or fewer repeats and the long category as having 7 or more repeats (Beach et al., 2012; Brody et al., 2012; McGeary, 2009). *DRD4* long 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 long allele versus two short alleles (Asghari et al., 1995; Levitan et al., 2006).

Converging evidence suggests that long forms of the *DRD4* allele are associated with differential susceptibility; that is, they amplify both positive and negative environmental inputs. We identified multiple studies that documented *DRD4* effects in amplifying rearing environmental inputs on children's self-regulatory outcomes such as externalizing problems, effortful control, and sensational seeking (see Bakermans-Kranenburg & van IJzendoorn, 2011). For example, high parental warmth–responsiveness was associated with low levels of

externalizing behavior when African American children carried at least one *DRD4* long allele (Propper, Willoughby, Halpern, Carbone, & Cox, 2007). Differential susceptibility effects have also been documented for parents carrying a long allele of *DRD4* (Beach et al., 2012; Cho & Kogan, 2016). For example, Beach et al. (2012) found that parents with *DRD4* long alleles, compared to those without them, displayed either more or less negative arousal, a proximal determinant of parenting behaviors, when they were exposed to contextual stress and support.

Although the precise biological mechanisms linking *DRD4* status to differential susceptibility effects are unclear, a plausible hypothesis has been advanced. The dopaminergic system is engaged in attentional, motivational, and reward systems (Robbins & Everitt, 1999). Reward signals initiate a phasic burst of midbrain dopamine neurons, which induce positive emotional states and organize the learning of cues that predict future rewards (Spear, 2000). *DRD4* long alleles are associated with reduced dopaminergic signaling. Researchers suggest that dampened dopaminergic signaling affects how individuals learn from the environment and the kinds of stimuli and cues that are intended to. Specifically, the downregulation of dopaminergic activity results in a preference for immediate rather than distal forms of reinforcement from the environment and suggests that the *DRD4* long allele will be associated with learning from high-intensity environmental cues (Tripp & Wickens, 2008). 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 Present Study

To date, most G × E research has focused on the ways 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 our knowledge have considered the interaction of parental genotype with child behavior to predict parenting behavior. Also, to our knowledge, there are no studies that investigate the ways in which parent and child genotypes jointly moderate 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 associated with both parent and child.

Study hypotheses are summarized in Figure 1. Responsive-supportive parenting was hypothesized to interact with children's *DRD4* status to predict increases in child self-regulation. Child self-regulation, in turn, was hypothesized to interact with parental *DRD4* status to forecast changes in responsive-supportive parenting from child ages 11 to 15 years. In each case, we hypothesized that *DRD4* would operate as a plasticity factor: Individuals with *DRD4* long alleles would be more susceptible to environmental influences. We further expected that the G × E effects would conform to a differential susceptibility rather than a diathesis-stress model.

Methods

Participants

Study hypotheses were tested with 309 African American children (168 girls and 141 boys) and their primary caregivers—typically their biological mothers (94.5%)—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 was controlled. Data were obtained at four time points spanning 4 years. Because the interval between the first two time points was short (6 months), data from these assessments were averaged; these means constitute the Time 1 values. Children's mean ages were 11.48 years at Time 1 (T1; *SD* = .51), 13.46 years at Time 2 (T2; *SD* = .50), and 15.49 at Time 3 (T3; *SD* = .50).

At T1, primary caregivers' mean age was 37.04 years (*SD* = 7.51), and a majority of the caregivers, 81.8%, had either completed high school or obtained a GED. Of the primary caregivers, 40.6% were married and living with their husbands. At T1, 72.9% of the primary caregivers were employed outside the home, and mean family income was $2,037 (*SD* = $1,480) per month. Although the primary caregivers in the sample worked an average of 39.4 hours per week, 46.3% of the participants lived below federal poverty standards, and another 50.4% lived within 150% of the poverty threshold. These families can be described as working poor.

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 contacted by research staff members to schedule assessments in the families' homes. Primary caregivers gave written consent to their own and their children's participation, and children gave written assent to their own participation. At the home visits, field researchers administered questionnaires to the primary caregiver and target child in an interview format via computer-assisted technology using laptop computers. Each interview was conducted privately, with no other family members present or able to overhear the conversation. Each family was paid $100 after each assessment. Primary caregivers also provided the names and locations of the target children's schools and authorized the children's teachers to provide the researchers with information about the children. Packets of teacher questionnaires were mailed to school secretaries, who distributed them to the teachers and returned the completed questionnaires. If teacher questionnaires were not returned within 3 weeks, up to three reminder telephone calls were made to the schools' secretaries. The response rate was 93.8%. Secretaries were paid $10 for each packet they returned, and each teacher who completed a packet was paid $20.

Measures

Responsive-supportive parenting—At T1 and T3, primary caregivers reported on their responsive-supportive parenting using a measure developed by Brody and colleagues (Brody & Ge, 2001; Brody et al., 2005) that included items assessing child monitoring, child management, and (lack of) harsh or inconsistent parenting. Five items concerning child monitoring (e.g., "How often do you know where your child is when he or she is away from home?" and "How often do you know when your child gets in trouble at school or someplace else away from home?") were rated on a 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 or 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 using four items (e.g., "When your child does something wrong, how often do you blow up at him/her?" and "When your child does something wrong, how often do you tell him/her to get out or lock him/her out of the house?") with a response set ranging from 1 (*never*) to 4 (*always*). The harsh-inconsistent parenting items were reverse scored. These subscales were submitted to an exploratory factor analysis to confirm the unidimensionality of responsive-supportive parenting. The analysis yielded a dominant factor that accounted for 53.29% of variance at T1 and for 55.22% of variance at T3. Factor loadings ranged from .61 to .79 at T1 and from .60 to .80 at T3. Thus, the items were standardized and summed to form the responsive-supportive parenting scale. 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), which has been used extensively with African American children and youth in prior studies (Brody et al., 2001, 2005; Wills et al., 2006). The measure included 12 items rated on a scale 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 or her behavior (reverse scored)." Cronbach's alphas for children's self-regulation were .89 at T1 and .88 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. The genotype at *DRD4* was determined for each participant using the primers F-GGCGTTGCCGCTCTGAATGC and R-GAGGGACTGAGCTGGACAACCAC, 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 long allele were assigned a code of 1 (46.6% of parents; 46.3% of children), and participants who were homozygous for the short allele were assigned a code of 0 (53.4% of parents; 53.7% of children). 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 that could influence the relations among study variables were controlled. Consistent with previous studies (Brody et al., 2014; Cho & Kogan, 2016), 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), single-mother-headed household structure, and caregiver education level less than high school graduation. Each indicator was coded dichotomously (0 = *absent*, 1 = *present*), and the scores were summed to form an index that ranged from 0 to 5 ($M = 1.65$, $SD = 1.19$). Child 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 (0 = *control*; 1 = *treatment*).

Analysis plan

Study hypotheses were tested using structural equation modeling (SEM) as implemented in Mplus (Muthén & Muthén, 1998-2012). Missing data were managed with full information likelihood estimation (FIML). Interaction terms were created to investigate the G × E hypotheses pertaining to both parent and child genotypes, as specified in Figure 1. To produce a common scale, standardized regression weights were used in which all study variables were standardized before the interaction terms were calculated (Dawson & Richter, 2006).

To investigate differential susceptibility effects, we 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 self-regulation) and categorical (i.e., genotypes) variables. Using the J-N technique, we examined crossover patterns and regions of significance, which refer to the ranges of moderator values for which the independent and dependent variable are significantly associated (Roisman et al., 2012). This procedure uses asymptotic variances, covariances, and other regression parameters to determine the upper and lower boundaries of the predictor variable at which groups representing a multilevel moderator differ significantly in terms of the outcome variable (Smith et al., 2012). The J-N technique indicates whether each G × E interaction effect suggests a differential susceptibility or a diathesis-stress effect.

Results

Preliminary analyses

Attrition analyses were conducted to evaluate predictors of non-participation on the basis of project attrition and unwillingness to provide DNA. From T1 to T3, 32 families (6.2%) left

the study. We investigated differences on all study variables between retained participants and those who left the study by T3; no differences were detected. At T3, 485 children and their parents were asked to provided DNA; 471 children (97.1%) and 429 parents (88.5%) agreed to provide samples. Among these participants, 415 children (88.1%) and 353 parents (82.3%) had valid information on their *DRD4* genotypes. Successful genotyping for both children and their parents was achieved for 309 families. We investigated differences on all study variable between participants who agreed to provide samples and had valid genotyping information and those who did not; no differences were detected. Study hypotheses were tested with the 309 children and their parents for whom genotyping was successful. We next checked for evidence of gene-environment correlation (*r*GE), a non-random distribution of environments among people with different genotypes, which can confound the interpretation of G × E effects (Caspi & Moffitt, 2006). Table 1 presents zero-order correlations for the study variables; no significant associations between parent or child genotypes and the study variables were identified, ruling out potential *r*GE 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. 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 2 presents the SEM for the hypothesized transactional relations between responsive-supportive parenting and child self-regulation. Consistent our hypotheses, responsive-supportive parenting was significantly associated with increases in child self-regulation ($\beta = .16$, $p < .05$), which in turn influenced changes in parenting practices ($\beta = .11$, $p < .05$). Figure 3 presents the final SEM for the hypothesized genetic moderation effects on transactional relations between responsive-supportive parenting and child self-regulation. In the sections that follow, we first describe the portion of the model representing the interaction effect of responsive-supportive parenting and child *DRD4* status on changes in child self-regulation. Then, we focus on the interaction effect of self-regulation and the parental *DRD4* status on changes in parenting practices. Each G × E process was delineated through post hoc analyses designed to determine whether these genetic moderation processes support the differential susceptibility or the diathesis-stress model.

Responsive-supportive parenting × child DRD4 effects on self-regulation

The results presented in Figure 3 indicate that the parameter representing the interaction between responsive-supportive parenting and child *DRD4* significantly predicted increases in child self-regulation ($\beta = .17$, $p < .05$). The J-N procedure was conducted to test the differential susceptibility hypothesis by assessing regions of significant difference between groups who had *DRD4* short alleles and at least one *DRD4* long allele. Figure 4a depicts the effect of children's *DRD4* status 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 at least one long allele of *DRD4* was significantly different from zero ($b = .31$, $p < .01$), whereas the slope for children carrying only *DRD4* short alleles was not significantly different from zero

(b =.05, p = .45). Thus, parenting practices were associated with child self-regulation only for children carrying *DRD4* long allele.

The graph of the interaction depicted in Figure 4a 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 4a 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 .43, which is within the range consistent with differential susceptibility effects (Roisman et al., 2012).

Child self-regulation × parent DRD4 effects on parenting practices

Consistent with our hypotheses, the data presented in Figure 3 demonstrate that the interaction term between child self-regulation and parental *DRD4* status was associated significantly with increases in responsive-supportive parenting (β = .22, p < .01). We then conducted the J-N procedure to assess regions of significant difference between groups who had *DRD4* short alleles and *DRD4* long alleles (see Figure 4b). Self-regulation enhanced responsive-supportive parenting only for parents with at least one *DRD4* long allele (b = .28, p < .01; for parents carrying *DRD4* short alleles, b = −.05, p = .24). The J-N technique identified a region of significant difference on both sides of the interaction effect when self-regulation was more than .83 *SD* or less than −.15 *SD* from the sample mean. The G × E interaction was more consistent with a differential susceptibility model than a diathesis-stress model (PoI = .42).

Discussion

Drawing on transactional (Sameroff & MacKenzie, 2003) and differential susceptibility (Belsky & Pluess, 2009) perspectives, 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, we examined the moderating effect of *DRD4* status. The influence of responsive-supportive parenting on increases in child self-regulation was moderated by children's *DRD4* status. Child self-regulation, in turn, interacted with parents' *DRD4* status to affect changes in parenting practices. Both G × E effects were consistent with a differential susceptibility model in which parents' and children's *DRD4* genes operated to increase environmental sensitivity in a "for better and for worse" manner rather than solely conferring vulnerability to negative environments.

Research based on transactional models informed by the family systems perspective underscore the bidirectional influence between parenting practices and child self-regulation (Brody & Ge, 2001; Moilanen et al., 2015; Yates et al., 2010). The present study extended this literature by examining the ways in which parent and child genotypes moderate this bidirectional influence process. Accumulating evidence supports differential influence of

parenting practices on child outcomes based on child genotypes (see Bakermans-Kranenburg & van IJzendoorn, 2011). No research, however, has examined the ways in which children constitute an environmental influence on parenting behavior within a G × E framework. To address this research need, the present study investigated transactional relations between parenting practices and child self-regulation by examining genetic moderation processes that took into consideration both parent and child genotypes.

Consistent with our hypotheses, the self-regulation of children who carried long alleles of *DRD4* was influenced significantly by responsive-supportive parenting. Our finding that child genotypes moderated the influence of parenting behavior on the development of self-regulation is consistent with a number of studies investigating child dopaminergic genes and self-regulatory outcomes (Bakermans-Kranenburg & van IJzendoorn, 2011; Smith et al., 2012). This G × E effect also conformed to a differential susceptibility model: Children carrying plasticity alleles manifested more self-regulation when they had high levels of responsive-supportive parenting, but less self-regulation if they were exposed to unsupportive parenting. Our finding is consistent with previous G × E studies testing interaction effects of parenting behaviors and child genotypes on child self-regulatory outcomes (see Bakermans-Kranenburg & van IJzendoorn, 2011; Belsky & Beaver, 2011). For example, Belsky and Beaver (2011) found that adolescents carrying more plasticity alleles were more positively and negatively influenced by 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' dopaminergic gene. We found that child self-regulation interacted with a parental *DRD4* status to predict changes in parenting practices. This result conformed to a differential susceptibility model rather than a diathesis-stress model. Parents carrying long alleles of *DRD4* demonstrated more supportive parenting when their child showed high levels of self-regulation, but less supportive parenting under low levels of child self-regulation. 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 (Beach et al., 2012; Cho & Kogan, 2016; Van IJzendoorn et al., 2008). For example, Van IJzendoorn et al. (2008) found evidence indicating that dopamine-related genes moderated the effect of daily hassles on parenting. Parents with 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. Focusing on parental *DRD4*, studies indicate that community context interacted with parental *DRD4* gene to predict protective parenting (Cho & Kogan, 2016) and parents' negative arousal, a proximal determinant of parenting behaviors (Beach et al., 2012). These findings, in conjunction with those from the present study, provide consistent support for the differential susceptibility model.

The present study builds on prior studies of parenting behavior by including child behavior in the range of environmental inputs that interact with parental genotypes. 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. Studies reveal

that parents with highly regulated children not only report less difficulty in parenting but also have an enhanced sense of efficacy and psychological well-being as parents (Brody & Ge, 2001; Moilanen et al., 2015; Yates et al., 2010). Brody (2004) referred to this process as a “basking” effect, in which one’s psychological well-being is heightened through close association with someone who possesses positive characteristics and displays competent behaviors. The current study suggests that the extent to which parents are affected by more or less well-behaved children may depend on the genetic susceptibility to the environment that the dopamine system confers.

The current study also underscores the importance of considering plasticity alleles in the dopaminergic system. Our findings indicate that *DRD4* long allele as a plasticity allele can function in ways that render individuals susceptible to both positive and negative environmental influences. Although the exact mechanisms through which dopaminergic genotypes confer differential susceptibility remain unclear, studies suggest that, given the role of the dopamine system in reward sensitivity and novelty seeking, individuals with more (compared with those with less) plasticity alleles may have more sensitive central nervous systems that respond readily to environmental influences (Dreher et al., 2009; Stice et al., 2012). Additional research is needed, however, to clarify the neurocognitive processes affected by dopaminergic genes that are associated with differential susceptibility to environmental inputs.

From an applied perspective, our findings suggest that an assessment of plasticity alleles in general, and *DRD4* in particular, may yield predictors of parent-child relationship quality in diverse environmental contexts. Heightened environmental sensitivity in a parent who has a highly active or difficult to manage child can lead to symmetrically escalating patterns of negative parenting and compromised child development (Patterson, 1997). In families in which both a child and a parent are reactive to environmental inputs, the potential for escalation will likely depend on other factors, such as the community environment and the overall family climate. In the case of generally positive environments, sensitivity in parents and children could result in a “basking” effect in which positive behavior forms a feedback loop. In contrast, if parents living in adverse rural communities are susceptible to environmental inputs, parental negativity can rapidly spill over to highly sensitive children (Cho & Kogan, 2016), who in turn reinforce this input. Recent research, however, also suggests that the same susceptibility patterns can operate to *increase the effectiveness* of preventive interventions (Brody, Yu, & Beach, 2015). Thus, particularly sensitive parents and children who cope with challenging environmental inputs both within and outside the parent-child relationship may benefit most from evidence-based, family-centered programming.

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 reports, whereas child self-regulation was teacher reported. We alternated parent reports and teacher reports at different time points; this research design mitigated the potential for self-report bias to inflate the association of parenting with self-regulation. We also used baseline controls to examine

changes in parenting practices and child self-regulation. To test effectively differential susceptibility hypotheses, it is important that measures of environmental factors and behavioral phenotypes have sufficient range to characterize both positive and negative aspects of the construct (Belsky & Beaver, 2011; Davies, Cicchetti, & Hentges, 2015). We assessed parenting practices and child self-regulation with measures that capture both positive and negative dimensions of the phenotypes. The composite of parenting practices included both positive (e.g., child monitoring, child management) and negative (e.g., harsh-inconsistent parenting) aspects of the construct. Similarly, child self-regulation was assessed using items reflecting both the presence and the lack of self-regulation.

Limitations of the study design are also apparent. The findings focused on a sample of African American families living in the resource poor rural communities; thus, the results may not generalize to other racial/ethnic or geographic groups. Also, only one genetic polymorphism was examined in the present study. Although there is considerable support for *DRD4*'s functioning as plasticity alleles (see Bakermans-Kranenburg & van IJzendoorn, 2011; Van IJzendoorn et al., 2008), several other polymorphisms in different neurotransmitter systems may also include plasticity alleles (Belsky & Beaver, 2011; Wickrama, O'Neal, & Lee, 2013). Future studies comparing the results of single versus multiple system indices are needed. These limitations notwithstanding, the present study expands understanding of the transactional relations between parenting practices and child development, documenting genetic moderation processes from both parents and children in the family system.

Acknowledgments

This research was supported by the National Institute of Child Health and Human Development under award number R01 HD030588. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

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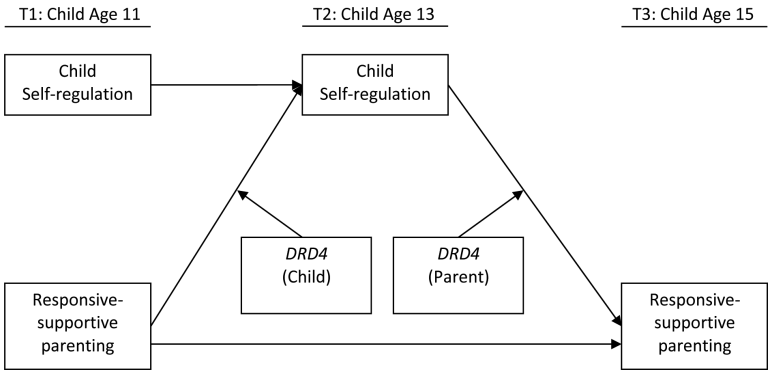

Figure 1.
Conceptual model

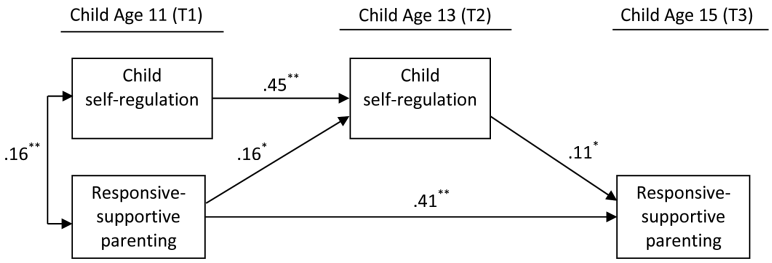

Figure 2.

Main effect model

Note. Standardized coefficients are shown. Child gender, family SES, and program intervention effects were controlled. χ = 4.37, df = 4, p = .35; RMSEA = .02; CFI = .99. *p < .05. **p > .01.

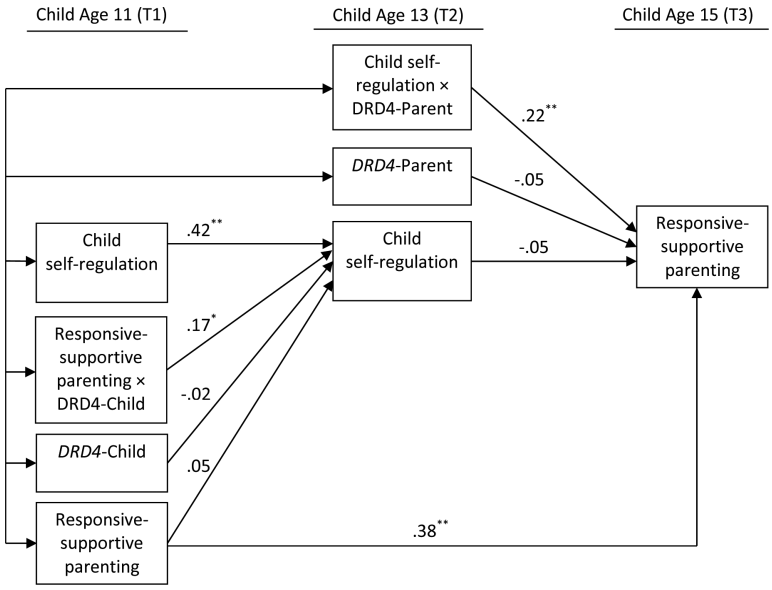

Figure 3.

Final model

Note. Standardized coefficients are shown. Child gender, family SES, and program intervention effects were controlled. χ = 19.36, df = 15, p = .19; RMSEA = .03; CFI = .98. *p < .05. **p < .01.1

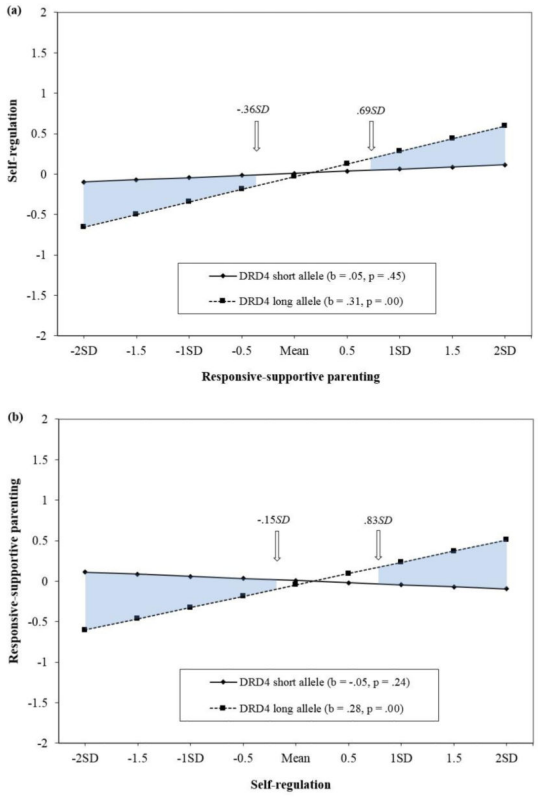

Figure 4.

(a) Responsive-supportive parenting × child DRD4 effects on self-regulation. (b) Self-regulation × parent DRD4 effects on responsive-supportive parenting.

Table 1

Correlations among the research variables

Variable	1	2	3	4	5	6	7	8	9
1. Child gender	–								
2. Family SES	.04	–							
3. Intervention	.03	.02	–						
4. Self-regulation T1	−.27**	−.10	.08	–					
5. Self-regulation T2	−.18**	−.02	.05	.46**	–				
6. Supportive parenting T1	−.01	−.09	.04	.18**	.25**	–			
7. Supportive parenting T3	−.03	−.10	.09	.16**	.23**	.41**	–		
8. *DRD4*-Child	−.01	−.02	.08	−.06	−.03	.02	−.03	–	
9. *DRD4*-Parent	.08	−.09	−.01	−.05	−.01	−.07	−.08	.43**	–
Mean	.45	1.65	.57	2.42	2.48	.00	.00	.46	.47
SD	.50	1.19	.49	.77	.88	11.83	7.45	.50	.50

*$p < .05$.

**$p < .01$.