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Interparental Relationship Sensitivity Leads to Adolescent Internalizing Problems: Different Genotypes, Different Pathways

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Abstract

Several studies have established that child interparental conflict evaluations link parent relationship functioning and adolescent adjustment. Using differential susceptibility theory and its vantage sensitivity complement as their framework, the authors examined differences between adolescents who vary in the *DRD4* 7 repeat genotype (i.e. 7+ vs. 7−) in how both interparental conflict and positivity affect adolescents' evaluations of interparental conflict (i.e., threat appraisals) and how these evaluations affect internalizing problems. Results from longitudinal multiple-group path models using PROSPER data (N = 452) supported the hypothesis that threat appraisals for 7+ adolescents would be more affected by perceptions of interparental positivity compared to 7− adolescents; however, threat appraisals for 7+ adolescents were also less affected by interparental conflict. Among 7− adolescents, interparental conflict perceptions were associated with higher threat appraisals, and no association was found for perceptions of positivity. For adolescents of both genotypes, higher threat was associated with greater internalizing problems.

Keywords

adolescent development/outcomes; interparental conflict; mental health; path analysis; sociobiology; systems theory

An extensive record of research documents that interparental conflict markedly disrupts child and adolescent development (Buehler et al., 1997; Cummings & Davies, 1994; Emery, 1982). Recent research on risk mechanisms has provided important insight and revealed meaningful heterogeneity regarding child and adolescent adjustment problems associated with interparental conflict (e.g., Davies & Cummings, 1994; Davies & Sturge-Apple, 2007; Fosco, DeBoard, & Grych, 2007; Grych & Fincham, 1990; Grych, Jouriles, Swank, McDonald, & Norwood, 2000). Germane to this work is an emergent developmental theory that has shown much promise for explicating for whom parenting and parenting processes

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affect children. In the general sense, *differential susceptibility theory* (DST; Belsky & Pluess, 2009; Boyce & Ellis, 2005; Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2011) posits heterogeneity in environmental sensitivity based on intrapersonal characteristics. More specifically, the perspective contends that child characteristics related to increased risk for maladjustment when exposed to harsh, unsupportive environments can also lead to exceptional positive adjustment when exposed to more supportive circumstances. Developed to complement DST, the *vantage sensitivity perspective* (Pluess & Belsky, 2013) emphasizes that some intrapersonal characteristics dispose greater sensitivity to specifically positive aspects of the environment relative to negative. These perspectives, as well as others (e.g., Davies, Martin, & Cicchetti, 2012), emphasize the importance of considering both positive and negative aspects of interparental relationships to understand their unique implications for adolescent maladjustment.

In the current study, we applied DST and its vantage sensitivity complement to prevailing perspectives on interparental conflict that place adolescent evaluations as the key link between parental acrimony and adolescent maladjustment (Davies & Cummings, 1994; Grych & Fincham, 1990). Specifically, we examined whether variation in the dopamine receptor *D4* (*DRD4*) gene is related to variation in sensitivity to both negative (conflict) as well as positive (positivity) dimensions of interparental relations. Below, we provide an overview of the research that links interparental conflict, adolescent evaluations, and adjustment. We then describe the implications of DST for this research.

Interparental Conflict, Subjective Evaluations, and Maladjustment

Children's emotional security in the interparental relationship (Davies & Cummings, 1994; Davies & Sturge-Apple, 2007) and cognitive appraisals of interparental conflict (Fosco et al., 2007; Grych & Fincham, 1990) have consistently been shown as a key link between exposure to interparental conflict and adolescent adjustment (Rhoades, 2008). Together, this body of research has identified three important factors related to interparental relationships and child adjustment: (a) adolescent perceptions, (b) adolescent evaluations, and (c) contextual factors.

Perceptions of Interparental Conflict

Perceptions of interparental conflict encompass the degree to which family members view conflicts as frequently occurring, intense, and poorly resolved. Adolescents' perceptions provide a gauge for actual exposure and awareness of interparental conflict that might not be captured by parent reports of relationship functioning. Consistent with this view, adolescents' perception of conflicts as intense or hostile is more closely related to their internalizing problems than more objective assessments of actual conflict within the household (Harold & Conger, 1997; Harold, Fincham, Osborne, & Conger, 1997). In addition, Fosco and Grych (2008) found only a moderate correlation between parent and child report of interparental conflict. Children's perceptions are thought to be the driving force behind their adjustment, and the degree to which adolescents perceive their parents' relationship as characterized by high levels of conflict (e.g., frequency, intensity) shapes their evaluations of the conflict (Grych & Fincham, 1990).

Considerable research supports the notion that destructive conflicts that are hostile, frequent, and poorly managed pose a greater risk for disruptions to adolescent development (e.g., Buehler et al., 1997; Cummings & Cummings, 1988; Goeke-Morey, Cummings, Harold, & Shelton, 2003). Alternatively, perception of interparental warmth, support, cooperation, and problem solving may benefit adolescents (Cummings & Davies, 1994). Studies investigating the implications of both positivity and conflict in parental relationships for adolescent outcomes are generally lacking, however. As Davies and colleagues (2012) pointed out, including both dimensions of interparental functioning can provide insight into the unique implications of each dimension for adolescent parental conflict evaluations (McCoy, Cummings, & Davies, 2009). No research has been published, however, that examines exposure to specific aspects of interparental warmth and support on children's perceptions of interparental conflict.

Evaluations of Interparental Conflict

Adolescents' evaluation of interparental conflict is an important factor that ties perceived interparental conflict to adolescent maladjustment (e.g., Davies, Harold, Goeke-Morey, & Cummings, 2002; Grych, Harold, & Miles, 2003). These evaluations, referred to as *threat appraisals* (Atkinson, Dadds, Chipuer, & Dawe, 2009; Grych & Fincham, 1990), reflect adolescents' worries about the implications of interparental conflict, including general fears that the conflict will result in something bad, or more specific concerns that conflict may lead to divorce, escalate into violence, lead to their involvement, or result in harm to the family or a family member (Atkinson et al., 2009; Grych, Seid, & Fincham, 1992). From an emotional security perspective, threat evaluations are thought to activate a social defense system to mobilize adolescents' strategies to preserve their sense of security in the context of the interparental relationship (Davies & Sturge-Apple, 2007). Within the cognitive-contextual framework, threat appraisals are conceptualized as a primary process that initiates coping strategies (Fosco et al., 2007; Fosco & Grych, 2010; Grych & Fincham, 1990). Although both perspectives conceptualize children's subjective experiences of parental conflict differently, they share the view that threat evaluations are central to the process of how interparental conflict affects coping and adjustment. Empirical research reliably supports threat appraisals as a key mechanism of risk for maladjustment (e.g. Cummings, George, McCoy, & Davies, 2012; Davies & Cummings, 1998; Davies et al., 2002; Fosco & Grych, 2007; Grych & Fincham, 1993; see Rhoades, 2008), most consistently for internalizing problems (Fosco & Grych, 2008; Grych et al., 2003). Inquiries into child characteristics that moderate these pathways may reveal different processes for different children and provide insight into specific points of intervention.

Contextual Factors of Interparental Conflict and Differential Susceptibility

Links among perception, threat appraisals, and adjustment all may be qualified by what Grych and Fincham (1990) termed *contextual factors*. Heavily studied are factors related to family relationships such as parental warmth and support or family emotional climate (Davies et al., 2002; DeBoard-Lucas, Fosco, Raynor, & Grych, 2010; Fosco & Grych, 2007). Child characteristics, such as gender or temperament (Davies & Lindsay, 2004; Lengua & Long, 2002), have received relatively little attention, however. This omission is conspicuous when viewed from a behavioral genetic perspective. For example, it is well

established within behavioral genetic studies that many child characteristics, ranging from children's temperament (Rowe & Plomin, 1977) to the perceptions of parenting they receive (Rowe, 1981, 1983), have significant genetic influence. Thus, a child's genotype may play an important role in modifying how individual children perceive exposure to interparental conflict (see also D'Onofrio & Lahey, 2010; Horwitz & Neiderhiser, 2011). Recent studies involving candidate genes, interpreted in the framework of the DST (Ellis et al., 2011), have led to new insights regarding parental effects on child outcomes (see Simons et al., 2013, for a recent parenting example).

At its core, DST proposes that individuals differ in the degree to which they are affected by environmental experiences (Ellis et al., 2011). Contrary to earlier models of risk, DST argues that individuals who are more sensitive to their environment are at greater risk for negative outcomes in adverse conditions but also gain disproportionally greater benefit in supportive contexts. Conversely, those who are less sensitive are less affected by exposure to adversity but also benefit less from supportive environments. Embedded within DST is the concept of vantage sensitivity, which focuses on individual factors that make one more susceptible specifically to positive aspects of the environment (Pluess & Belsky, 2013). Such positive aspects may be related to affluence, positive peer affiliations, family environment, and positive interparental relationships. Although these perspectives overlap conceptually, they are distinct in the sense that differential susceptibility specifically predicts sensitivity to both positivity and negativity, whereas vantage sensitivity is more narrowly focused in that it predicts sensitivity only to environmental positivity. Relevant to the current study is that both DST and vantage sensitivity suggest that exposure to interparental relations should be conditioned by endogenous factors such as child genotype. Such factors can provide a more complete picture of family influence on adolescent well-being.

DRD4 × Environment Research

Variation in the dopamine receptor D4 gene (*DRD4*) has generated considerable attention in the Gene × Environment interaction (see D'Onofrio & Lahey, 2010, for a review) research literature. Several studies have demonstrated that variation in *DRD4* moderates the impact of environmental exposures on an array of developmental outcomes (Bakermans-Krannenburg & van IJzendoorn, 2011; Bakermans-Kranenburg, van IJzendoorn, Mesman, Alink, & Juffer, 2008; Bakermans-Kranenburg, van IJzendoorn, Pijlman, Mesman, & Juffer, 2008; Beach, Brody, Lei, & Philibert, 2010; Belsky & Pluess, 2013; Knafo, Israel, & Ebstein, 2011). Specifically, the *DRD4* 7 variable number of repeats (VNTR) allele has been associated with variation in sensitivity to the social environment (see Bakermans-Kranenburg & van IJzendoorn, 2011, for a review). In brief, children who possess at least one copy of the 7-repeat allele (7+) tend to be more sensitive to contextual factors (e.g., parenting) compared to children who do not possess a 7-repeat copy (7−). Although much of this research has been cast within DST, we should emphasize that the results of these studies —that children who are 7+ show greater sensitivity to positive environments—are consistent with the vantage sensitivity perspective (Belsky & Pluess, 2013; Bakermans-Kranenburg & van IJzendoorn, 2011; Pluess, Stevens, & Belsky, 2013). This research indicates the *DRD4*

7+ allele preferentially disposes susceptibility to positive aspects of the environment, including aspects of positive interparental relationships.

There are many *DRD4* alleles and genotypes, and how to analyze them varies somewhat. Most methods revolve around separating the two most common alleles (4- and 7-repeat) into separate groups based largely on the work of Asghari et al. (1995), who showed the 7-repeat allele functions more poorly than the 4-repeat allele. One method is to collapse the alleles into short (≤ 5 repeats) and long (6 or more), but it is not clear that the number of repeats is directly related to function (Wong, Buckle, & Van Tol, 2000). With this issue in mind, and given that there is evidence that the third most common allele (2-repeats) functions similarly to the 4-repeat allele (Schoots & Van Tol, 2003), we chose to follow what is common in the literature related to DST, in which variation in the *DRD4* variable is studied as the presence or absence of 7 repeats of a 48 base pair sequence (i.e. 7− vs. 7+). The 7+ variant is associated with decreased neuronal dopamine signaling and thus provides a potential functional link between genetics and behaviors related to threat assessment. In addition, *DRD4* is expressed at relatively high levels in the prefrontal cortex (Oak, Oldenhof, & Van Tol, 2000), a region of the brain related to cognitive control. Given the implications for dopamine-related neurological processes, *DRD4* has been the subject of study in many areas, including a large literature on novelty seeking and attention-deficit/hyperactivity disorder (e.g., Faraone, Doyle, Mick, Biederman, 2001; Kluger, Siegfried, & Ebstein, 2002).

The Current Study

We took advantage of longitudinal data from families who participated in intensive in-home interviews that afforded tests of the DST and vantage sensitivity hypotheses. We tested a model that included both positive and negative parental assessments of their own relationship with their partner to capture interparental of conflict and positivity. We hypothesized that the presence of these factors will affect adolescents' perception of their parents' relationship (Harold et al., 1997; Harold & Conger, 1997). We expected conflict and positivity within the parent couple to be related to higher adolescent conflict perception and lower positivity perception. Similarly, we expected that couple positivity will be related to lower adolescent conflict perception and higher positivity perceptions.

In turn, we hypothesized that adolescents' perceptions of interparental conflict and positivity would better explain the degree to which they appraise parental conflicts as threatening than their parents' own assessment of their relationship. Indicators of couple relationship quality may reflect only a portion of adolescent exposure or awareness of their parents' relationship. Because DST and vantage sensitivity posit variation in sensitivity to environmental exposures, indicators of actual exposure captured by adolescent perception of interparental relationships is key for these hypotheses. As a result, we expected that couple relationship quality will not be directly related to appraisals of threat, after accounting for adolescents' perceptions. Adolescents' perception of conflict and positivity is expected to mediate the relation between parent relationship assessments and adolescent threat appraisals. Higher perceived conflict will lead to higher threat appraisals; higher perceived positivity will lead to lower threat. Last, as found in prior research, we expected that adolescents who report higher levels of threat to be at higher risk for internalizing problems.

We also tested the hypothesis that adolescents' perceptions of interparental conflict and positivity will be differentially associated with conflict appraisals as threatening as a function of having a more sensitive (7+) or less sensitive (7−) *DRD4* genotype. Drawing from both DST and vantage sensitivity, we hypothesized an association between couple positivity and threat appraisals for 7+ adolescents and null associations for 7− adolescents. Specific to DST, we further hypothesized an association between couple conflict and threat appraisals for 7+ adolescents only.

Method

Participants

Data used in this study come from the PROSPER (PROmoting School–community–university Partnerships to Enhance Resilience) project (Spoth, Greenberg, Bierman, & Redmond, 2004). PROSPER is designed to study the impact of a partnership mode of delivering preventive interventions through a university–school–cooperative extension collaboration. The PROSPER study includes 28 school districts in Iowa and Pennsylvania randomized into control and intervention conditions. A random sample of 2,267 families of adolescents from the first wave of in-school data collection was invited to participate in home-based family data collection; 979 (43%) participated.

In-home–based visits were conducted twice in sixth grade (in the 2003 fall and spring, Waves 1 and 2) and annually in the spring thereafter for 3 years (Waves 3, 4, and 5). The in-home procedures included written questionnaires completed independently by the adolescent; mother; and, if present, father. During Wave 5 of the in-home assessment, in which 749 families took part, parents were asked to consent for adolescent DNA data collection. In addition to the 537 DNA samples collected at that point, a later data collection by mail in young adulthood provided an additional 57 samples, for a total of 594 in-home participants who provided saliva samples for DNA data. Of those who provided DNA, 98.5% were successfully genotyped for the *DRD4* polymorphism. For purposes of the current article, children were included in these analyses only if they lived in a household where the coresident parents were married or in a marriage-like relationship at the initial assessment (see also Fosco & Feinberg, 2014). The final analysis sample consisted of 452 families. Comparisons between our analytic sample and the larger population of PROSPER two-parent families (N = 8,485) revealed few differences. There were slightly fewer two-biological-parent families (76.9% vs. 80.2%), fewer adolescents on free/reduced-price lunches (20.9% vs. 28.1%), and more Caucasian participants (91.1% vs. 86.0%) in our analysis sample. Effect sizes for these differences were all small (r < .05). Parent warmth/support, hostility, involvement, and family cohesion did not differ between the samples.

Measures

Means, standard deviations, and correlations can be seen in Table 1.

Couple conflict and positivity—Couple conflict and couple positivity were assessed as separate constructs during Wave 1 using seven- and four-item scales for each construct, respectively. Example conflict items included: During the past month when you and your

partner have spent time talking or doing things together, how often did your partner "Get angry at you," "Argue with you whenever you disagreed about something," and "Shout, yell, or scream at you." The couple positivity measure shared the same stem and included items such as "Let you know he/she really cares about you" and "Act loving and affectionate toward you." All items were scored on a scale that ranged from 1 (*never*) to 7 (*always*). For both couple conflict and couple positivity, mothers and fathers completed the scales twice: once with reference to their partner's behavior toward them and a second time with reference to their own behavior toward their partner. Thus, each measure comprised four scales—both father and mother reporting on self and other behavior. Regarding couple conflict, each seven-item scale showed good internal consistency (α = .89–.84) and the four scales had a high mean inter-item correlation (r = .62). These four scales were averaged to create a couple conflict composite. Higher scores indicated greater couple conflict. Regarding couple positivity, each four-item scale showed good internal consistency (α = .90–.92), and the four scores were also highly intercorrelated (r = .57). These four scales were averaged to create a couple positivity composite. For both measures, only mother report was included for cases missing father report (N = 62).

Adolescent perception of interparental conflict and positivity—One item was available at Wave 1 for adolescent perceived interparental conflict: "Thinking about your parents or guardians, how often would you say they argue or disagree with each other?" This item was scored on a scale that ranged from 1 (*never*) to 5 (*always*). Higher values indicate greater child perception of interparental conflict. Adolescent perceived interparental positivity was assessed at Wave 1 using two items: (a) "Thinking about your parents or guardians, in general, how happy do you think they are with their relationship?" and (b) "How often do your parents or guardians hug, kiss, hold hands, or say nice things to each other?" The former item was scored on scale that ranged from 1 (*very unhappy*) to 5 (*very happy*) and the latter on one that ranged from 1 (*never*) to 5 (*always*) scale. These two items, with r = .46, were averaged to create a composite measure.

Threat appraisals—Adolescents' threat appraisals were assessed at Wave 2 with four items drawn from the Threat subscale of the Children's Perceptions of Interparental Conflict Scale (Grych et al., 1992). The Children's Perceptions of Interparental Conflict Scale has demonstrated consistent reliability with adolescents (Fosco & Grych, 2010; Grych et al., 1992). Items on the Threat subscale began "When my parents argue. . ." followed by "I'm afraid something bad will happen," "I worry that one of them will get hurt," "I'm afraid that they will yell at me too," or "I worry that they might get divorced" (α = .88). Items were scored 1 (*strongly disagree*) to 5 (*strongly agree*) and were averaged to create an overall scale that reflects fears and potential stress that may result from interparental conflict.

Internalizing problems—During the Wave 3 in-home assessment adolescents completed the Youth Self-Report 11–18 (Achenbach, 1991). The Internalizing Problems subscale consisted of 14 items designed to measure problems related to anxiety and depression (α = .88). Example items include "I am too fearful or anxious" and "I feel lonely." The response options for each item were 0 = "not true," 1 = "somewhat or sometimes true," and 2 = ("very true or often true." Higher values indicate greater internalizing problems.

***DRD4* genotyping—**DNA was collected by buccal swabs and extracted using a modified phenol-chloroform technique (Freeman et al., 2003). A portion of the collected DNA was genotyped for the polymorphic VNTR site in the *DRD4* gene at the Penn State Genomics Core (Anchordoquy, McGeary, Liu, Krauter, & Smolen, 2003) using primer sequences developed by Lichter et al. (1993) with the forward primer fluorescently labeled. Amplification products were analyzed using a 3730XL DNA Analyzer and Genotyper software, Version 4.0 (Applied Biosystems, Foster City, CA). Nine alleles were detected, ranging in size from 2 repeats to 10 repeats. Frequencies of the most common alleles (> 5%) were 2-repeat (9%), 4-repeat (64%), and 7-repeat (20%). The remaining alleles (3-, 5-, 6-, 8-, 9-, and 10-repeats) summed to 3%. A total of 23 distinct genotypes were detected, the five most common being 7/7 at 3.9%, 3/4 at 4.1%, 2/4 at 12.8%, 4/7 at 27.4%, and 4/4 at 39.3%. Regenotyping ~10% of the samples revealed an error rate of 7.5% (4/53). For the present study, *DRD4* variability was coded on the basis of the presence (7+) versus absence (7−) of at least one copy of the *DRD4* 7-repeat allele. For clarity, based on this coding scheme, adolescents without a 7-repeat but with at least one longer allele (i.e. 8, 9, or 10) were included in the 7− category. When the alleles are collapsed to 7+ and 7− the error rate falls to 5.7% (3/53) due to one of the four errors not changing their status as being 7+. This rate is not surprising given the difficulty of amplifying the 7-copy allele in the presence of the 4-copy allele. In addition, the genotypes were in Hardy–Weinberg equilibrium, $\chi^2(1) = 0.03$, *ns*, increasing confidence that the number of individuals who were 7+ heterozygotes (the difficult genotype to assess) was accurately reported. It is important to note that if error were nonrandom, bias might be introduced into our results. To explore this possibility, we examined correlations between variables that are included in the path model and *DRD4* (see Table 1). We also considered child gender, child age, parent age, parent education, and household income. All correlations with *DRD4* were |.08| or less, suggesting the error rate did not introduce bias. Participants with at least one copy of the 7-repeat allele were coded 1 (7+; $N = 158$, 35.0%); all other participants were coded 0 (7− ; $N = 294$, 65.0%). Although there are alternative *DRD4* coding methods (e.g. 4/4 vs. 7+; short vs. long), we adopt the 7+ versus 7− coding on the basis of its function (Asghari et al., 1995; Schoots & Van Tol, 2003; Wong, et al., 2000) and to maintain consistency with other DST studies that include *DRD4* (Bakermans-Kranenberg & van IJzendoorn, 2011).

Plan of Analysis

Path analysis was implemented to examine pathways from couple relationships to adolescent internalizing problems. Although different analytic approaches could be used to evaluate components of the proposed model, by simultaneously estimating all possible associations path modeling conservatively estimates model parameters. Critical to our different pathways hypotheses is that indirect effects can also be estimated. Models were estimated using Mplus Version 6.1 (Muthén & Muthén, 1998–2012). Full-information maximum-likelihood estimation was used to reduce potential bias incurred due to missing data at later waves (see Schlomer, Bauman, & Card, 2010). Analyses were conducted by first examining overall model fit indices, chi-square, the comparative fit index (CFI), the nonnormed or Tucker–Lewis index (TLI), and the root-mean-square error of approximation (RMSEA) and its standardized root-mean-square residual (SRMR). Models met criteria for adequate overall fit

when CFI/TLI values were greater than .95, RMSEA values were less than .08, and SRMR values were less than .08 (Hu & Bentler, 1999).

To evaluate differences in the model across genotypes, three steps were undertaken. First, to obtain baseline information, a saturated model was conducted on the entire sample (e.g., collapsed across genotype). Second, because our moderator variable was dichotomous, a multiple group structural equation model was conducted using *DRD4* genotype (7− vs. 7+) as the grouping variable (see Cortina, Chen, & Dunlap, 2001). Paths that were nonsignificant in both groups were trimmed from the model. Because hypotheses stemming from DST intrinsically involve moderation, paths that were significant in only one or both groups were retained. Thus, in the third step the final trimmed model was analyzed. Chi-square difference tests were used to determine group differences. Moderated paths were further tested for reliability through bootstrapping standard errors (n = 1,000 draws) and constructing 95% confidence intervals (CIs). Bootstrapped CIs estimate the population distribution of possible unstandardized parameter estimates based on n random draws from the analysis sample (see Mooney & Duval, 1993). Parameter reliability is evidenced when the bootstrapped 95% CI does not include zero. Last, bootstrapping was similarly implemented to test indirect effects (see MacKinnon, Lockwood, & Williams, 2004).

Results

Preliminary Analyses

To test variance/covariance equivalence, we conducted a multivariate analysis of variance using all model variables as dependent variables (parent and child interparental conflict and positivity, threat appraisal, internalizing problems) and *DRD4* status as the fixed effect. Box's M was statistically significant (M = 36.10; F(21, 270,897) = 1.69, p < .05) indicating overall (or omnibus) heterogeneity in variances/covariance between genotypes. As an additional check, we conducted a second multivariate analysis of variance similar to the first but substituting PROSPER intervention condition (0 = control, 1 = intervention) as the fixed effect. Box's M was not statistically significant (M = 29.82), F(21, 367,153) = 1.40, *ns*. This result suggests the current findings are not conditioned by intervention participation.

Primary Analyses

Step 1: Saturated model—The results of the baseline saturated model can be seen in Figure 1. Noteworthy were the null associations between internalizing problems and all conflict and positivity measures; these four effects ranged from β = .00 to .09. Threat appraisals during seventh grade prospectively affected internalizing problems in eighth grade (β = .24), however. In addition, both couple conflict (β = .22) and adolescent perceived interparental conflict (β = .21) at sixth grade significantly affected threat appraisals in the seventh grade, consistent with previous research (Fosco & Grych, 2008; Grych, et al., 2003). Neither couple positivity nor adolescent perceived interparental positivity were associated with threat appraisals (βs = .03 and −.02, respectively).

Step 2: Multiple group model—Multiple group structural equation modeling grouped by *DRD4* genotype (7− vs. 7+) was applied to the model depicted in Figure 1. The path from

adolescent perceived interparental positivity to threat was significant for 7+ adolescents (β = −.21, $p < .05$) but not for 7− adolescents (β = .06, *ns*). All other nonsignificant paths remained nonsignificant across genotypes and were set to zero (i.e., dropped). This trimmed model was then reestimated for the full sample (collapsed across genotypes) and revealed good fit to the data, $\chi^2(5) = 5.54$, *ns*; CFI = 1.00; TLI = 1.00; RMSEA = .02; SRMR = .02, suggesting that dropping these paths from the saturated model did not worsen model fit. Last, we reanalyzed the saturated multiple group model and constrained the paths that were dropped in the trimmed model to be equal across groups. The resulting model showed good fit to the data, $\chi^2(5) = 3.32$, *ns*; CFI = 1.00; TLI = 1.00; SRMR = .02, indicating these paths were not genetically moderated.

Step 3: Trimmed multiple group model—The results of the final trimmed model are depicted in Figure 2. Overall, the trimmed multiple group model showed a good fit to the data, $\chi^2(10) = 9.87$, *ns*; CFI = 1.00; TLI = 1.00; RMSEA = .00; SRMR = .03. Individual directional paths were constrained be equal across groups and resulted in significant model misfit, $\Delta\chi^2(8) = 15.56$, $p < .05$, suggesting differences across groups (also evidenced by the Box's *M* above). To describe results from the trimmed model, we will highlight first the direct effects and group differences in direct effects, followed by indirect effects.

Direct effects—Adolescents with either the *DRD4* 7− or 7+ genotype showed similar direct effects of couple conflict on threat appraisals (β = .21 and β = .20, respectively). Thus, irrespective of genotype, couple conflict influenced adolescents' appraisals of threat. In addition, couple conflict was positively associated with adolescent conflict perception for both genotypes (β = .32, β = .46, 7− and 7+, respectively). Similarly, couple positivity was associated with adolescent perception of positivity for both genotypes (β = .37, β = .23). Last, higher couple conflict was associated with lower adolescent perceived interparental positivity for adolescents of either genotype (β = −19, −.24). Only the 7− adolescents showed a significant relation between couple positivity and adolescent perceived interparental conflict (β = −.19); 7+ adolescents showed no association (β = −.06). The difference between these paths was not significant, however, $\Delta\chi^2(1) = 1.37$, *ns*.

Different pathways to threat emerged across genotypes. *DRD4* 7− adolescents were more affected by interparental conflict, whereas 7+ adolescents were affected by positivity. This difference is evidenced by the association between perceived interparental conflict and threat appraisals among 7− adolescents (β = .27; bootstrapped parameters: *b* = .36, 95% CI [. 16, .56]) that was absent among 7+ (β = .05; *b* = .06, 95% CI [−.20, .32]). These paths differed significantly across genotypes, $\Delta\chi^2(1) = 3.69$, $p = .05$. Furthermore, no association was found between perceptions of interparental positivity and threat for 7− adolescents (β = . 08; *b* = .13, 95% CI [−.09, .35]); this relation was present among 7+ adolescents (β = −.22; *b* = −.41, 95% CI [−.76, −.07]). These paths also differed significantly between genotypes, $\Delta\chi^2(1) = 6.78$, $p < .01$. Last, children of either genotype demonstrated greater internalizing problems in eighth grade when threat was higher in the seventh (β = .28 and β = .30, respectively).

Indirect effects—Tests of indirect effects and differences by genotype are provided in Table 2. We constrained pathway sets across groups to assess genotype differences.

Although positivity was not directly associated with threat appraisals among 7− adolescents, the indirect path (couple positivity → adolescent perception of interparental conflict → threat appraisal) was statistically significant (i.e., indirect path 4 in Table 2). A similar indirect association from couple conflict to threat appraisal was also found (see indirect path 1). In addition, the four-step pathway—couple conflict/positivity → adolescent perceived interparental conflict → threat appraisals → internalizing problems—was also significant for 7− adolescents (indirect paths 8 and 11). These results suggest that for 7− adolescents both couple conflict and positivity prospectively affect threat appraisals and internalizing problems. Nonetheless, couple relationships affect threat appraisals among 7− adolescents with primarily through perceived conflict. Although not different across genotypes, among 7− adolescents, the adolescent perceived interparental conflict → threat appraisals → internalizing problems path was significant (indirect path 5), as well was the couple conflict → threat appraisals → internalizing problems path (indirect path 7).

Among 7+ adolescents, interparental conflict and positivity affected threat appraisals and internalizing problems primarily through child perceived interparental positivity. For example, the couple conflict/positivity → adolescent perceived interparental positivity → threat appraisals paths (indirect paths 2 and 3 in Table 2), were marginally significant. In addition, these pathways to threat were significantly larger among 7+ adolescents compared to 7− adolescents (see Table 2). Furthermore, the adolescent perceptions of interparental positivity → threat appraisals → internalizing problems path also was significant (indirect path 6) and was significantly different from the path for the 7− group. These results suggest the primary pathway by which interparental relationships affect threat appraisals and internalizing problems among 7+ adolescents appears to be perceptions of interparental positivity rather than conflict per se. The couple conflict → threat appraisals → internalizing problems path was significant for both groups of adolescents (indirect path 7) and did not significantly differ. Last, none of the four-step pathways from couple conflict/positivity to internalizing problems (8, 9, 10, and 11) were significant among 7+ adolescents. Nonetheless, pathway 9 did differ across genotypes. The meaning of this difference is ambiguous given that neither indirect effect was significantly different from zero.

Discussion

This is the first study to apply DST and vantage sensitivity to research on interparental conflict and positivity, adolescents' appraisals of these interparental behaviors, and internalizing problems. In this inquiry we first tested a model in which couple conflict and positivity were linked to adolescents' perceptions of their parents' relationship, that was then related to their threat evaluations, and finally associated with risk for internalizing problems (Grych & Fincham, 1990). The second step was to test hypotheses regarding genetic moderation related to DST.

The preliminary model tested in the first set of analyses advanced existing work on cognitive-contextual models by expanding interparental relationship assessment to include positive exchanges. Consistent with our hypotheses, couple positivity and conflict were related to adolescents' perceptions of conflict and positivity in their parents' relationship. In turn, adolescents who perceived more frequent parental conflict were more likely to perceive

conflicts as threatening. Finally, adolescents who evaluated parental conflicts as threatening were more likely to report higher levels of internalizing problems 1 year later. These findings are consistent with prior work examining threat appraisals of interparental conflict and internalizing problems (e.g., Fosco & Grych, 2008; Fosco & Feinberg, in press; Grych et al., 2003)

Two findings in this model ran counter to our hypotheses. First, we expected that adolescents' interparental relationship perceptions would fully account for the association between interparental conflict and positivity and threat appraisals. Instead, both interparental conflict and adolescent perceived conflict were each uniquely associated with adolescent threat evaluations. It is possible the unidimensional (i.e., conflict frequency) measurement of adolescent perceptions of parental conflicts, that omit intensity and resolution of parents' arguments, may explain this result. Further research is needed to better understand this finding. Second, we expected that interparental positivity, independent of level of conflict, would be related to lower levels of perceived threat; however, no such association was found for the whole sample.

On the basis of DST and vantage sensitivity perspectives, we examined whether the *DRD4* 7-repeat genotype moderated association patterns linking interparental relationships to adolescent internalizing problems. Among adolescents who were *DRD4* 7+, but not for those who were 7−, perception of interparental positivity was linked to lower threat appraisals. Furthermore, 7+ adolescents were also relatively unaffected by interparental conflict compared to 7− adolescents. This result is contrary to DST, which posits sensitivity for both positivity and negativity, but it is consistent with vantage sensitivity (Pluess & Belsky, 2013). It is currently unclear, however, whether sensitivity due to *DRD4* is domain general or domain specific, although accumulating research points to domain specificity (e.g., Bakermans-Kranenburg & van IJzendoorn, 2011; Bakermans-Kranenburg, Van IJzendoorn, Mesman, et al., 2008; Belsky & Pluess, 2013; Knafo et al., 2011). These studies and the findings reported herein support the possibility that adolescents who are DRD4 7+ may be relatively more sensitive to environmental positivity than negativity, consistent with vantage sensitivity.

An opposite pattern emerged among 7− adolescents: Perceived interparental conflict was associated with higher levels of threat, but perceived positivity was not. These results clearly contradict hypotheses derived from both the DST and vantage sensitivity perspectives. It is important to note that DST and vantage sensitivity conceptualize environmental sensitivity as a matter of *degree* (plasticity) rather than as falling into discrete categories (sensitive vs. fixed). Even individuals who would be categorized as "fixed" can be influenced by sufficiently strong environmental exposures (e.g., intense interparental conflict; Ellis et al., 2011). These findings suggest that exposure to interparental conflict is sufficiently potent to affect 7− adolescents' threat appraisals, which is consistent with research highlighting interparental conflict as one of the most stressful social experiences children identify (Lewis, Siegel, & Lewis, 1984). However, additional studies are needed to explore these findings for 7− adolescents.

Last, threat appraisals were associated with higher risk for internalizing problems for both 7+ and 7− adolescents. This finding reaffirms the integral role threat appraisals play in understanding pathways of risk from interparental relationships to internalizing problems. The shared risk from threat appraisals also supports genotypic sensitivity, consistent with DST hypotheses. Sensitivity was shown specifically to environmental stimuli rather than altering the progression from threat appraisals to internalizing psychopathology.

Implications for Interparental Conflict, Threat Appraisals, and Child Adjustment

Researchers interested in understanding mechanisms by which interparental conflict is related to child adjustment have focused on child evaluations of conflict (Davies & Cummings, 1994; Davies & Sturge-Apple, 2007; Fosco et al., 2007; Grych & Fincham, 1990). The findings of this study advance this area of research by further integrating a biological perspective into models of interparental conflict and adolescent adjustment (e.g., Sturge-Apple, Cicchetti, Davies, & Suor, 2012). By incorporating variation in the *DRD4* allele we were able to ask questions about "for whom" perceptions of interparental conflict and positivity are salient for threat evaluations. Our findings are consistent with an equifinality conceptualization of risk, in which different pathways may lead to a single outcome (von Bertalanffy, 1968). These findings highlight the need to continue work delineating individual differences, both genotypic and phenotypic, that will illuminate unique pathways to key risk factors for maladjustment. Such work may require broadening environmental assessments (e.g., including positive interparental relations) and including other genetic factors (e.g., haplotypes, genetic risk scores, etc.).

Future work is needed to replicate our findings. In many domains of research, replication can be a problem and concern has also been raised with regard to in Gene × Environment research (e.g., Duncan & Keller, 2011). Without replication, the critique remains that a particular study may be unduly influenced by factors unrelated to what is being studied and thereby produce artifactual results. To bolster confidence in our results, we created bootstrapped 95% confidence intervals, which offer insight into reliability of the moderated paths through multiple sample draws. Random sample splits are considered a particularly strong replication method because of homogeneity of samples, measurement, and methods (e.g. Johnston, Lahey, & Matthys, 2013). In addition, our findings add to a growing literature replicating *DRD4* 7−/7+ environmental moderation (e.g., Bakermans-Kranenberg & van IJzendoorn, 2011), and most of these studies indicate 7+ carriers are relatively more sensitive to environmental positivity, as we have shown in this study (see Pluess & Belsky, 2013). Finally, our findings also are largely consistent with the theories they tested. Thus, although independent sample replication is desirable, our analytic techniques, reliance on theory to guide our analysis, and theoretically consistent findings provide confidence that our results are not unique to these data.

Limitations and Conclusion

The primary limitation of this study is the measurement of adolescents' perceptions of interparental positivity and conflict. Although they were predictive of threat, these measures clearly did not capture the multifaceted aspects of interparental relationships. Second, this

study was limited to a relatively homogeneous sample and may not generalize to other populations.

Despite these limitations, this study illustrates the value of studying unique pathways from interparental functioning to adolescent development. We have shown that interparental positivity may be one such pathway that has been little considered in the existing literature. Furthermore, integrating DST has shed light on questions that may not have otherwise been asked. Additional research should focus on replication of these findings, and additional factors associated with sensitivity should be considered.

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References

Achenbach, TM. Integrative guide for the 1991 CBCL/14-18, YSR and TRF Profiles. Burlington: University of Vermont, Department of Psychiatry; 1991.

Anchordoquy HC, McGeary C, Liu L, Krauter KS, Smolen A. Genotyping of three candidate genes after whole-genome preamplification of DNA collected from buccal cells. Behavioral Genetics. 2003; 33:73–78.

Asghari V, Sanyal S, Buchwaldt S, Paterson A, Jovanovic V, Van Tol HH. Modulation of intracellular cyclic AMP levels by different human dopamine D4 receptor variants. Journal of Neurochemistry. 1995; 65:1157–1165. [PubMed: 7643093]

Atkinson ER, Dadds MR, Chipuer H, Dawe S. Threat is a multidimensional construct: Exploring the role of children's threat appraisals in the relationship between interparental conflict and child adjustment. Journal of Abnormal Child Psychology. 2009; 37:281–292.10.1007/s10802-008-9275-z [PubMed: 18855133]

Bakermans-Kranenberg MJ, van IJzendoorn MH. Differential susceptibility to rearing environment depending on dopamine related genes: New evidence and meta-analysis. Development and Psychopathology. 2011; 23:39–52. [PubMed: 21262038]

Bakermans-Kranenburg MJ, Van IJzendoorn MH, Mesman J, Alink LRA, Juffer F. Effects of an attachment-based intervention on daily cortisol moderated by dopamine receptor D4: A randomized control trial on 1- to 3-year-olds screened for externalizing behavior. Development and Psychopathology. 2008; 20:805–820.10.1017/s0954579408000382 [PubMed: 18606032]

Bakermans-Kranenberg MJ, van IJzendoorn MH, Pijlman FTA, Mesman J, Juffer F. Experimental evidence for differential susceptibility: Dopamine D4 receptor polymorphism (*DRD4 VNTR*) moderates intervention effects on toddlers' externalizing behavior in a randomized controlled trial. Developmental Psychology. 2008; 44:293–300. [PubMed: 18194028]

Beach SRH, Brody GH, Lei M, Philibert RA. Differential susceptibility to parenting among African American youths: Testing the *DRD4* hypothesis. Journal of Family Psychology. 2010; 24:513–521.10.1037/a0020835 [PubMed: 20954761]

Belsky J, Pluess M. Beyond diathesis stress: Differential susceptibility to environmental influences. Psychological Bulletin. 2009; 135:885–908.10.1037/a0017376 [PubMed: 19883141]

Belsky J, Pluess M. Genetic moderation of early childcare effects on social functioning across childhood: A developmental analysis. Child Development. 2013; 84:1209–1225.10.1111/cdev.12058 [PubMed: 23432522]

von Bertalanffy, L. Organismic psychology and systems theory. Barre, MA: Clark University Press; 1968.

Boyce WT, Ellis BJ. Biological sensitivity to context: I. An evolutionary–developmental theory of the origins and functions of stress reactivity. Development and Psychopathology. 2005; 17:271–301.10.1017/s0954579405050145 [PubMed: 16761546]

Buehler C, Anthony C, Krishnakumar A, Stone G, Gerard J, Pemberton S. Interparental conflict and youth problem behaviors: A meta-analysis. Journal of Child and Family Studies. 1997; 6:233–247.

Cortina JM, Chen G, Dunlap WP. Testing interaction effects in LISREL: Examination and illustration of available procedures. Organizational Research Methods. 2001; 4:324–360.10.1177/109442810144002

Cummings EM, Cummings JL. A process-oriented approach to children's coping with adults' angry behavior. Developmental Review. 1988; 8:296–321.

Cummings, EM.; Davies, P. Children and marital conflict: The impact of family dispute and resolution. New York: Guilford Press; 1994.

Cummings EM, George MRW, McCoy KP, Davies PT. Interparental conflict in kindergarten and adolescent adjustment: Prospective investigation of emotional security as an explanatory mechanism. Child Development. 2012; 83:1703–1715.10.1111/j.1467-8624.2012.01807.x [PubMed: 22694264]

Davies PT, Cummings EM. Marital conflict and child adjustment: An emotional security perspective. Psychological Bulletin. 1994; 116:387–411. [PubMed: 7809306]

Davies PT, Cummings EM. Exploring children's emotional security as a mediator of the link between marital relations and child adjustment. Child Development. 1998; 69:124–139. [PubMed: 9499562]

Davies PT, Harold GT, Goeke-Morey M, Cummings EM. Child emotional security and interparental conflict. Monographs of the Society for Research in Child Development. 2002; 67(3 Serial No 270)

Davies PT, Lindsay LL. Interparental conflict and adolescent adjustment: Why does gender moderate early adolescent vulnerability? Journal of Family Psychology. 2004; 18:160–170.10.1037/0893-3200.18.160 [PubMed: 14992618]

Davies PT, Martin MJ, Cicchetti D. Delineating the sequelae of destructive and constructive interparental conflict for children within an evolutionary framework. Developmental Psychology. 2012; 48:939–955.10.1037/a0025899 [PubMed: 22004336]

Davies, PT.; Sturge-Apple, ML. Advances in the formulation of emotional security theory: An ethologically-based perspective. In: Kail, RV., editor. Advances in child behavior and development. Vol. 35. New York: Elsevier; 2007. p. 87-137.

DeBoard-Lucas RL, Fosco GM, Raynor SR, Grych JH. Interparental conflict in context: Exploring relations between parenting processes and children's conflict appraisals. Journal of Clinical Child & Adolescent Psychology. 2010; 39:163–175.10.1080/15374410903532593 [PubMed: 20390808]

D'Onofrio BM, Lahey BB. Biosocial influences on the family: A decade in review. Journal of Marriage and Family. 2010; 72:762–782.10.1111/j.1741-3737.2010 [PubMed: 24009400]

Duncan LE, Keller MC. A critical review of the first 10 years of candidate Gene-by-Environment interaction research in psychiatry. The American Journal of Psychiatry. 2011; 168:1041–1049. [PubMed: 21890791]

Ellis BJ, Boyce WT, Belsky J, Bakermans-Kranenburg MJ, van IJzendoorn MH. Differential susceptibility to the environment: An evolutionary–neurodevelopmental theory. Development and Psychopathology. 2011; 23:7–28. [PubMed: 21262036]

Emery RE. Interparental conflict and the children of discord and divorce. Psychological Bulletin. 1982; 92:310–330. [PubMed: 7146231]

Faraone SV, Doyle AE, Mick E, Biederman J. Meta-analysis of the association between the 7-repeat allele of the dopamine D_4 receptor gene and attention deficit hyperactivity disorder. American Journal of Psychiatry. 2001; 158:1052–1057. [PubMed: 11431226]

Fosco GM, DeBoard RL, Grych JH. Making sense of family violence: Implications of children's appraisals of interparental aggression for their short-term and long-term functioning. The European Psychologist. 2007; 12:6–16.

Fosco GM, Feinberg ME. Cascading effects of interparental conflict in adolescence: Linking threat appraisals, self-efficacy, and adjustment. Development and Psychopathology. 2014 Advance online publication. 10.1017/S0954579414000704

Fosco GM, Grych JH. Emotional expression in the family as a context for children's appraisals of interparental conflict. Journal of Family Psychology. 2007; 21:248–258. [PubMed: 17605547]

Fosco GM, Grych JH. Emotional, cognitive, and family systems mediators of children's adjustment to interparental conflict. Journal of Family Psychology. 2008; 22:843–854. [PubMed: 19102605]

Fosco GM, Grych JH. Adolescent triangulation into parental conflicts: Longitudinal implications for appraisals and adolescent–parent relations. Journal of Marriage and Family. 2010; 72:254–266.10.1111/j.1741-3737.2010.00697.x

Freeman B, Smith N, Curtis C, Huckett L, Mill J, Craig IW. DNA from buccal swabs recruited by mail: Evaluation of storage effects on long-term stability and suitability for multiplex polymerase chain reaction genotyping. Behavioral Genetics. 2003; 33:67–72.

Goeke-Morey MC, Cummings EM, Harold GT, Shelton KH. Categories and continua of destructive and constructive marital conflict tactics from the perspective of U.S. and Welsh children. Journal of Family Psychology. 2003; 17:327–338. [PubMed: 14562457]

Grych JH, Fincham FD. Marital conflict and children's adjustment: A cognitive-contextual framework. Psychological Bulletin. 1990; 108:267–290. [PubMed: 2236384]

Grych JH, Fincham FD. Children's appraisals of interparental conflict: Initial investigations of the cognitive-contextual framework. Child Development. 1993; 64:215–230. [PubMed: 8436030]

Grych JH, Harold GT, Miles CJ. A prospective investigation of appraisals as mediators of the link between interparental conflict and child adjustment. Child Development. 2003; 74:1176–1193. [PubMed: 12938712]

Grych JH, Jouriles EN, Swank PR, McDonald R, Norwood WD. Patterns of adjustment among children of battered women. Journal of Consulting and Clinical Psychology. 2000; 68:84–94. [PubMed: 10710843]

Grych JH, Seid M, Fincham FD. Assessing marital conflict from the child's perspective: The Children's Perceptions of Interparental Conflict Scale. Child Development. 1992; 63:558–572. [PubMed: 1600822]

Harold GT, Conger RD. Marital conflict and adolescent distress: The role of adolescent awareness. Child Development. 1997; 68:333–350. [PubMed: 9180005]

Harold GT, Fincham FD, Osborne LN, Conger RD. Mom and Dad are at it again: Adolescent perceptions of marital conflict and adolescent psychological distress. Developmental Psychology. 1997; 33:333–350. [PubMed: 9147841]

Horwitz BN, Neiderhiser JM. Gene–environment interplay, family relationships, and child adjustment. Journal of Marriage and Family. 2011; 73:804–816. [PubMed: 22162877]

Hu L, Bentler PM. Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. Structural Equation Modeling. 1999; 6:1–55.10.1080/10705519909540118

Johnston C, Lahey BB, Matthys W. Editorial policy for candidate gene studies. Journal of Abnormal Child Psychology. 2013; 41:511–514.10.1007/s10802-013-9741-0

Kluger AN, Siegfried Z, Ebstein RP. A meta-analysis of the association between *DRD4* polymorphism and novelty seeking. Molecular Psychiatry. 2002; 7:712–717.10.1038/si.mp.4001082 [PubMed: 12192615]

Knafo A, Israel S, Ebstein RP. Heritability of children's prosocial behavior and differential susceptibility to parenting by variation in the dopamine receptor *D4* gene. Development and Psychopathology. 2011; 23:53–67.10.1017/s0954579410000647 [PubMed: 21262039]

Lengua LJ, Long AC. The role of emotionality and self-regulation in the appraisal–coping process: Tests of direct and moderating effects. Journal of Applied Developmental Psychology. 2002; 23:471–493.

Lewis CE, Seigel JM, Lewis MA. Feeling bad: Exploring sources of distress among pre-adolescent children. American Journal of Public Health. 1984; 74:117–122. [PubMed: 6691520]

Lichter JB, Barr CL, Kennedy JL, Van Tol HH, Kidd KK, Livak KJ. A hypervariable segment in the human dopamine receptor *D4* (*DRD4*) gene. Human Molecular Genetics. 1993; 2:767–773. [PubMed: 8353495]

MacKinnon DP, Lockwood CM, Williams J. Confidence limits for the indirect effect: Distribution of the product and resampling methods. Multivariate Behavioral Research. 2004; 39:99–128.10.1207/s15327906mbr3901_4 [PubMed: 20157642]

McCoy K, Cummings EM, Davies PT. Constructive and destructive marital conflict, emotional security, and children's prosocial behavior. Journal of Child Psychology and Psychiatry. 2009; 50:270–279. doi:101111/j.1469*-7610.2008.01945.x. [PubMed: 18673403]

Mooney, CZ.; Duval, RD. Bootstrapping: A nonparametric approach to statistical inference. Thousand Oaks, CA: Sage; 1993.

Muthén, LK.; Muthén, BO. Mplus User's Guide. 6. Los Angeles, CA: Muthén & Muthén; 1998–2012.

Oak JN, Oldenhof J, Van Tol HHM. The dopamine *D4* receptor: One decade of research. European Journal of Pharmacology. 2000; 405:303–327. [PubMed: 11033337]

Pluess M, Belsky J. Vantage sensitivity: Individual differences in response to positive experiences. Psychological Bulletin. 2013; 139:901–916.10.1037/a0030196 [PubMed: 23025924]

Pluess, M.; Stevens, SE.; Belsky, J. Differential susceptibility: Developmental and evolutionary mechanisms of gene–environment interactions. In: Legerstee, M.; Haley, DW.; Bornstein, MH., editors. The infant mind: Origins of the social brain. New York: Guilford Press; 2013.

Rhoades KA. Children's responses to interparental conflict: A meta-analysis of their associations with child adjustment. Child Development. 2008; 79:1942–1956. [PubMed: 19037959]

Rowe DC. Environmental and genetic influences on dimensions of perceived parenting: A twin study. Developmental Psychology. 1981; 17:203–208.

Rowe DC. A biometrical analysis of perceptions of family environment: A study of twin and singleton sibling kinships. Child Development. 1983; 54:416–423. [PubMed: 6683621]

Rowe DC, Plomin R. Temperament in early childhood. Journal of Personality Assessment. 1977; 41:150–156. [PubMed: 856967]

Schlomer GL, Bauman S, Card NA. Best practices for missing data management in counseling psychology. Journal of Counseling Psychology. 2010; 57:1–10.10.1037/a0018082 [PubMed: 21133556]

Schoots O, Van Tol HH. The human dopamine *D4* receptor repeat sequences modulate expression. Pharmacogenomics Journal. 2003; 3:343–348. [PubMed: 14581929]

Simons RL, Simons LG, Lei M, Beach SRH, Brody GH, Gibbons FX, Philibert RA. Genetic moderation of the impact of parenting on hostility toward romantic partners. Journal of Marriage and Family. 2013; 75:325–341.10.1111/jomf.12010 [PubMed: 24379481]

Spoth R, Greenberg M, Bierman K, Redmond C. PROSPER community–university partnership model for public education systems: Capacity-building for evidence-based, competence-building prevention. Prevention Science. 2004; 5:31–39. [PubMed: 15058910]

Sturge-Apple ML, Cicchetti D, Davies PT, Suor JH. Differential susceptibility in spillover between interparental conflict and maternal parenting practices: Evidence for *OXTR* and *5-HTT* genes. Journal of Family Psychology. 2012; 26:431–442.10.1037/a0028302 [PubMed: 22563705]

Wong AH, Buckle CE, Van Tol HH. Polymorphisms in dopamine receptors: What do they tell us? European Journal of Pharmacology. 2000; 410:183–203. [PubMed: 11134669]

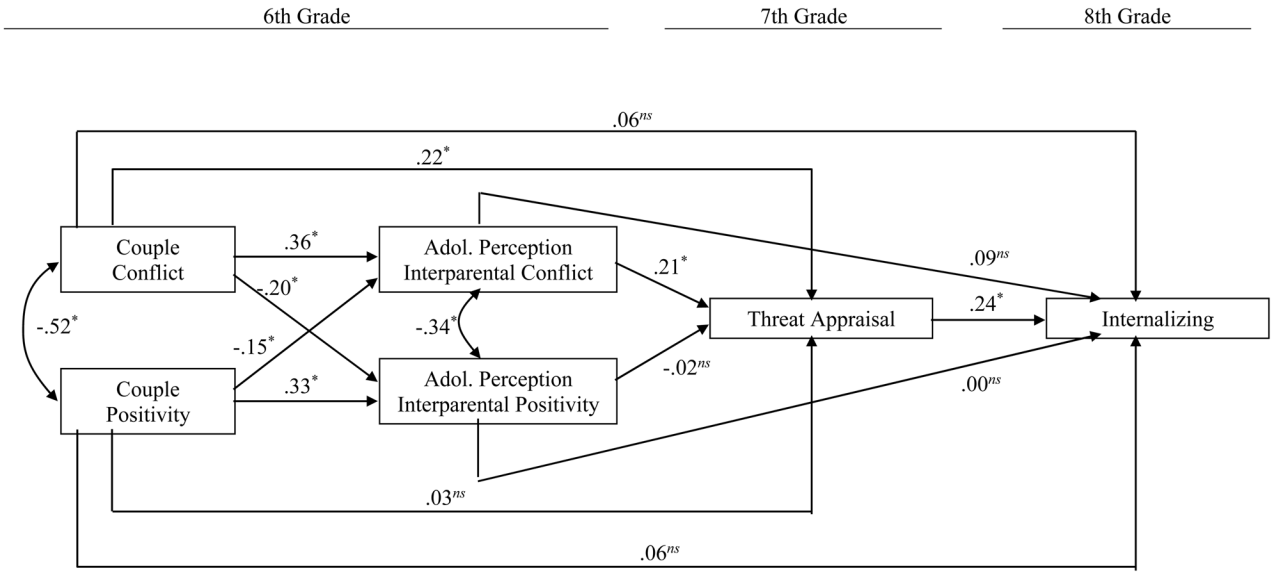

Figure 1. Results of the Saturated Model Collapsed Across Genotypes

Note. Adol. = Adolescent; *ns* = not significant. *$p < .05$.

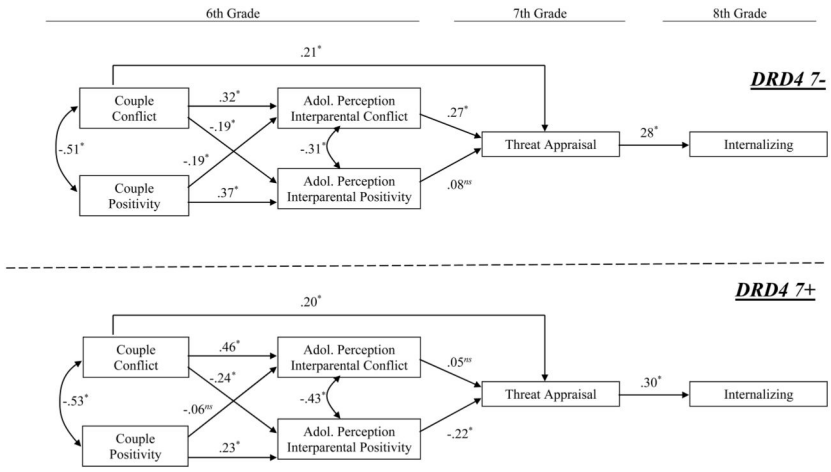

Figure 2. Results of the Trimmed Multiple Group Structural Equation Modeling by *DRD4* Genotype

Note. $\chi^2(10) = 9.87$, *ns;* comparative fit index = 1.00; root-mean-square error of approximation = .00; 95% confidence interval [.00, .07]. Adol. = Adolescent; *ns* = not significant. *$p < .05$.

Table 1

Summary of Intercorrelations, Means, and Standard Deviations for Measures Used in Path Models

Measure	1	2	3	4	5	6	7
1. Couple conflict	—						
2. Couple positivity	−.52**	—					
3. Adolescent's perception of interpersonal positivity	.44**	−.33**	—				
4. Adolescent's perception of interparental positivity	−.37**	.43**	−.47**	—			
5. Threat appraisal	.30**	−.16**	.31**	−.19**	—		
6. Internalizing problems	.19**	−.06	.19**	−.17**	.34**	—	
7. *DRD4* 7-repeat	−.01	.01	−.05	.05	.02	−.08	—
M	2.04	5.36	2.44	4.39	2.15	0.20	
SD	0.70	1.02	0.85	0.68	1.10	0.20	

** $p < .01$.

Table 2

Genetically Moderated Indirect Effects

Indirect path	*DRD4* 7−		*DRD4* 7+		$\chi^2(df)$
	Indirect effect	Total effect	Indirect effect	Total effect	
1. C-Conflict → APIC → TA	**.087***	.297	.021	.221	5.74(2)
2. C-Positivity → APIP → TA	.031	.031	**−.050†**	−.050	**11.13(2)***
3. C-Conflict → APIP → TA	−.016	−.016	**.053†**	.053	**6.79(2)***
4. C-Positivity → APIC → TA	**−.052***	−.051	−.003	−.003	5.00(2)
5. APIC → TA → Internalizing	**.075***	.075	.014	.014	3.70(2)
6. APIP → TA → Internalizing	.023	.023	**−.066***	−.066	**6.79(2)***
7. C-Conflict → TA → Internalizing	**.058***	.058	**.060***	.060	.02(2)
8. C-Conflict → APIC→ TA → Internalizing	**.024***	.024	.006	.006	5.75(3)
9. C-Positivity→ APIP → TA → Internalizing	.009	.009	−.015	−.015	**11.13(3)***
10. C-Conflict → APIP → TA → Internalizing	−.004	−.004	.016	.016	6.80(3)
11. C-Positivity → APIC → TA → Internalizing	**−.014***	−.014	−.001	−.001	5.00(3)

Note. Statistically significant values are in boldface type. C-Conflict/Positivity = couple conflict/positivity; APIC/P = adolescent perceived interparental conflict/positivity; TA = threat appraisals; $\Delta\chi^2$ = increase from between-group constraints. Critical $\chi^2(2) = 5.99$, $\chi^2(3) = 7.82$.

† $p \leq .10$.

* $p < .05$.