J Youth Adolescence (2015) 44:1441–1456
DOI 10.1007/s10964-015-0304-2

EMPIRICAL RESEARCH

Externalizing Problem Behavior in Adolescence: Dopaminergic Genes in Interaction with Peer Acceptance and Rejection

Annelies Janssens[1,2] · Wim Van Den Noortgate[3] · Luc Goossens[2] · Karine Verschueren[2] · Hilde Colpin[2] · Steven De Laet[2] · Stephan Claes[4] · Karla Van Leeuwen[1]

Received: 25 March 2015 / Accepted: 13 May 2015 / Published online: 26 May 2015

Abstract Molecular gene-by-environment studies primarily have focuses on the parent–child relationship as an environmental factor, whereas studies including peer relationships as environmental factor are rare. However, the effects of the peer context may not be the same for all adolescents due to biological characteristics. This study examined whether the effects of peer rejection and acceptance on externalizing behavior depend upon adolescents' genotype for the dopamine transporter (DAT1) or receptor D4 (DRD4) gene. In a sample of 563 adolescents (52 % girls; *M* age = 13.81), saliva samples, within-classroom peer nominations, and multi-informant behavior ratings were collected. Peer rejection, but not acceptance, was associated with externalizing problems. One out of eight models tested for rule-breaking behavior showed genetic moderation. According to the Roisman criteria, there was evidence for the differential susceptibility hypothesis. DAT1 10R carriers showed more rule-breaking behavior according to parents when experiencing high peer rejection, but less rule-breaking behavior when experiencing low peer rejection. The long DRD4 variant was associated with less aggression, but no moderation effects were found. The results are discussed in light of the differential susceptibility hypothesis and the reward sensitivity mechanism.

Keywords Adolescence · Gene-by-environment interaction · Externalizing problem behavior · Peer relationships · Dopaminergic genes

✉ Annelies Janssens
Annelies.Janssens@ppw.kuleuven.be

[1] Parenting and Special Education Research Unit, Faculty of Psychology and Educational Sciences, KU Leuven, Leopold Vanderkelenstraat 32, Box 3765, 3000 Leuven, Belgium

[2] School Psychology and Child and Adolescent Development, Faculty of Psychology and Educational Sciences, KU Leuven, Tiensestraat 102, Box 3717, 3000 Leuven, Belgium

[3] Methodology of Educational Sciences, Faculty of Psychology and Educational Sciences, KU Leuven, Tiensestraat 102, Box 3762, 3000 Leuven, Belgium

[4] Research Group Psychiatry, Department of Neurosciences, KU Leuven, UZ Herestraat 49, Box 7003 37, 3000 Leuven, Belgium

Introduction

High prevalence of externalizing problem behavior in adolescence, such as delinquency and aggression, is an important concern in most western societies (e.g., Reitz et al. 2005). Externalizing behaviors have both immediate and long-term adverse consequences for the individual, the immediate environment such as family and friends, and wider society, as these problems can lead to addiction, impaired family relationships, decreased educational and occupational attainment, and continuing criminal activity (Huesmann et al. 2009; Reef et al. 2011). For decades, scholars have examined the biological and social antecedents of externalizing behaviors, but the interplay between both kinds of factors has gained much interest in the past 20 years (Bronfenbrenner and Morris 2006; Rutter 2006). Research on molecular gene–environment interactions (G × E) examines whether individuals' behavior in response to certain environmental influences can be predicted by specific genetic variants. So far, most G × E research has focused on the parent–child relationship as either a supportive or a detrimental environment (e.g., Bakermans-Kranenburg and van IJzendoorn 2011; Beach et al. 2010). However, in order to gain a better understanding of G × E processes, it is necessary to expand the focus to other environmental contexts, such as peer interactions. Adolescents spend an

increasing amount of time with peers. These peer interactions play an essential role in adolescents' identity formation and have important rewarding value (Waas 2006). However, activities with peers may also create risks, as they are less closely monitored by adults than in childhood (Parker et al. 2006). Adolescents experiencing a negative peer environment are at greater risk for adverse developmental outcomes, whereas positive peer relations seem to stimulate adequate development and buffer against problematic behavior (Rubin et al. 2006).

However, effects of the peer environment may not be the same for all adolescents. Biological characteristics could determine the degree to which a person is influenced by the environment. Evidence suggests that children and adolescents with inadequate self-regulation or heightened reward sensitivity are more susceptible to negative peer influences (Gardner et al. 2008; Goodnight et al. 2006). Genes regulating dopamine neurotransmission have been associated repeatedly with excessive reward sensitivity and externalizing problems (e.g., Blum et al. 2011; Dreher et al. 2009). Two of the most well-known dopaminergic polymorphisms are the dopamine transporter (DAT1) and dopamine receptor D4 (DRD4) variable number tandem repeats (VNTRs). In the present study, we examined whether these polymorphisms moderate the effects of peer rejection and acceptance by classmates on adolescent externalizing problem behavior.

Peer Relationships and Externalizing Behavior

Relationships with peers provide a powerful context to observe and acquire social skills involving communication, cooperation, and emotion regulation (Waas 2006). An important aspect of peer experiences is the degree to which one is liked or accepted versus disliked or rejected by the peer group. Peer acceptance determines the accessibility of peer learning opportunities and is an important source for feelings of self-esteem and belongingness (Baumeister and Leary 1995). Especially during adolescence, when identity is developing and peers become important socializing agents, being accepted by peers is a highly aspired psychosocial goal. In contrast, the experience of being rejected by peers is a salient source of psychosocial stress for adolescents (Masten et al. 2009). Acceptance and rejection are independent dimensions of peer status, typically showing negative and moderate correlations (e.g., $r = -.30$ for girls and $r = -.42$ for boys) (Sentse et al. 2010). These constructs are often combined in a social preference score (i.e., the difference between the number of positive and negative nominations) or five social status categories (i.e., popular, rejected, neglected, controversial, and average) (Gifford-Smith and Brownell 2003). In the present study, we chose to examine acceptance and rejection separately in order to differentiate between positive and negative peer influences and to take advantage of the continuity of our data.

Multiple studies have indicated that peer acceptance and peer rejection independently predict problem behavior (e.g., Dishion et al. 2005; Veronneau and Dishion 2010). Although problem behavior itself can contribute to peer rejection (Gifford-Smith and Brownell 2003), experiences of being disliked or rejected by peers have been shown to increase risk for negative developmental outcomes such as low academic performance (e.g., Gorman et al. 2011), physical health problems (e.g., Brendgen and Vitaro 2008), internalizing problems like loneliness and depression (e.g., Kiesner 2002; Vanhalst et al. 2014), and externalizing problem behavior (e.g., Gorman et al. 2011; Laird et al. 2001). These problems may arise from the lack of positive learning opportunities due to the inaccessibility of well-adjusted peers and the tendency to become friends with other less adjusted peers who may model deviant behaviors (Light and Dishion 2007). Experiences of being liked or accepted by peers, on the other hand, have been associated with more prosocial behavior, high academic competence, and low levels of loneliness (e.g., Gorman et al. 2011). Further, being accepted by peers seems to be a protective factor in relation to externalizing or internalizing problems (e.g., Grills-Taquechel et al. 2010; Sentse et al. 2010). In contrast, some studies have found that being accepted by peers can also enhance problem behavior (e.g., Allen et al. 2005; Veronneau and Dishion 2010). These studies suggest that adolescents experiencing high levels of peer acceptance may be more encouraged by peers to experiment with minor rule-breaking behavior.

DAT1 and DRD4 Genes and Externalizing Behavior

Dopamine functionality in the human brain is regulated by genes such as DAT1 and DRD4, which contain well-known variable number tandem repeat (VNTR) polymorphisms. A VNTR is a location in the genome where a short sequence of basic units is repeated several times (Haddley et al. 2008). The number of repeats, which differs among individuals, influences the expression of the gene and therefore dopamine functionality. The 40 base pair (bp) VNTR in the DAT1 gene is most commonly repeated 10 or 9 times (10R or 9R) (Mitchell et al. 2000). It remains unclear which variant promotes the most adequate gene expression levels (see Haddley et al. 2008, for a review). Nevertheless, researchers have found a direct association with externalizing behavior. Young et al. (2002) showed more externalizing problems in children with the 9R allele, whereas later studies on adolescent and adult samples suggest the 10R allele as a risk factor for problem behavior (e.g., Blum et al. 2011; Burt and Mikolajewski 2008; Guo et al. 2007; Lee et al. 2007).

The most common variants of the 48 bp VNTR in the DRD4 gene are the 2-repeat, 4-repeat, and 7-repeat alleles (Chang et al. 1996). Shorter variants have been found to code for a more efficient gene compared to longer 7R

alleles (Ebstein 2006). Longer DRD4 genotypes have been associated with more aggression in children (Farbiash et al. 2014; Schmidt et al. 2002), more externalizing behavior and less delinquency abstention in adolescents (Boutwell and Beaver 2008; Hohmann et al. 2009), and higher delinquency, short temper, and thrill seeking in adult males (Dmitrieva et al. 2011). However, a recent meta-analysis by Vassos et al. (2014) on aggression and related traits found no support for an association with DRD4.

Most evidence seems to suggest a higher prevalence of externalizing problems in DAT1 10R carriers and DRD4 7R carriers, but current findings are mixed. Reviews and meta-analyses have pointed to the high heterogeneity between studies (e.g., measurement, population), a lack of power, and publication bias (e.g., Ebstein 2006; Padmanabhan and Luna 2014; Vassos et al. 2014). Since the effects of single genetic polymorphisms on complex behaviors, such as externalizing problems, are known to be small, future research should use larger sample sizes to increase power, use consistent conceptualization and reliable measurement of complex behaviors, and include gene-by-environment or gene-by-gene interactions (Vassos et al. 2014).

Alternative Hypotheses on Gene–Environment Interactions

Molecular gene-by-environment (G × E) research has known a spectacular increase during the past 20 years. Different hypotheses have been formulated according to which genetic moderation of environmental effects can occur. The most well-known hypotheses are diathesis stress, vantage sensitivity, and differential susceptibility (Pluess and Belsky 2013). Most early G × E research is in line with the classic dual-risk or diathesis–stress hypothesis (see Fig. 1a), which implies that the combination of genetic and environmental risk (i.e., dual risk) increases problem behavior. In the absence of environmental adversity, there will be no difference between individuals carrying the concerned genetic variants (further called carriers) and resilient non-carriers. A more recently formulated vantage sensitivity hypothesis (see Fig. 1b) focuses on the 'bright side' of environmental susceptibility (Pluess and Belsky 2013). Carriers are more sensitive than non-carriers exclusively to positive experiences and not to adverse experiences. Combining both hypotheses, the differential susceptibility hypothesis (see Fig. 1c) suggests that carriers are not just vulnerable to adversity or sensitive to advantage, but more generally susceptible to all environmental influences (Bakermans-Kranenburg and van IJzendoorn 2011; Belsky 2005). In other words, this hypothesis encompasses both the "dark side" of susceptibility to negative experiences and the "bright side" of susceptibility to positive experiences (Bakermans-Kranenburg and van IJzendoorn 2011). Roisman et al. (2012) have criticized previously used methods to distinguish differential susceptibility from diathesis stress models and provided concrete solutions for evaluating G × E hypotheses. We will follow these suggestions when analyzing the data.

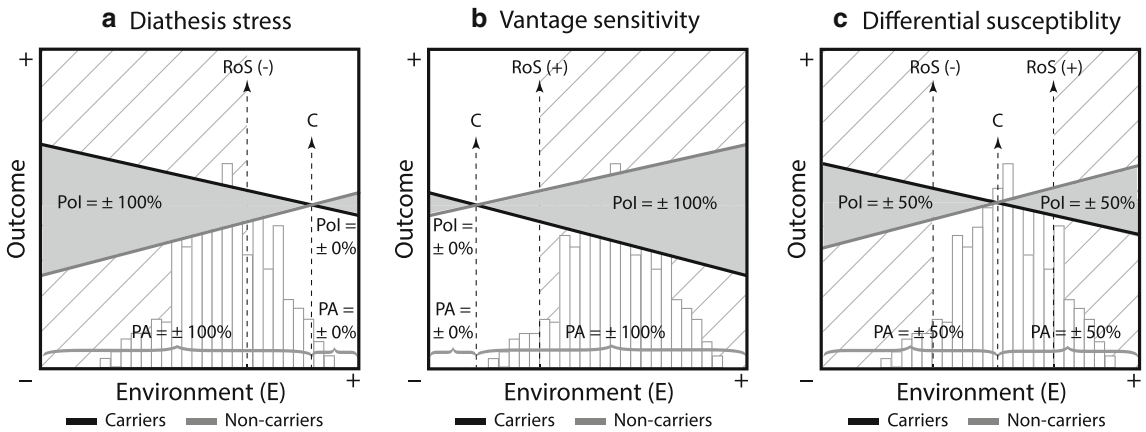

Fig. 1 Schematic representation of Roisman criteria to evaluate G × E hypotheses. Diathesis stress (model **a**) is supported if only a negative Region of Significance [RoS(−)] is found and the crossover point (*C*) is situated at the positive side of the environmental variable (*E*). This indicates that the association between gene and outcome is statistically significant only for a negative *E*. Further, two indexes are calculated on each side of *C*: the proportion of the interaction between the regression lines (PoI; Proportion of Interest) and the proportion of cases on *E* (PA; Proportion of Affected). Whereas PoI is dependent upon how ranges of *E* are defined, PA provides a more pragmatic way for evaluation based on the raw data. In case of diathesis stress, both indexes are close to 0 % at the positive side of *C*. When these criteria show an opposite pattern, they provide evidence for vantage sensitivity (model **b**). Finally, differential susceptibility (model **c**) is supported when boundaries of both positive and negative RoS as well as *C* fall within the range of *E*, indicating a significant association between gene and outcome at both ends of *E*. PoI and PA are closer to 50 % on both sides of *C*. In order to speak of differential susceptibility, Roisman et al. (2012) suggest a threshold for PA of 16 % above *C*

Genetic Moderation of Peer Effects on Externalizing Behavior

Despite the developmental impact of peers, peer relationships have often been neglected in G × E research (Brendgen 2012). Most G × E studies on externalizing problems have been focusing on the home environment. For the DRD4 gene, these studies have supported differential susceptibility: individuals with the DRD4 long or 7R allele were more susceptible to both low and high quality parenting (e.g., Bakermans-Kranenburg and van IJzendoorn 2011; Beach et al. 2010). The DAT1 gene is less frequently studied with regard to externalizing problems, but studies on ADHD samples seem to suggest that 9R carriers are more susceptible to the quality of parental expressed emotion (Sonuga-Barke et al. 2009) and observed maternal parenting (Lahey et al. 2011), resulting in more or less conduct problems. Like parents, peers can serve as either a risk or protective factor in adolescent problem behavior (Sentse et al. 2010). To gain a deeper understanding of G × E processes, we need to expand the focus to other environmental contexts besides the family, such as peer interactions.

The few molecular genetic studies examining genetic moderation of peer effects by the DRD4 or DAT1 genes show mixed results. In line with the diathesis stress hypothesis, peers' drinking behavior had stronger effects on alcohol use among adults carrying the DRD4 long or 7R allele, but not in adolescents (e.g., Larsen et al. 2010; Mrug and Windle 2014). Another study on the effect of substance-using peers found evidence for differential susceptibility (Watts and McNulty 2015). Adolescents carrying two DAT1 10R alleles, compared to non-carriers, were more likely to engage in criminal behavior when affiliating with substance-using peers, but less likely when affiliations with such peers were minimal or absent. Contrary to expectations, adolescents with the DRD4 4R allele, not the 7R allele, were more susceptible to the effects of peer victimization and social well-being, resulting in more or less delinquent behavior (Kretschmer et al. 2013). Further, two studies on affiliation with deviant peers found a suppressing effect indicating that the influence of genes is reduced by the presence of a specific environmental condition. The DRD4 long allele and the DAT1 10R allele had no effect for, respectively, preschoolers and adolescents interacting with highly aggressive or delinquent peers (DiLalla et al. 2009; Vaughn et al. 2009). The effect was only present in a low aggressive or delinquent peer environment. We intend to expand on these findings by examining the moderating role of DAT1 and DRD4 on the effect of peer acceptance and rejection on externalizing problems. So far, only one molecular G × E study has examined this association; the focus was on moderation by the serotonin transporter polymorphism (5-HTTLPR). Peer rejection predicted antisocial behavior more strongly for adolescents carrying the susceptible short–short allele, suggesting evidence for diathesis stress (Kretschmer et al. 2014). No effects were found for peer acceptance.

The Present Study

In the present study, we examined the effects of rejection and acceptance by classmates on adolescent externalizing problem behavior and moderation of these effects by adolescents' genotype for the DAT1 or DRD4 gene. With regard to main effects, we expected higher levels of externalizing problem behavior in adolescents experiencing high peer rejection or low acceptance and in adolescents carrying the DAT1 10-repeat allele or the DRD4 long variant (i.e., 7-repeat or longer). With regard to interaction effects, we hypothesized that the effects of peers would be stronger for adolescents carrying the DAT1 10-repeat allele or the DRD4 long variant (i.e., 7-repeat or longer). In line with recent G × E studies (e.g., Bakermans-Kranenburg and van IJzendoorn 2011; Watts and McNulty 2015) and the concept of environmental sensitivity (Pluess 2015), we expected to find support for the *differential susceptibility hypothesis* (see Fig. 1c). In that case, adolescents carrying the DAT1 10R or the DRD4 long variant show more externalizing behavior than non-carriers when experiencing high peer rejection or low acceptance, but less externalizing behavior in case of low rejection and high acceptance. However, because few studies have examined different types of genetic moderation simultaneously, we also evaluated diathesis–stress and vantage sensitivity models using the criteria of Roisman et al. (2012). A *diathesis–stress hypothesis* (see Fig. 1a) is supported when carrying the DAT1 10R or the DRD4 long variant makes adolescents more vulnerable than non-carriers to developing externalizing problems when experiencing high levels of peer rejection or low levels of peer acceptance. In case of low rejection or high acceptance, we expected no difference. A *vantage sensitivity hypothesis* (see Fig. 1b) is supported when carriers are more sensitive than non-carriers to the benefits of low levels of peer rejection or high levels of peer acceptance, but not to the negative effects of adverse peer relationships (i.e., high rejection or low acceptance).

In the present study, we examined Achenbach's broadband Externalizing Problems Scale along with its two syndrome scales of aggressive and rule-breaking behavior (Achenbach 1991a, b) for two reasons. First, researchers have argued for disaggregating subtypes of externalizing behavior, given their unique underlying factors and developmental courses (Timmermans et al. 2009). Second, twin studies have found evidence for a latent externalizing

factor with a common genetic liability, but phenotypes within the spectrum were also determined by unique genetic and environmental influences (e.g., Dick et al. 2005; Krueger et al. 2002). Besides subtypes of externalizing problems, we also distinguished among informants of these behaviors. Adolescent and parent ratings of problem behavior typically show low to moderate correlations (Achenbach et al. 1987; Van Roy et al. 2010). Children and adolescents seem to report more symptoms than their parents, whereas parents seem to use a higher threshold for describing behavior as problematic and show more consistency in their evaluation (Sourander et al. 1999; Van Roy et al. 2010). Based on the assumption that both informants can provide valid judgments (De Los Reyes 2011), we explore whether associations between variables can be replicated across informants.

Methods

Participants and Procedure

Participants in the STRATEGIES project (i.e., Studying Transactions in Adolescence: Testing Genes in Interaction With Environments) were recruited from nine secondary schools in Flanders, the Dutch-speaking part of Belgium. The total sample consisted of 1116 adolescents from 121 classes in Grades 7–9. The present study used a subsample based on three inclusion criteria. First, we included only classes in which more than 60 % of classmates participated in the study ($N = 628$) taking into account the suggestion of Marks et al. (2013) for establishing reliable sociometric data. Second, from the 40 sibling pairs in our sample, we randomly selected one sibling to control for shared genetic background ($N = 588$). Third, only adolescents of European descent (i.e., all grandparents born in Europe) were included to account for population stratification ($N = 563$). In our final sample of 563 adolescents, the mean age was 13.81 years ($SD = .91$) and 52 % were girls. Mothers' highest level of education, as a measure of socio-economic status, was representative for the general Flemish population of females between 25 and 64 years old (Research Department of the Flemish Government 2011); with 32 % high school graduates, 47 % bachelor's degrees, 15 % master's degrees, and 6 % other levels. Adolescent data (i.e., questionnaires and saliva samples) were collected during school visits. To ensure that participants had spent enough time with their classmates to form peer relationships, assessment took place in the second semester (between February and June 2012). Within each school, all adolescents independently filled out the questionnaire at the same time. Unfinished questionnaires and parent questionnaires were completed at home. Parents and adolescents signed an active informed consent form and permission for the study was obtained from the Institutional Review Board of the Faculty of Medicine at the University of Leuven.

Measures

Peer Relationships

Peer acceptance and rejection were assessed by two items, "*Who do you most like to be with?*" (liking) and "*Who do you least like to be with?*" (disliking), using a within-classroom peer nomination procedure. Adolescents were given an alphabetical list of all classroom peers in which each peer had a unique number to facilitate nomination. Nominators were allowed to write down an unlimited number of peers for each of the two items. Self-nominations were not included. This method is a well-known and reliable procedure extensively used in previous research (Bukowski et al. 2000). The average number of nominations received for like was 2.36 ($SD = 1.85$; min: 0, max: 10) and for dislike 1.05 ($SD = 1.94$, min: 0, max: 17). Using SocStat software (Thissen-Pennings and Bendermacher 2002), nominations were summed for each individual adolescent and afterwards standardized (with $M = 0$ and $SD = 1$) within classrooms to control for variability in classroom size. A high peer acceptance score indicates that the adolescent is frequently nominated by classmates as liked most, whereas a high peer rejection score indicates frequent nominations as liked least.

Adolescent Problem Behavior

Adolescents and parents (i.e., mother and father were asked to fill out one CBCL questionnaire together) provided ratings on externalizing problem behavior by filling out the Youth Self Report (YSR; Achenbach 1991b) and the Child Behavior Checklist (CBCL; Achenbach 1991a), respectively. The broad-band Externalizing Problems scales (YSR: 31 items, $\alpha = .82$; CBCL: 35 items, $\alpha = .88$) each can be further divided into two narrow-band subscales: aggressive behavior (YSR: 17 items, $\alpha = .78$; CBCL: 18, $\alpha = .85$) and rule-breaking behavior (YSR: 14 items, $\alpha = .58$; CBCL: 17 items, $\alpha = .71$). An example item for aggressive behavior is: "I/my child get/gets in many fights", and for rule-breaking behavior is: "I/my child break/breaks rules at home, school or elsewhere". All items were answered on a 3-point rating scale from 0 = *not true*, 1 = *somewhat or sometimes true*, to 2 = *very true or often true*. Mean scale scores were computed, with higher scores indicating the presence of more externalizing problem behaviors.

Genotyping

Adolescents were genotyped using Oragene DNA kits for saliva sampling (DNA Genotek; Ontario, Canada). The present study examines the 40-bp VNTR in the DAT1 gene (forward primer: 5′-VIC-TGCGGTGTAGGGAACGGC CTGAG-3′; reverse primer: 5′-CTTCCTGGAGGTCACG GCTCAAGG-3′) and the 48-bp VNTR in the DRD4 gene (forward primer: 5′-NED-GCGACTACGTGGTCTACT CG-3′; reverse primer: 5′-AGGACCCTCATGGCCTT G-3′). These polymorphisms were genotyped by polymerase chain reaction (PCR) followed by a fragment analysis protocol. The PCR amplification mixture included 50 ng genomic DNA, 12.5 µl Master Mix (Promega), 0.5 µmol/l of each forward and reverse primer, 1 M Betaine solution (Sigma-Aldrich), and 1.5 µl water. The cycling conditions for the PCR started with 5 min at 95 °C, followed by 35 cycles of 30 s at 95 °C, 30 s at 60 °C, and 90 s at 72 °C, afterwards followed by 7 min at 72 °C. After finishing PCR, the DNA mixture was cooled down to 4 °C. Fragment analysis was performed with a mix contained 0.5 µl of the PCR product, 0.5 µl GeneScan 600 LIZ Size Standard V2.0 (Applied Biosystems) and 10 µl Hi-Di formamide. After a final denaturation step at 95 °C for 3 min, analysis followed on an ABI 3730xl Genetic Analyzer (Applied Biosystems). GeneMarker software Version 1.91 (SoftGenetics 2010) was used to print out the results.

Genetic information was available for 96 % of our sample (2 % absence during genotyping and 2 % technical failure). Genotypes were dummy coded based on existing neurobiological and behavioral genetic research. For the DAT1 gene, 10R carriers (i.e., at least one 10R, $n = 90$ %) were distinguished from non-10R carriers (i.e. both 9R, $n = 10$ %). Adolescents with alternative genotypes were coded as missing ($n = 14$). For the DRD4 gene, long carriers (i.e. at least one 7R or longer, $n = 34$ %) were distinguished from non-long carriers (i.e., both shorter than 7R, $n = 66$ %) in line with previous research (e.g., Dreber et al. 2009; Propper et al. 2007). Both VNTRs were in Hardy–Weinberg equilibrium ($p > .98$), indicating that allele and genotype frequencies of our sample were similar to what would be expected in the population. Other studies with comparable samples reported similar distributions (e.g., Guo et al. 2007; Mrug and Windle 2014).

Analytic Strategy

Hierarchical regression analyses were used to examine main and interaction effects of genes (i.e., DAT1 and DRD4) and peer relationships (i.e., acceptance and rejection) on adolescent problem behavior (i.e., externalizing problems, rule-breaking behavior, and aggressive behavior). Model 1 included control variables and main effects of gene and peer relationships, after which the interaction term was added in a second step (Model 2). Control variables included adolescent's sex and age, and mother's highest degree of education. Evidence suggests that externalizing problems are more prevalent in boys (e.g., Bongers et al. 2004) and low SES families (e.g., Martin et al. 2010), whereas different trajectories of externalizing problems may depend on developmental age (e.g., Bongers et al. 2004). We also controlled for gene-environment correlation to account for the possible direct association between dopaminergic genes and peer experiences. The advisability of multilevel analyses was examined by estimating the size of design effects (due to the nesting of students in classes and schools) for the outcomes of externalizing problems, aggressive behavior, and rule-breaking behavior. Design effects ranged from 1.00 to 1.88, which is less than the cut off of 2.00 suggested by Muthen and Satorra (1995). Therefore, multilevel analysis was not required. Regression analyses were performed in Mplus Version 6.1 (Muthén and Muthén 1998–2010). A combination of maximum likelihood (MLR) and full information maximum likelihood (FIML) estimation was used to account for, respectively, non-normality and missing data.

Significant interaction effects were further evaluated according to four critical tests suggested by Roisman et al. (2012). First, in order to make sure that a significant interaction effect was not an artifact of enforcing a linear model onto a non-linear phenomenon, an additional model was tested including the non-linear predictor terms E^2 and GE^2. Second, a conservative Bonferroni adjusted alpha value of .006 ($\alpha/n = .05/8$) was used to control for multiple testing of eight different models (i.e., two aspects of peer relationships, two genetic polymorphisms, and two informants for outcome behavior). Due to large correlations (i.e., r ranges from .65 to .96), subtypes of externalizing problems were not counted as independent models. Third, we calculated the Regions of Significance (RoS) to determine the range of the environmental variable (E) for which the association between the gene and the outcome variable was statistically significant. Fourth and finally, the crossover point (C) was calculated along with its reliability interval and two indexes: the proportion of the interaction (PoI; Proportion of Interest) and the proportion of cases on E (PA; Proportion of Affected) situated on the left versus the right side of C. The two final tests determined which of the three G × E frameworks is applicable, as explained in Fig. 1. RoS, PoI, and PA were calculated using the web application designed by Fraley (2012).

Results

Table 1 presents correlations among study variables. Adolescent and parent reports of problem behavior were only moderately correlated (r range from .39 to .42, $p < .001$), indicating different informant perspectives. Correlations between subtypes of problem behavior reported by the same informant were high (r range from .65 to .96, $p < .001$). More rejection was associated with more parent-reported problem behavior (r range from .13 to .16, $p < .01$), more adolescent-reported aggressive behavior ($r = .09$, $p < .05$), and lower peer acceptance ($r = -.47$, $p < .001$). DAT1 and DRD4 were uncorrelated with peer acceptance and rejection, indicating the absence of a direct link between genes and environment (*r*GE). Adolescent's sex and age, mother's highest degree of education, and gene–environment correlation were included as control variables in regression analyses. Boys reported more externalizing problems than girls (t (555) = 1.54, $p < .05$), especially rule-breaking behavior (t (555) = 4.50, $p < .001$). Parents also reported more externalizing problems in boys (t (445) = 3.51, $p < .001$), both aggressive (t (445) = 2.70, $p < .01$) and rule-breaking behavior (t (445) = 4.31, $p < .001$). Compared to girls, boys had more DAT1 10R alleles (χ^2 (1) = 5.61, $p < .05$) and DRD4 long alleles (χ^2 (1) = 4.51, $p < .05$). Older adolescents reported more externalizing problems ($r = .09$, $p < .05$), more specifically rule-breaking behavior ($r = .15$, $p < .001$). No significant differences were found for mothers' highest degree of education. Table 2 presents the standardized coefficient estimates of hierarchical regression analyses for main effects (M1) and interaction effects (M2).

Peer Acceptance

Main effects of peer acceptance did not reach statistical significance at .05 in any of the models. However, we did find marginally significant effects in the expected direction on parent-reported externalizing problems ($\beta = -.08$, $p < .10$), and more specifically on aggressive behavior ($\beta = -.08$, $p < .10$). Contrary to our expectations, adolescents carrying the long variant of the DRD4 showed less parent-reported externalizing problems ($\beta = -.10$, $p < .05$) and more specifically less aggressive behavior ($\beta = -.11$, $p < .05$). No interaction effects were found.

Peer Rejection

Peer rejection significantly and positively predicted parent-reported externalizing problems ($\beta = .16$, $p < .01$), both aggressive ($\beta = .16$, $p < .05$) and rule-breaking behavior ($\beta = .13$, $p < .01$). For adolescent ratings, only the positive effect on aggressive behavior reached statistical significance ($\beta = .10$, $p < .05$), whereas the effect on externalizing problems in general was marginally significant ($\beta = .08$, $p < .10$). Also in the peer rejection models, we found that long DRD4 carriers showed less parent-reported Externalizing ($\beta = -.11$, $p < .05$) and aggressive behavior ($\beta = -.12$, $p < .01$). When interaction terms were added, we found a significant interaction

Table 1 Correlations among study variables

Variables	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
(1) Externalizing-adolescent reported										
(2) Externalizing-parent reported	.43***									
(3) Aggressive-adolescent reported	.95***	.42***								
(4) Aggressive-parent reported	.38***	.96***	.39***							
(5) Rule-breaking-adolescent reported	.87***	.36***	.67***	.29***						
(6) Rule-breaking-parent reported	.43***	.83***	.38***	.65***	.42***					
(7) Peer acceptance	−.03	−.09†	−.04	−.09†	.00	−.08†				
(8) Peer rejection	.07†	.16***	.09*	.16***	.03	.13**	−.47***			
(9) DAT1	−.04	−.03	−.04	−.03	−.02	−.02	−.04	.04		
(10) DRD4	−.03	−.08	−.04	−.09†	−.02	−.04	−.05	.06	.00	
Mean	.26	.11	.29	.16	.22	.06	2.36	1.05	/	/
SD	.18	.14	.22	.20	.17	.10	1.85	1.94	/	/

The table presents correlations among study variables including problem behavior (i.e., externalizing, aggressive, and rule-breaking behavior, reported by adolescent or parent), peer relationships (i.e., peer acceptance and rejection), and dopaminergic VNTRs (dopamine transporter gene DAT1 and dopamine receptor D4 gene DRD4)

† $p < .10$; * $p < .05$; ** $p < .01$; *** $p < .001$

Table 2 Externalizing problem behavior regressed on DAT1 and DRD4 in interaction with peer acceptance and rejection ($N = 563$)

Outcome	Informant	Model	Acceptance							
			DAT1				DRD4			
			R^2	E	G	G × E	R^2	E	G	G × E
Externalizing problems	Adolescent	M1	.02†	−.02	−.05		.02†	−.02	−.05	
		M2	.03†	.15	−.04	−.18	.02†	.00	−.05	−.04
	Parents	M1	.04*	−.08†	−.05		.05**	−.08†	−.10*	
		M2	.04*	−.10	−.05	.02	.05**	−.04	−.10*	−.06
Aggressive behavior	Adolescent	M1	.01	−.04	−.05		.01	−.04	−.05	
		M2	.01	.18	−.04	−.23	.01	.00	−.05	−.07
	Parents	M1	.03†	−.08†	−.05		.04*	−.08†	−.11*	
		M2	.03†	−.11	−.05	.03	.04*	-.04	−.10*	−.06
Rule-breaking behavior	Adolescent	M1	.06**	.01	−.04		.06**	.01	−.04	
		M2	.06**	.08	−.03	−.07	.06**	.00	−.04	.03
	Parents	M1	.05**	−.06	−.04		.05**	−.06	−.06	
		M2	.05**	−.05	−.04	−.01	.05**	−.03	−.06	−.05
Outcome	Informant	Model	Rejection							
			DAT1				DRD4			
			R^2	E	G	G × E	R^2	E	G	G × E
Externalizing problems	Adolescent	M1	.03†	.08†	−.05		.03†	.08†	−.05	
		M2	.03†	.05	−.05	.03	.03*	.05	−.05	.05
	Parents	M1	.06*	.16**	−.05		.07*	.17**	−.11*	
		M2	.06*	.10	−.05	.07	.07*	.14	−.11*	.04
Aggressive behavior	Adolescent	M1	.01	.10*	−.05		.01	.10*	−.05	
		M2	.01	.06	−.05	.03	.02	.04	−.05	.09
	Parents	M1	.05†	.16*	−.05		.06*	.17**	−.12**	
		M2	.05†	.19	−.06	−.03	.06*	.15	−.12**	.02
Rule-breaking behavior	Adolescent	M1	.06**	.04	−.04		.06**	.04	−.04	
		M2	.06**	.02	−.04	.02	.06**	.05	−.04	−.02
	Parents	M1	.06**	.13**	−.05		.07**	.14**	−.07†	
		M2	.07**	−.11	−.03	.25**	.07**	.09	−.07	.06

Regression analyses examining the effects of peer relationships (acceptance and rejection) and dopaminergic genes (DAT1 and DRD4) on externalizing problems, subdivided in aggressive and rule-breaking behavior. Outcomes were reported upon by adolescents and parents. Model 1 (M1) included control variables (i.e., adolescent's sex and age, mother's highest degree of education, and gene–environment correlation) and main effects of the environment (E) and the gene (G). In Model 2 (M2), interaction effects are added (G × E). Values are standardized regression estimates

† $p < .10$; * $p < .05$; ** $p < .01$; *** $p < .001$

effect of the DAT1 gene and peer rejection on parent-reported rule-breaking behavior ($\beta = .25$, $p < .01$).

Further Study of the Significant Interaction Effect

One out of eight models tested for rule-breaking behavior showed a significant interaction effect, that is, between DAT1 and peer rejection on parent-reported rule-breaking behavior. Further analyses showed that this interaction was not the result of the curvilinear nature of the relation (i.e., the non-linear terms E^2 and GE^2 were non-significant). The p value of .008 of the interaction effect approximated closely the conservative Bonferroni adjusted alpha value of .006, therefore we considered further inspection of this effect valuable. When examining alternative G × E frameworks, we found support for differential susceptibility (presented in Fig. 2). The boundaries of both positive and negative Regions of Significance (RoS(+) = 1.77; RoS(−) = −.98) fell within the range of the environmental variable (E: min = −1.39; max = 3.47), showing that the interaction effect was significant at both ends of E. The crossover point (C = .32) fell within the range of E and the

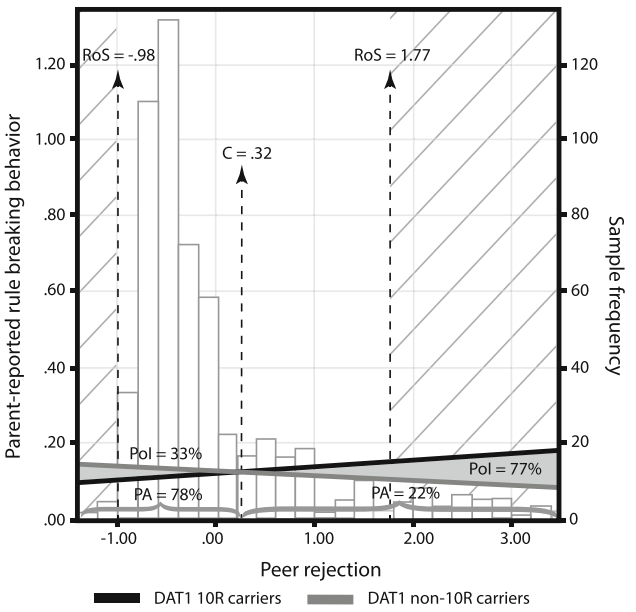

Fig. 2 Interaction between DAT1 and peer rejection associated with parent-reported Rule-Breaking Behavior. Adolescents with DAT1 10R allele (*black line*) showed less rule-breaking behavior according to parents when experiencing low peer rejection, but more rule-breaking behavior when experiencing high peer rejection, compared to non-carriers (*grey line*). The differential susceptibility hypothesis was supported by the critical tests of Roisman et al. (2012). The hatched Regions of Significance (RoS) show that the two regression lines significantly differ on both sides of the interaction. The cross-over point (C = .32) fell within the range observed for peer rejection (min = −1.39; max = 3.47). The Proportion of Interest (PoI), presented by the grey area between the regression lines, approximates 50 % on both sides of *C*. The Proportion of Affected (PA) is calculated on the sample frequency of peer rejection (right y-axis) and shows that 22 % of our sample experiences higher peer rejection than the cross-over value of .32

proportion of the interaction was close to 50 % on both the left (33 %) and the right side of C (77 %). When examining the raw data of our sample, we found that 22 % of the values of E fell above C (PA = left: 78 %; right: 22 %), which means that more adolescents were differentially affected by the interaction than the threshold of 16 % (Roisman et al. 2012). Compared to adolescents without the DAT1 10R allele, carriers showed more rule-breaking behavior according to parents when experiencing high peer rejection, but less rule-breaking behavior when experiencing low peer rejection.

Discussion

The current study examined the interplay between dopaminergic genes (i.e., DAT1 and DRD4) and peer relationships (i.e., rejection and acceptance) on externalizing problem behavior in adolescents. Despite the importance of peer experiences for adolescent development, research exploring gene-by-peer interaction is rare (Brendgen 2012). Based on previous research, we expected more externalizing problem behavior in adolescents experiencing peer rejection, and an association in the opposite direction with peer acceptance, but especially for those carrying the DAT1 10-repeat allele or the DRD4 long variant (i.e., 7R or longer). Our findings support the hypothesized link between problem behavior and peer rejection, but not the link between problem behavior and peer acceptance. We also found a main effect of the DRD4 gene suggesting more aggression in non-long carriers, which is not in line with previous research (e.g., Hohmann et al. 2009; Schmidt et al. 2002). In addition, there was one significant interaction effect indicating that the association between problem behavior and peer rejection was moderated by the DAT1 gene. Further evaluation using the Roisman criteria provided evidence for the differential susceptibility hypothesis. Adolescents carrying at least one 10R allele showed more rule-breaking behavior according to their parents, compared to non-carriers, when experiencing more rejection from their classmates. However, in the absence of rejection, parents of 10R carriers reported less rule-breaking behavior than parents of non-carriers. In other words, the DAT1 10R allele makes adolescents more susceptible to an adverse environment with high peer rejection, but also to a more favorable environment with low peer rejection.

Reward Sensitivity as an Underlying Mechanism

Little is known about the mechanisms underlying gene–environment interactions on externalizing problem behavior, but an interesting line of reasoning involves reward sensitivity. Evidence suggests that the general increase in risk-taking behavior in adolescents and their heightened susceptibility to peer influences is due to a neurological imbalance of two brain systems (Steinberg 2007). On the one hand, the cognitive control system is still developing during adolescence, resulting in a lack of self-control, whereas on the other hand, adolescents' reward system is oversensitive due to an increase in dopamine activity in this developmental period. Heightened dopamine activity has been associated with higher novelty seeking (Padmanabhan and Luna 2014). Adolescents carrying genetic variants associated with increased dopamine functionality might be even more sensitive to rewarding stimuli in the environment and, therefore, processes of social reinforcement and observational learning. Rejected young people have less access to well-adjusted peers, causing them to associate with and learn from other deviant and rejected peers (Light and Dishion 2007). Deviant friends respond positively to antisocial behavior, observe each other breaking the rules, and stimulate conversation about deviant activities. These

processes are referred to as deviancy training and make externalizing behavior interesting and rewarding, which reinforces its presence (Dishion et al. 1994). The rewarding effect of peer interactions might be especially relevant during adolescence, because young people spend increasing amounts of time with peers and value these interactions greatly. However, the opposite effect has also been suggested. Individuals with decreased dopaminergic activity have been found less reactive to simulation, which might lead them to seek out more extreme thrills and place them at high risk for externalizing problems (Matthys et al. 2013). It is therefore plausible that both increases and decreases from the optimal dopamine level lead to impaired functioning (Robbins and Arnsten 2009). What is considered as an optimal level depends upon the developmental stage and the specific situation. Similar to dopamine increases observed during stressful situations (Pani et al. 2000), the general increase in dopamine activity during adolescence may be adaptive to cope with important developmental tasks, such as identity formation and changes in social relationships (Negriff and Susman 2011).

The interaction between DAT1 and peer rejection, in the present study, might be interpreted in light of the reward sensitivity mechanism. Multiple in vitro and in vivo studies have suggested higher gene expression levels in DAT1 10R carriers, although opposite findings have also been reported (see Haddley et al. 2008, for a review). Higher levels of gene expression would promote more DAT protein production, which results in more effective reuptake of dopamine and eventually in reduced dopamine transmission in the brain (Dreher et al. 2009). If the 10R allele would be associated with higher gene expression and therefore reduced dopamine functionality, individuals carrying this variant might resolve insufficiently low dopamine levels by actively seeking thrills and risky situations. Due to the strong desire for stimulation and lack of self-regulation, these adolescents might be more sensitive to environmental influences, such as rejection by peers (Gardner et al. 2008; Goodnight et al. 2006). It is important to realize that hypothesizing underlying mechanisms remains rather ambiguous, because evidence on the functional effects of the DAT1 VNTR is inconclusive. Research incorporating neurobiological processes while examining G × E interactions provides an important challenge for future research (Caspi and Moffitt 2006).

Differential Effects of Peer Acceptance and Rejection

Similar to the findings of Kretschmer et al. (2014), we observed genetic moderation only for the effect of peer rejection and not peer acceptance. Because this result seems to suggest that genes influence problem behavior only in the presence of an adverse peer environment, Kretschmer and colleagues interpreted their findings as evidence for diathesis stress rather than differential susceptibility. However, the diathesis stress hypothesis indicates that in the absence of adversity there should be no differences in adjustment between vulnerable and resilient individuals (Pluess and Belsky 2013). In our study, the absence of peer rejection predicted less rule-breaking behavior in susceptible compared to non-susceptible adolescents. Although the absence of peer rejection is not the same as the presence of peer acceptance, it can been seen as a more positive environment and therefore function according to the differential susceptibility principle. A similar point can be made with regard to the outcome variable, because the absence of problem behavior is not the same as the presence of positive behavior. Failure to find an effect for peer acceptance, in the present study and the study by Kretschmer et al. (2014), might be due to the absence of a positive outcome variable, such as prosocial behavior or empathy.

Main Effect of DRD4: From Risk to Susceptibility

In contrast to the general consensus that the long DRD4 variant is associated with less adequate human behavior (Ebstein 2006), our results provide evidence for a positive effect: Adolescents with at least one long DRD4 allele showed fewer externalizing problems, more specifically aggression, according to their parents. Research examining desistance from delinquency has pointed to a similar result: Individuals with one or more DRD4 long variants were more likely to abstain from delinquency (Beaver et al. 2008). These findings may seem inconsistent because molecular genetic research strongly focusses on genetic risk. However, the results may rather indicate the susceptible character of this variant. Carrying a long DRD4 allele might not be universally favorable or adverse, as its effect depends upon the environment. In the present study, we were unable to identify a significant interaction between DRD4 and peer relationships, but the effect of DRD4 might depend on other environmental influences. For example, multiple studies examining parenting effects on externalizing problem behavior indicated differential susceptibility in individuals with the longer DRD4 variant. Carrying the 7R or longer DRD4 variant made children and adolescents more sensitive to the adverse effects of unsupportive parental environments (e.g., insecure attachment, low-quality parenting) and to the beneficial effects of a more supportive environment (e.g., secure attachment, parenting intervention) (e.g., Bakermans-Kranenburg and van IJzendoorn 2011; Beach et al. 2010).

Distinguishing Informants and Subtypes of Externalizing Problem Behavior

In order to grasp differential G-by-E interactions associated with externalizing behavior, we used two informants (i.e., adolescents versus parents) and explored two subtypes (i.e., aggressive and rule-breaking behavior). With regard to informants, all associations were present for parent ratings of problem behavior, but they were not replicated in adolescent ratings (except the association between peer rejection and aggression). Different perspectives among informants might provide an explanation. Research indicates that children and adolescents do not perceive all of these problems as problematic enough to impair their daily lives. Parents, on the other hand, seem to have a higher threshold for describing behavior as problematic and are more consistent in their evaluation (Van Roy et al. 2010). Therefore, parent ratings might better distinguish between minor problems and severe problematic situations. This might be especially true during adolescence, as parents are less aware of minor violations because young people are sometimes reticent to discuss problems with their parents and spend an increasing amount of time outside the home environment (Sourander et al. 1999).

With regard to subtypes of externalizing behavior, we observed a main effect of DRD4 specifically for aggressive behavior, whereas a significant interaction with DAT1 emerged specifically for rule-breaking behavior. These differential effects for externalizing subtypes are in line with earlier studies in behavioral genetics showing predominantly genetic influences on aggressive behavior, whereas non-aggressive rule-breaking behavior was influenced by both genetic and environmental aspects (e.g., Burt 2009; Eley et al. 2003). This pattern of findings would suggest direct genetic effects on aggressive behavior, whereas the etiology of rule-breaking behavior is predicted more strongly by the interplay between genes and environment. Earlier evidence also suggests that the genetic overlap between the two subtypes of externalizing behavior is only moderate, which could indicate that different genes contribute to each subtype (Wang et al. 2013). A molecular genetic study by Burt and Mikolajewski (2008) examined this hypothesis and found a direct effect of DAT1 specifically for rule-breaking behavior. Although no main effect of DAT1 was present in our study, this genetic variant did contribute specifically to rule-breaking behavior in interaction with peer rejection, whereas the effect of DRD4 proved to be unique for aggressive behavior. So, our results indicate, in line with earlier work, that it is important to distinguish between behavioral subtypes in the study of their genetic, environmental, or joint underpinnings.

Limitations and Directions for Future Research

Although the present study has important strengths, such as multi-informant data, differentiating subtypes of externalizing behavior, and a comprehensive G × E analysis, the results should be interpreted in light of some limitations. First, because our analyses were performed on cross-sectional and non-experimental data, causal inferences cannot be made. We analyzed the effect of peer rejection on externalizing problems in line with several studies showing such effects (e.g., Gorman et al. 2011; Laird et al.2001). There are other studies indicating a bidirectional relation (e.g., Gifford-Smith and Brownell 2003) or mediation through friendship selection and deviancy training (e.g., Light and Dishion 2007). In the present study, it was not our intention to disentangle these complex processes, rather to show the joint effects of genes and environment in a similar way as previous G × E studies on parenting behavior, a construct that is also bidirectional in nature. Longitudinal research might provide more insight in the underlying mechanisms.

Second, it is important to realize that the present study includes only a selection of many interesting variables. Future research should also incorporate other aspects of peer experiences, such as perceived popularity, friendships, peer networks, and victimization, which have been conceptualized as distinct but partially overlapping (Gifford-Smith and Brownell 2003). In addition, we should also be attentive to peer relationships in contexts other than the classroom, for example at school level, in the neighborhood, and during leisure activities. Further, including prosocial behavior, which is not the same as the absence of problem behavior, could yield more significant associations with the positive environmental factor of peer acceptance, thereby generating a more comprehensive approach for testing the differential susceptibility hypothesis. Finally, although we see a candidate gene study as a first valuable step towards understanding G × E effects, studying more complex networks of genes may be advantageous, for example by adopting a genetic pathway approach (Matthys et al. 2013; Steinberg 2007).

Third, power-analyses conducted with Quanto software (Gauderman and Morrison 2006) confirmed that our sample of 563 adolescents was sufficiently large to detect a small G × E effect (R^2 = .02 to .03) with 80 % power. Nevertheless, the significant interaction effect for rule-breaking behavior was only one out of eight models tested. We ruled out the possibility of a false positive result, because the p value of the interaction effect (p = .008) approximated closely the conservative Bonferroni adjusted alpha value of .006. Future studies with even larger samples and similar variables are needed to replicate our results.

Fourth, out of the total STRATEGIES sample of 1116 adolescents, a subsample of 563 was selected based on three inclusion criteria: a class participation rate of 60 % or higher, no siblings in the sample (in each sibling pair one member was randomly selected), and European descent. These criteria were necessary to generate more reliable peer nomination data, to control for shared genetic background among siblings, and to account for population stratification (i.e., differences in genetic make-up among ancestry groups). Unfortunately, applying these criteria resulted in a loss of data (i.e., excluded adolescents showed significantly more problem behavior and had lower educated mothers) and limited generalizability of our findings due to the lack of diversity (i.e., only European descent). Further, other unknown factors may have influenced the attrition between classes or the validity of our measures such as events that occurred prior to data collection (e.g., bullying seminar).

Conclusion

The present study was one of the first to investigate gene–environment interactions involving peer relationships on externalizing problem behavior in adolescence. We presented evidence for the notion that peer rejection is related to more externalizing behavior (i.e., a main effect of the peer environment), but we were unable to confirm our hypothesis for peer acceptance. Future research should include more positive outcome variables, such as empathy and prosocial behavior, which might be more related to positive peer experiences. A genetic main effect was found for DRD4, but not in the expected direction. Parents of adolescents without the long DRD4 variant reported more aggressive behavior than parents of long-carriers. This finding might indicate the susceptible character of the DRD4 polymorphism: carrying a long DRD4 allele might be favorable or adverse depending upon environmental influences. When we included G × E interaction in our models, we found one significant moderation effect for rule-breaking behavior out of eight models tested. Further evaluation by Roisman criteria supported evidence for differential susceptibility. When being rejected by classmates, adolescents carrying the DAT1 10R allele showed more rule-breaking behavior according to parents compared to non-carriers. However, in the absence of rejection, parents of 10R carriers reported less rule-breaking behavior. Finding an effect specifically for parent reported rule-breaking behavior emphasizes the importance of distinguishing between subtypes and informants of externalizing problems. We also explored the possible underlying mechanism of reward sensitivity. Through differences in dopamine functionality, which influences individuals' reward system and sensation seeking behavior, the DAT1 polymorphism might underlie differences in sensitivity to environmental clues. Longitudinal research using large samples is needed to replicate our findings and to further explore other aspects of peer experiences, positive outcome behaviors, and more complex gene networks.

Acknowledgments The authors received a Concerted Research Action Grant from the KU Leuven Research Fund (GOA/12/009). The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript. The authors did not have any competing interests that might be seen as influencing the research. Permission for the study was obtained from the Institutional Review Board of the Faculty of Medicine at the University of Leuven, Belgium. This manuscript is an original contribution which has not been previously published and is not being simultaneously submitted elsewhere. All authors are in agreement with the content of the manuscript and authorship in the indicated order.

Conflict of interest The authors report no conflict of interests.

Author contributions All authors conceived and designed the study. A.J. performed the data collection and statistical analysis and drafted the manuscript; W.V.D.N. coordinated the study, supported the data analysis, and helped to draft the manuscript; L.G., K.V., and H.C. coordinated the study and helped to draft the manuscript; S.D.L. performed the measurement and helped to draft the manuscript; S.C. coordinated the study; K.V.L. coordinated the study, supported the data analysis, and helped to draft the manuscript. All authors read and approved the final manuscript.

References

Achenbach, T. M. (1991a). *Manual for the child behavior checklist/4-18 and 1991 profile*. Burlington, VT: University of Vermont, Department of Psychiatry.

Achenbach, T. M. (1991b). *Manual for the youth self-report and 1991 profile*. Burlington, VT: University of Vermont, Department of Psychiatry.

Achenbach, T. M., McConaughy, S. H., & Howell, C. T. (1987). Child/adolescent behavioral and emotional problems: Implications of cross-informant correlations for situational specificity. *Psychological Bulletin, 101*, 213–232. doi:10.1037/0033-2909.101.2.213.

Allen, J. P., Porter, M. R., McFarland, F. C., Marsh, P., & McElhaney, K. B. (2005). The two faces of adolescents' success with peers: adolescent popularity, social adaptation, and deviant behavior. *Child Development, 76*, 747–760. doi:10.1111/j.1467-8624.2005.00875.x.

Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2011). Differential susceptibility to rearing environment depending on dopamine-related genes: New evidence and a meta-analysis. *Development and Psychopathology, 23*, 39–52. doi:10.1017/S0954579410000635.

Baumeister, R. F., & Leary, M. R. (1995). The need to belong: Desire for interpersonal attachments as a fundamental human-motivation. *Psychological Bulletin, 117*, 497–529. doi:10.1037/0033-2909.117.3.497.

Beach, S. R., Brody, G. H., Lei, M. K., & Philibert, R. A. (2010). Differential susceptibility to parenting among African American youths: Testing the DRD4 hypothesis. *Journal of Family Psychology, 24*, 513–521. doi:10.1037/a0020835.

Beaver, K. M., Wright, J. P., DeLisi, M., & Vaughn, M. G. (2008). Desistance from delinquency: The marriage effect revisited and extended. *Social Science Research, 37*, 736–752. doi:10.1016/j.ssresearch.2007.11.003.

Belsky, J. (2005). Differential susceptibility to rearing influence: An evolutionary hypothesis and some evidence. In B. Ellis & D. Bjorklund (Eds.), *Origins of the social mind: Evolutionary psychology and child development* (pp. 139–163). New York, NY: Guilford.

Blum, K., Chen, A. L. C., Oscar-Berman, M., Chen, T. J. H., Lubar, J., White, N., & Bailey, J. A. (2011). Generational association studies of dopaminergic genes in reward deficiency syndrome (RDS) subjects: Selecting appropriate phenotypes for reward dependence behaviors. *International Journal of Environmental Research and Public Health, 8*, 4425–4459. doi:10.3390/ijerph8124425.

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

Boutwell, B. B., & Beaver, K. M. (2008). A biosocial explanation of delinquency abstention. *Criminal Behaviour and Mental Health, 18*, 59–74. doi:10.1002/cbm.678.

Brendgen, M. (2012). Genetics and peer relations: A review. *Journal of Research on Adolescence, 22*, 419–437. doi:10.1111/j.1532-7795.2012.00798.x.

Brendgen, M., & Vitaro, F. (2008). Peer rejection and physical health problems in early adolescence. *Journal of Developmental and Behavioral Pediatrics, 29*, 183–190. doi:10.1097/DBP.0b013e318168be15.

Bronfenbrenner, U., & Morris, P. A. (2006). The bioecological model of human development. In R. M. Lerner & W. Damon (Eds.), *Handbook of child psychology* (6th ed., Vol. 1, pp. 793–828). Hoboken, NY: Wiley.

Bukowski, W. M., Sippola, L., Hoza, B., & Newcomb, A. F. (2000). Pages from a sociometric notebook: An analysis of nomination and rating scale measures of acceptance, rejection, and social preference. *New Directions for Child and Adolescent Development, 2000*, 11–26. doi:10.1002/cd.23220008804.

Burt, S. A. (2009). Are there meaningful etiological differences within antisocial behavior? Results of a meta-analysis. *Clinical Psychology Review, 29*, 163–178. doi:10.1016/j.cpr.2008.12.004.

Burt, S. A., & Mikolajewski, A. J. (2008). Preliminary evidence that specific candidate genes are associated with adolescent-onset antisocial behavior. *Aggressive Behavior, 34*, 437–445. doi:10.1002/Ab.20251.

Caspi, A., & Moffitt, T. E. (2006). Gene–environment interactions in psychiatry: Joining forces with neuroscience. *Nature Reviews Neuroscience, 7*, 583–590. doi:10.1038/nrn1925.

Chang, F. M., Kidd, J. R., Livak, K. J., Pakstis, A. J., & Kidd, K. K. (1996). The world-wide distribution of allele frequencies at the human dopamine D4 receptor locus. *Human Genetics, 98*, 91–101. doi:10.1007/s004390050166.

De Los Reyes, A. (2011). Introduction to the special section: More than measurement error: Discovering meaning behind informant discrepancies in clinical assessments of children and adolescents. *Journal of Clinical Child and Adolescent Psychology, 40*, 1–9. doi:10.1080/15374416.2011.533405.

Dick, D. M., Viken, R. J., Kaprio, J., Pulkkinen, L., & Rose, R. J. (2005). Understanding the covariation among childhood externalizing symptoms: Genetic and environmental influences on conduct disorder, attention deficit hyperactivity disorder, and oppositional defiant disorder symptoms. *Journal of Abnormal Child Psychology, 33*, 219–229. doi:10.1007/s10802-005-1829-8.

DiLalla, L. F., Elam, K. K., & Smolen, A. (2009). Genetic and gene–environment interaction effects on preschoolers' social behaviors. *Developmental Psychobiology, 51*, 451–464. doi:10.1002/dev.20384.

Dishion, T. J., Patterson, G. R., & Griesler, P. C. (1994). Peer adaptations in the development of antisocial behavior: A confluence model. In R. L. Huesmann (Ed.), *Aggressive behavior: Current perspectives* (pp. 61–95). New York, NY: Plenum Press.

Dishion, T. J., Nelson, S. E., & Yasui, M. (2005). Predicting early adolescent gang involvement from middle school adaptation. *Journal of Clinical Child and Adolescent Psychology, 34*, 62–73. doi:10.1207/s15374424jccp3401_6.

Dmitrieva, J., Chen, C., Greenberger, E., Ogunseitan, O., & Ding, Y. C. (2011). Gender-specific expression of the DRD4 gene on adolescent delinquency, anger and thrill seeking. *Social Cognitive and Affective Neuroscience, 6*, 82–89. doi:10.1093/scan/nsq020.

Dreber, A., Apicella, C. L., Eisenberg, D. T. A., Garcia, J. R., Zamore, R. S., Lum, J. K., & Campbell, B. (2009). The 7R polymorphism in the dopamine receptor D4 gene (DRD4) is associated with financial risk taking in men. *Evolution and Human Behavior, 30*, 85–92. doi:10.1016/j.evolhumbehav.2008.11.001.

Dreher, J. C., Kohn, P., Kolachana, B., Weinberger, D. R., & Berman, K. F. (2009). Variation in dopamine genes influences responsivity of the human reward system. *Proceedings of the National Academy of Sciences of the United States of America, 106*, 617–622. doi:10.1073/pnas.0805517106.

Ebstein, R. P. (2006). The molecular genetic architecture of human personality: Beyond self-report questionnaires. *Molecular Psychiatry, 11*, 427–445. doi:10.1038/sj.mp.4001814.

Eley, T. C., Lichtenstein, P., & Moffitt, T. E. (2003). A longitudinal behavioral genetic analysis of the etiology of aggressive and nonaggressive antisocial behavior. *Development and Psychopathology, 15*, 383–402. doi:10.1017/S095457940300021X.

Farbiash, T., Berger, A., Atzaba-Poria, N., & Auerbach, J. G. (2014). Prediction of preschool aggression from DRD4 risk, parental ADHD symptoms, and home chaos. *Journal of Abnormal Child Psychology, 42*, 489–499. doi:10.1007/s10802-013-9791-3.

Fraley, R. C. (2012). Probing interactions in differential susceptibility research. University of Illinois. Retrieved from http://www.yourpersonality.net/interaction/ros2.pl.

Gardner, T. W., Dishion, T. J., & Connell, A. M. (2008). Adolescent self-regulation as resilience: Resistance to antisocial behavior within the deviant peer context. *Journal of Abnormal Child Psychology, 36*, 273–284. doi:10.1007/s10802-007-9176-6.

Gauderman, W. J., & Morrison, J. M. (2006). QUANTO 1.1: A computer program for power and sample size calculations for genetic-epidemiology studies. Retrieved from http://biostats.usc.edu/Quanto.html.

Gifford-Smith, M. E., & Brownell, C. A. (2003). Childhood peer relationships: Social acceptance, friendships, and peer networks. *Journal of School Psychology, 41*, 235–284. doi:10.1016/S0022-4405(03)00048-7.

Goodnight, J. A., Bates, J. E., Newman, J. P., Dodge, K. A., & Pettit, G. S. (2006). The interactive influences of friend deviance and reward dominance on the development of externalizing behavior during middle adolescence. *Journal of Abnormal Child Psychology, 34*, 573–583. doi:10.1007/s10802-006-9036-9.

Gorman, A. H., Schwartz, D., Nakamoto, J., & Mayeux, L. (2011). Unpopularity and disliking among peers: Partially distinct dimensions of adolescents' social experiences. *Journal of Applied Developmental Psychology, 32*, 208–217. doi:10.1016/j.appdev.2011.05.001.

Grills-Taquechel, A. E., Norton, P., & Ollendick, T. H. (2010). A longitudinal examination of factors predicting anxiety during the transition to middle school. *Anxiety Stress and Coping, 23*, 493–513. doi:10.1080/10615800903494127.

Guo, G., Roettger, M. E., & Shih, J. C. (2007). Contributions of the DAT1 and DRD2 genes to serious and violent delinquency among adolescents and young adults. *Human Genetics, 121*, 125–136. doi:10.1007/s00439-006-0244-8.

Haddley, K., Vasiliou, A. S., Ali, F. R., Paredes, U. M., Bubb, V. J., & Quinn, J. P. (2008). Molecular genetics of monoamine transporters: Relevance to brain disorders. *Neurochemical Research, 33*, 652–667. doi:10.1007/s11064-007-9521-8.

Hohmann, S., Becker, K., Fellinger, J., Banaschewski, T., Schmidt, M. H., Esser, G., & Laucht, M. (2009). Evidence for epistasis between the 5-HTTLPR and the dopamine D4 receptor polymorphisms in externalizing behavior among 15-year-olds. *Journal of Neural Transmission, 116*, 1621–1629. doi:10.1007/s00702-009-0290-1.

Huesmann, L. R., Dubow, E. F., & Boxer, P. (2009). Continuity of aggression from childhood to early adulthood as a predictor of life outcomes: Implications for the adolescent-limited and life-course-persistent models. *Aggressive Behavior, 35*, 136–149. doi:10.1002/ab.20300.

Kiesner, J. (2002). Depressive symptoms in early adolescence: Their relations with classroom problem behavior and peer status. *Journal of Research on Adolescence, 12*, 463–478. doi:10.1111/1532-7795.00042.

Kretschmer, T., Dijkstra, J. K., Ormel, J., Verhulst, F. C., & Veenstra, R. (2013). Dopamine receptor D4 gene moderates the effect of positive and negative peer experiences on later delinquency: The tracking adolescents' individual lives survey study. *Development and Psychopathology, 25*, 1107–1117. doi:10.1017/S0954579413000400.

Kretschmer, T., Sentse, M., Dijkstra, J. K., & Veenstra, R. (2014). The interplay between peer rejection and acceptance in preadolescence and early adolescence, serotonin transporter gene, and antisocial behavior in late adolescence: The TRAILS study. *Merrill-Palmer Quarterly, 60*, 193–216. doi:10.1353/mpq.2014.0008.

Krueger, R. F., Hicks, B. M., Patrick, C. J., Carlson, S. R., Iacono, W. G., & McGue, M. (2002). Etiologic connections among substance dependence, antisocial behavior and personality: Modeling the externalizing spectrum. *Journal of Abnormal Psychology, 111*, 411–424. doi:10.1037//0021-843x.111.3.411.

Lahey, B. B., Rathouz, P. J., Lee, S. S., Chronis-Tuscano, A., Pelham, W. E., Waldman, I. D., & Cook, E. H. (2011). Interactions between early parenting and a polymorphism of the child's dopamine transporter gene in predicting future child conduct disorder symptoms. *Journal of Abnormal Psychology, 120*, 33–45. doi:10.1037/a0021133.

Laird, R. D., Jordan, K. Y., Dodge, K. A., Pettit, G. S., & Bates, J. E. (2001). Peer rejection in childhood, involvement with antisocial peers in early adolescence, and the development of externalizing behavior problems. *Development and Psychopathology, 13*, 337–354. doi:10.1017/S0954579401002085.

Larsen, H., van der Zwaluw, C. S., Overbeek, G., Granic, I., Franke, B., & Engels, R. C. M. E. (2010). A variable-number-of-tandem-repeats polymorphism in the dopamine D4 receptor gene affects social adaptation of alcohol use: Investigation of a gene–environment interaction. *Psychological Science, 21*, 1064–1068. doi:10.1177/0956797610376654.

Lee, S. S., Lahey, B. B., Waldman, I., Van Hulle, C. A., Rathouz, P., Pelham, W. E., & Cook, E. H. (2007). Association of dopamine transporter genotype with disruptive behavior disorders in an eight-year longitudinal study of children and adolescents. *American Journal of Medical Genetics Part B, 144*, 310–317. doi:10.1002/Ajmg.B.30447.

Light, J. M., & Dishion, T. J. (2007). Early adolescent antisocial behavior and peer rejection: A dynamic test of a developmental process. *New Directions for Child and Adolescent Development, 118*, 77–89. doi:10.1002/cd.202.

Marks, P. E. L., Babcock, B., Cillessen, A. H. N., & Crick, N. R. (2013). The effects of participation rate on the internal reliability of peer nomination measures. *Social Development, 22*, 609–622. doi:10.1111/j.1467-9507.2012.00661.x.

Martin, M. J., Conger, R. D., Schofield, T. J., Dogan, S. J., Widaman, K. F., Donnellan, M. B., & Neppl, T. K. (2010). Evaluation of the interactionist model of socioeconomic status and problem behavior: A developmental cascade across generations. *Development and Psychopathology, 22*, 695–713. doi:10.1017/S0954579410000374.

Masten, C. L., Eisenberger, N. I., Borofsky, L. A., Pfeifer, J. H., McNealy, K., Mazziotta, J. C., & Dapretto, M. (2009). Neural correlates of social exclusion during adolescence: Understanding the distress of peer rejection. *Social Cognitive and Affective Neuroscience, 4*, 143–157. doi:10.1093/scan/nsp007.

Matthys, W., Vanderschuren, L. J. M. J., & Schutter, D. J. L. G. (2013). The neurobiology of oppositional defiant disorder and conduct disorder: Altered functioning in three mental domains. *Development and Psychopathology, 25*, 193–207. doi:10.1017/S0954579412000272.

Mitchell, R. J., Howlett, S., Earl, L., White, N. G., McComb, J., Schanfield, M. S., & Crawford, M. H. (2000). Distribution of the 3'VNTR polymorphism in the human dopamine transporter gene in world populations. *Human Biology, 72*, 295–304.

Mrug, S., & Windle, M. (2014). DRD4 and susceptibility to peer influence on alcohol use from adolescence to adulthood. *Drug and Alcohol Dependence, 145*, 168–173. doi:10.1016/j.drugalcdep.2014.10.009.

Muthén, L. K., Muthén, B. O. (1998–2010). *Mplus user's guide* (6th ed.). Los Angeles, CA: Muthén & Muthén.

Muthen, B. O., & Satorra, A. (1995). Complex sample data in structural equation modeling. *Sociological Methodology, 25*, 267–316. doi:10.2307/271070.

Negriff, S., & Susman, E. J. (2011). Pubertal timing, depression, and externalizing problems: A framework, review, and examination of gender differences. *Journal of Research on Adolescence, 21*, 717–746. doi:10.1111/j.1532-7795.2010.00708.x.

Padmanabhan, A., & Luna, B. (2014). Developmental imaging genetics: Linking dopamine function to adolescent behavior. *Brain and Cognition, 89*, 27–38. doi:10.1016/j.bandc.2013.09.011.

Pani, L., Porcella, A., & Gessa, G. L. (2000). The role of stress in the pathophysiology of the dopaminergic system. *Molecular Psychiatry, 5*, 14–21. doi:10.1038/sj.mp.4000589.

Parker, J. G., Rubin, K. H., Erath, S. A., Wojslawowicz, J. C., & Buskirk, A. A. (2006). Peer relationships, child development, and adjustment: A developmental psychopathology perspective. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology, Vol 1: Theory and method* (2nd ed., pp. 419–493). Hoboken, NJ: Wiley.

Pluess, M. (2015). Individual differences in environmental sensitivity. *Child Development Perspectives,*. doi:10.1111/cdep.12120.

Pluess, M., & Belsky, J. (2013). Vantage sensitivity: Individual differences in response to positive experiences. *Psychological Bulletin, 139*, 901–916. doi:10.1037/a0030196.

Propper, C., Willoughby, M., Halpern, C. T., Carbone, M. A., & Cox, M. (2007). Parenting quality, DRD4, and the prediction of externalizing and internalizing behaviors in early childhood. *Developmental Psychobiology, 49*, 619–632. doi:10.1002/dev.20249.

Reef, J., Diamantopoulou, S., van Meurs, I., Verhulst, F. C., & van der Ende, J. (2011). Developmental trajectories of child to adolescent externalizing behavior and adult DSM-IV disorder: Results of a 24-year longitudinal study. *Social Psychiatry and Psychiatric Epidemiology, 46*, 1233–1241. doi:10.1007/s00127-010-0297-9.

Reitz, E., Deković, M., & Meijer, A. M. (2005). The structure and stability of externalizing and internalizing problem behavior during early adolescence. *Journal of Youth and Adolescence, 34*, 577–588. doi:10.1007/s10964-005-8947-z.

Research Department of the Flemish Government. (2011). *Scholingsgraad van de bevolking (25-64 jaar), in %*. [Level of education of the population (25–64 years old), in %]. Retrieved from http://www4dar.vlaanderen.be/sites/svr/Cijfers/Exceltabellen/onderwijs/kwaliteitszorg/ONDEKWAL012.xls.

Robbins, T. W., & Arnsten, A. F. T. (2009). The neuropsychopharmacology of fronto-executive function: Monoaminergic modulation. *Annual Review of Neuroscience, 32*, 267–287. doi:10.1146/annurev.neuro.051508.135535.

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

Rubin, K. H., Bukowski, W. M., & Parker, J. G. (2006). Peer interactions, relationships, and groups. In N. Eisenberg (Ed.), *Handbook of child psychology* (6 ed., Vol. Social, emotional, and personality development, pp. 571–645). Hoboken, NJ: Wiley.

Rutter, M. (2006). *Genes and behavior: Nature–nurture interplay explained*. Oxford, UK: Blackwell Publishing.

Schmidt, L. A., Fox, N. A., Rubin, K. H., Hu, S., & Hamer, D. H. (2002). Molecular genetics of shyness and aggression in preschoolers. *Personality and Individual Differences, 33*, 227–238. doi:10.1016/S0191-8869(01)00147-7.

Sentse, M., Lindenberg, S., Omvlee, A., Ormel, J., & Veenstra, R. (2010). Rejection and acceptance across contexts: Parents and peers as risks and buffers for early adolescent psychopathology. The TRAILS study. *Journal of Abnormal Child Psychology, 38*, 119–130. doi:10.1007/s10802-009-9351-z.

SoftGenetics. (2010). GeneMarker (Version 1.91) [Computer software]. State College, PA, USA SoftGenetics.

Sonuga-Barke, E. J., Oades, R. D., Psychogiou, L., Chen, W., Franke, B., Buitelaar, J., & Faraone, S. V. (2009). Dopamine and serotonin transporter genotypes moderate sensitivity to maternal expressed emotion: The case of conduct and emotional problems in attention deficit/hyperactivity disorder. *Journal of Child Psychology and Psychiatry, 50*, 1052–1063. doi:10.1111/j.1469-7610.2009.02095.x.

Sourander, A., Helstela, L., & Helenius, H. (1999). Parent–adolescent agreement on emotional and behavioral problems. *Social Psychiatry and Psychiatric Epidemiology, 34*, 657–663. doi:10.1007/s001270050189.

Steinberg, L. (2007). Risk taking in adolescence: New perspectives from brain and behavioral science. *Current Directions in Psychological Science, 16*, 55–59. doi:10.1111/j.1467-8721.2007.00475.x.

Thissen-Pennings, M. C. E., & Bendermacher, A. N. H. (2002). *SOCSTAT: A program for sociometric status*. Nijmegen, NL: Research Technische Ondersteuningsgroep, Radboud University.

Timmermans, M., van Lier, P. A. C., & Koot, H. M. (2009). Pathways of behavior problems from childhood to late adolescence leading to delinquency and academic underachievement. *Journal of Clinical Child and Adolescent Psychology, 38*, 630–638. doi:10.1080/15374410903103502.

Van Roy, B., Groholt, B., Heyerdahl, S., & Clench-Aas, J. (2010). Understanding discrepancies in parent–child reporting of emotional and behavioural problems: Effects of relational and socio-demographic factors. *BMC Psychiatry,*. doi:10.1186/1471-244x-10-56.

Vanhalst, J., Luyckx, K., & Goossens, L. (2014). Experiencing loneliness in adolescence: A matter of individual characteristics, negative peer experiences, or both? *Social Development, 23*, 100–118. doi:10.1111/Sode.12019.

Vassos, E., Collier, D. A., & Fazel, S. (2014). Systematic meta-analyses and field synopsis of genetic association studies of violence and aggression. *Molecular Psychiatry, 19*, 471–477. doi:10.1038/Mp.2013.31.

Vaughn, M. G., Delisi, M., Beaver, K. M., & Wright, J. P. (2009). DAT1 and 5HTT are associated with pathological criminal behavior in a nationally representative sample of youth. *Criminal Justice and Behavior, 36*, 1113–1124. doi:10.1177/0093854809342839.

Veronneau, M. H., & Dishion, T. J. (2010). Predicting change in early adolescent problem behavior in the middle school years: A mesosystemic perspective on parenting and peer experiences. *Journal of Abnormal Child Psychology, 38*, 1125–1137. doi:10.1007/s10802-010-9431-0.

Waas, G. A. (2006). Peer relationships. In G. G. Bear & K. M. Minke (Eds.), *Children's needs III: Development, prevention, and intervention* (pp. 325–340). Bethesda, MD: National Association of School Psychologists.

Wang, P., Niv, S., Tuvblad, C., Raine, A., & Baker, L. A. (2013). The genetic and environmental overlap between aggressive and non-aggressive antisocial behavior in children and adolescents using the self-report delinquency interview (SR-DI). *Journal of Criminal Justice, 41*, 277–284. doi:10.1016/j.jcrimjus.2013.06.011.

Watts, S. J., & McNulty, T. L. (2015). Delinquent peers and offending: Integrating social learning and biosocial theory. *Youth Violence and Juvenile Justice, 13*, 190–206. doi:10.1177/1541204014523797.

Young, S. E., Smolen, A., Corley, R. P., Krauter, K. S., DeFries, J. C., Crowley, T. J., & Hewitt, J. K. (2002). Dopamine transporter polymorphism associated with externalizing behavior problems in children. *American Journal of Medical Genetics Part A, 114*, 144–149. doi:10.1002/Ajmg.10155.

Annelies Janssens is a doctoral candidate in the faculty of Psychology and Educational Sciences at the KU Leuven in Belgium. Her doctoral dissertation research addresses gene-by-environment interaction in externalizing problem behaviors in adolescence. Her research interests are adolescent behavior, parent–adolescent relationships, peer relationships, aggression, and rule breaking behavior.

Wim Van den Noortgate is Professor of statistics and methodology at the Faculty of Psychology and Educational Sciences at the KU Leuven. He obtained his PhD in Educational Sciences at the same university. His research interests include multilevel analysis, meta-analysis and psychometrics.

Luc Goossens is a Full Professor in Developmental Psychology at the KU Leuven in Belgium. He obtained his PhD in Psychology at the same university. His major research interests include adolescent identity, loneliness, and gene–environment interactions.

Karine Verschueren is a Full Professor in School Psychology at the KU Leuven in Belgium. She received her Ph.D. in Developmental Psychology from the KU Leuven. Her major research interests include teacher–child and peer relationships, self-concept, and academic engagement.

Hilde Colpin is a Professor of School Psychology at KU Leuven in Belgium. She received her PhD in Parenting Studies from the KU Leuven. Her research mainly focuses on (interventions targeted at) proximal interactions affecting children's behavioral development in school settings.

Steven De Laet is a doctoral student at the Research Unit School Psychology and Developmental Psychology of Children and Adolescents at KU Leuven in Belgium. His research mainly focuses on teacher–child and peer relationships, school engagement, and externalizing behavior.

Stephan Claes is Professor of Psychiatry at the KU Leuven in Belgium and Senior Clinical Researcher of the Fund for Scientific Research (FWO) in Flanders. He obtained his Ph.D. in Human Genetics at the KU Leuven. His main research interest is the genetic and epigenetic background of stress sensitivity and mood disorders.

Karla Van Leeuwen is an Associate Professor at the Parenting and Special Education Research Unit, Faculty of Psychology and Educational Sciences (KU Leuven). She received her doctorate in Psychology from Ghent University. Her research interests include child/adolescent characteristics (temperament, personality, genes) by environment interactions in the prediction of child/adolescent outcome, and assessment of parenting.