The Interplay Between Peer Rejection and Acceptance in Preadolescence and Early Adolescence, Serotonin Transporter Gene, and Antisocial Behavior in Late Adolescence: The TRAILS Study

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Gene–environment studies on adolescents' peer contexts are important for understanding the interplay between biological and social antecedents of adolescent psychopathology. To this end, this study examined the roles of serotonin transporter (*5-HTTLPR*) and preadolescent and early adolescent peer rejection and acceptance, as well as the interaction between genotype and environment as predictors of antisocial behavior. Longitudinal data from TRAILS (TRacking Adolescents' Individual Lives Survey), a Dutch cohort study into adolescent development that includes peer reports of rejection and acceptance assessed at 11.1 and 13.6 years and self-reported antisocial behavior at 19.1 years was used. The interaction between *5-HTTLPR* and preadolescent peer rejection predicted antisocial behavior with carriers of the *5-HTTLPR* short–short variant most strongly affected. No main or interaction effects were found for early adolescent rejection or interactions involving peer acceptance. Our results extend the gene–environment interaction literature by focusing on peer relationship experiences.

Antisocial behavior has been a focus of research in several disciplines for at least a century. Different theories regarding its antecedents have emerged—some of which focused on social and others on biological roots

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Merrill-Palmer Quarterly, April 2014, Vol. 60, No. 2, pp. 193–216. Copyright © 2014 by Wayne State University Press, Detroit, MI 48201.

of antisocial behavior, but the past few decades have seen an increase in studies that acknowledge the interplay of environmental and biological factors (Dodge & Pettit, 2003; for an overview of studies, see Raine, 2002, and Rutter, 2006). Spurred by quantitative genetic findings that antisocial behavior is substantially heritable (Mason & Frick, 1994; Rhee & Waldman, 2002), accounts have focused on tracing specific candidate genes that may have a direct effect on antisocial behavior (e.g., Burt & Mikolajewski, 2008) or interact with environmental risks in their prediction of antisocial behavior (Caspi et al., 2002). A popular candidate gene for both mechanisms is the serotonin transporter gene, likely because of its association with antisocial behavior and related types of externalizing behaviors (e.g., Feinn, Nellissery, & Kranzler, 2005; Vaughn, DeLisi, Beaver, & Wright, 2009) and implication in emotional stimuli processing.

The majority of gene–environment interaction (G $\times$ E) studies has been conducted on children's family environment. However, especially as young people transition from childhood into adolescence, peers gain crucial importance. Establishing friendships and being liked by peers are important developmental tasks in early adolescence and associated with later adjustment (e.g., Berndt, 1982; Rose-Krasnor, 1997). Given the problems linked to peer rejection in preadolescence and early adolescence (Kupersmidt & Coie, 1990; Parker & Asher, 1987), searching for factors that buffer or elevate this risk is vital. Aiming to do so and informed by

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This research is part of the TRacking Adolescents' Individual Lives Survey (TRAILS). TRAILS participating centers include various departments of the University of Groningen and University Medical Center Groningen, Erasmus University Medical Center Rotterdam, University of Utrecht, Radboud Medical Center Nijmegen, and the Parnassia Bavo group, all in the Netherlands. TRAILS has been financially supported by various grants from the Netherlands Organization for Scientific Research NWO (Medical Research Council program grant GB-MW 940-38-011; ZonMW Brainpower grant 100-001-004; ZonMw Risk Behavior and Dependence grants 60-60600-97-118; ZonMw Culture and Health grant 261-98-710; Social Sciences Council medium-sized investment grants GB-MaGW 480-01-006 and GB-MaGW 480-07-001; Social Sciences Council project grants GB-MaGW 452-04-314 and GB-MaGW 452-06-004; NWO large investment grant 175.010.2003.005; NWO Longitudinal Survey and Panel Funding 481-08-013), the Dutch Ministry of Justice (WODC), the European Science Foundation (EuroSTRESS project FP-006), Biobanking and Biomolecular Resources Research Infrastructure BBMRI-NL (CP 32), and the participating universities. Part of this research was funded by the Netherlands Organisation for Scientific Research NOW Vernieuwingsimpuls (VENI) project 451-10-012 awarded to Jan Kornelis Dijkstra (2010). We are grateful to all adolescents, their parents, and their teachers who participated in this research and to everyone who worked on this project and made it possible.

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previous G × E studies, we focused specifically on *5-HTTLPR,* a common polymorphism in the serotonin transporter gene. Serotonin levels in the amygdala—a brain region responsible for processing emotional stimuli such as social rejection—are partly regulated by *5-HTTLPR,* which makes this gene a prime candidate to study as a moderating factor of the association between peer rejection/acceptance and maladjustment.

Benefiting from peer and self-reports from adolescents acquired over several time points, the current study examined direct effects of peer rejection and acceptance assessed in preadolescence and early adolescence and *5-HTTLPR* on late adolescent antisocial behavior. Moreover, it was tested whether *5-HTTLPR* moderated the hypothesized risk of peer rejection and the potentially protective effect of peer acceptance on late adolescent antisocial behavior. This design responds to recent calls to pay attention to potential developmental variability in (genetic) vulnerability or susceptibility to environmental effects (Ellis, Boyce, Belsky, Bakermans-Kranenburg, & Van IJzendoorn, 2011). Moreover, by including negative as well as positive peer relationship indicators, we were able to test more stringently whether *5-HTTLPR* contributes to some individuals' greater vulnerability or susceptibility to the environment. That is, a differential vulnerability model is supported if carriers of specific *5-HTTLPR* variants differ in how much they are affected by peer rejection but not acceptance. In contrast, a pattern where carriers of different variants of the serotonin transporter polymorphism vary in their susceptibility to both peer rejection and acceptance would correspond to a differential susceptibility model. In sum, the current study informs about potential developmental variation in how specific genes and risk-promoting and development-enhancing environments interact to predict antisocial behavior and is one of the first studies into genetic moderation of peer relationship experiences on later (mal)adjustment (Brendgen, 2012).

Peer Relationship Quality as Predictor of Antisocial Behavior

One of the most important human needs in life is the need to belong, which motivates people to gain enduring, positive relationships with significant others (Baumeister & Leary, 1995). Although parents are important attachment figures especially in early childhood, when children transition into adolescence, peers become increasingly central for fulfilling the need to belong. That is, adolescents spend twice as much time with peers than with parents and tend to rely less on their parents as compared to their peers for problem solving and help (Agnew, 2003). Reasons for the increased importance of peers are that peer relations are less controlling, less judgmental, and more egalitarian than relationships with adults (Giordano, 1995).

Being accepted by peers thus provides adolescents with a sense of belonging to the peer group (e.g., Parker & Asher, 1993). Although rejection and acceptance should not be understood as opposite ends of a continuum but rather as distinct peer experiences that can coexist, rejection, just like the absence of acceptance, is associated with maladjustment.

Numerous studies showed that being rejected by peers has detrimental effects on the mental and social development of adolescents, and puts them at risk for later maladjustment, especially externalizing problems (Kupersmidt & Coie, 1990; Parker & Asher, 1987; Rubin, Bukowski, & Parker, 2006; Sentse, Lindenberg, Omvlee, Ormel, & Veenstra, 2010). It can be argued that externalizing problems such as aggression and rule-breaking behavior may be antecedents to *and* consequences of peer rejection. Several studies showed that peer rejection not only results from childhood aggressive behavior (Coie & Kupersmidt, 1983; Dodge, 1983) but also predicts continued aggression over the years (Dodge et al., 2003). More generally, peer rejection in late childhood is linked to both concurrent and later antisocial behavior (for a review, see Dodge, Coie, & Lynam, 2006). This effect even held when controlling for behavioral stability (Laird, Jordan, Dodge, Pettit, & Bates, 2001). In contrast, peer acceptance has been shown to have a positive effect on academic (O'Neil, Welsh, Parke, Wang, & Strand, 1997) and psychological adjustment (Ladd & Burgess, 2001).

Genetic Moderation of Peer Relationship Quality

Importantly, not all adolescents show negative or positive effects after peer rejection and acceptance, respectively. It stands to reason that sensitivity to the effects of peer relation characteristics is genetically influenced. For instance, Brendgen and colleagues (2008) showed that effects of peer victimization in kindergarten on aggression were moderated by genetic effects in that girls (but not boys) with a greater genetic susceptibility for aggression were more strongly affected by victimization.

Genetic factors are likely to affect the association particularly between exposure to peer rejection and antisocial behavior given the biological implications of social rejection. That is, several studies have provided support for neurobiological correlates of social rejection (Eisenberger, Lieberman, & Williams, 2003; Lau et al., 2011; Masten et al., 2009). Particularly the variation in amygdala activation observed by Lau and colleagues (2011) is of interest to the current study as individual differences in amygdala activation are partly due to *5-HTTLPR,* a common deletion polymorphism in the serotonin transporter–linked region (Munafò, Brown, & Hariri, 2008). That is, the amygdala has been found to be hyperreactive in carriers of the short allele,

increasing the time it takes to disengage from emotional stimuli (e.g., Furman, Hamilton, Joormann, & Gotlib, 2011; Hariri et al., 2002). Although much less is known about the biological effects of peer acceptance on later behavior, our study design enables us to explore whether *5-HTTLPR* also differentiates the effects of this positive measure of social interaction.

Serotonin Transporter Polymorphism: 5-HTTLPR

5-HTTLPR transcribes for a protein that is associated with reuptake speed of serotonin at brain synapses and constitutes an important source of variation in serotonin levels in the brain. Whereas the majority of people of Caucasian descent are carriers of the heterozygous short–long variant (~50%) of this polymorphism, the homozygous short–short variant is observed less often than the homozygous long–long variant (Noskova et al., 2008). Because of its reduced efficiency in terminating synaptic serotonin activity, the short allele has been treated as the risk allele in most studies (but, for a review on psychopathology associated with the long allele, see Glenn, 2011).

The *5-HTTLPR* short allele has been linked to personality traits such as neuroticism (Schinka, Busch, & Robichaux-Keene, 2004; Sen, Burmeister, & Ghosh, 2004) as well as mood disorders (Rosenthal et al., 1998). Short-allele carriers were also found to be slower in disengaging from emotional stimuli (Beevers, Wells, Ellis, & McGeary, 2009). The polymorphism is also implicated in behaviors and traits that show overlap with antisocial behavior. For instance, studies have reported associations between the short allele and substance use (Feinn et al., 2005) and higher levels of impulsivity (Paaver et al., 2007). Retz, Retz-Junginger, Supprian, Thome, and Rösler (2004) showed that the short allele was overrepresented in males displaying recurrent and overt violent behavior, whereas Sakai and colleagues (2010) showed that female carriers of the short allele were more likely to display chronic conduct problems. Lastly, Vaughn and colleagues (2009) reported a significant association between the serotonin transporter and pathological criminal behavior in youth.

In addition to direct associations, *5-HTTLPR* has been a candidate for moderation of adverse environmental effects on (mal)adjustment. Caspi and colleagues (2003) showed that child maltreatment was linked to depression especially for carriers of the short allele. Depression was also more common among female short–short variant carriers who reported high levels of perceived stress (Beaver, Vaughn, Wright, & DeLisi, 2012). Li and Lee (2010) found that *5-HTTLPR* interacted with child maltreatment in the prediction of covert problems in girls, again with short-allele carriers being at elevated risk. Similarly, Douglas et al. (2011) reported

a significant interaction between adverse childhood events and *5-HTTLPR* in the prediction of antisocial personality disorder (albeit for only a part of their sample). Vaske, Newsome, and Wright (2012) showed that the polymorphism interacted with childhood neglect in the prediction of criminal behavior and substance use.

The Present Study

The first aim of the current study was to test whether experiences of peer rejection and acceptance in preadolescence and early adolescence are associated with later antisocial behavior. The peer assessments of rejection and acceptance used in the current study are unique in that they span primary (preadolescence) as well as secondary school (early adolescence), thus covering an important transition in children's lives. This design enables us to examine associations between peer rejection/acceptance and later antisocial behavior by using different contexts. In line with previous studies, we hypothesized that, regardless of timing, higher levels of peer rejection are predictive of higher levels of antisocial behavior and that peer acceptance should be negatively associated with antisocial behavior. Secondly, we examined the association between *5-HTTLPR* and antisocial behavior, expecting greater risk of antisocial behavior for carriers of the short allele, particularly the short–short variant. Finally, we examined interaction effects between measures of peer rejection/acceptance and *5-HTTLPR*. A differential vulnerability model would be supported if carriers of the short allele are at greater risk for antisocial behavior in the presence of peer rejection but at equivalent risk as carriers of the long–long variant in the absence thereof. In contrast, a differential susceptibility model would be supported if adolescents carrying the short allele of the serotonin transporter polymorphism show increased antisocial behavior in the presence of peer rejection but also significantly decreased levels of antisocial behavior in the presence of peer acceptance.

Method

Sample and Participants

The present study includes data from three waves of the TRacking Adolescents' Individual Lives Survey (TRAILS). TRAILS is a prospective cohort study of Dutch adolescents, with biennial or triennial follow-up assessments. Data at the first assessment wave (Time 1 [T1]) were collected in 2001 and 2002 (mean age 11.1 years), at the second wave (T2)

in 2003 and 2004 (mean age 13.6 years), at the third wave (T3) in 2006 and 2007 (mean age 16.3 years), and at the fourth assessment (T4) wave in 2008–10 (mean age 19.1 years). The TRAILS target sample comprised young adolescents from five municipalities in the north of the Netherlands, including both urban and rural areas. Details about the study are published elsewhere (Huisman et al., 2008; Nederhof et al., 2012; De Winter et al., 2005). Both the parents and the children provided written consent for participation in the study.

Measures

Adolescent antisocial behavior. To measure antisocial behavior, we used T1 and T4 scores on the Antisocial Behavior Questionnaire (ASBQ), a measure comparable to the Self-Report Delinquency Scale (Moffitt & Silva, 1988). The ASBQ consists of 31 items at T1 (assessing lifetime) and 29 items at T4 (assessing the past 12 months) (e.g., "How often have you destroyed something on purpose?" and "How often have you used a weapon?"). Questions were rated as 0 = *never,* 1 = *once,* 2 = *two or three times,* 3 = *four to six times,* and 4 = *seven times or more*. For subsequent analyses, the mean of all items comprising the scale was taken. The internal consistency of the ASBQ scale was .88 at T1 and .82 at T4. We corrected for skew in the outcome variable by using square-root transformation.

Peer rejection and acceptance. At T1 and T2, a subsample of TRAILS participants took part in a classroom-based peer nomination assessment in which TRAILS participants and their classmates nominated each other on a range of domains (see also Dijkstra, Lindenberg, Verhulst, Ormel, & Veenstra, 2009; Veenstra et al., 2005). Peer nominations at T1 were collected only in classrooms with at least 10 TRAILS respondents. After selection of school classes and the agreement of the school to participate, schools provided the names of classmates of TRAILS respondents. Subsequently, classmates of regular TRAILS respondents were approached by their tutor and received an information letter in which they as well as their parents were asked to participate in TRAILS on this occasion only. If pupils and/or parents refrained from participation, they had to send a reply card within 10 days. This method of informed consent was used in order to maximize participation. A total of 98 pupils, of which three were regular TRAILS respondents, used this opportunity and refused to participate. Young people in special education (5.6% of the sample), those in small schools (6.4%), and those who repeated a grade (16.9%) or skipped a grade (2.2%) were excluded. As a result, the peer information can only be

generalized to a population who attend regular elementary schools and did not repeat a grade. At T1, 1,065 TRAILS respondents met these criteria and participated in peer nominations. As described in Table 1, an additional 639 children took part in this assessment, but these were not regular TRAILS participants, so genetic and behavioral data are missing.

At T2, peer nominations were conducted in classrooms with at least three regular TRAILS participants or with two participants on the condition that both had also participated in T1 peer nominations. (Again, peer nominations assessed were classroom-based, including non-TRAILS participants.) At T2, 1,007 TRAILS respondents participated in the peer nomination procedure. A total of 671 TRAILS respondents took part in both peer nomination studies.

The assessments of the peer nominations lasted for about 15 minutes and took place during regular lessons. After brief instructions in which a TRAILS staff member emphasized that information would be kept confidential, the participants received the questionnaire with the names of the classmates listed. The teacher and TRAILS staff member remained in the classroom during the administration of the peer nominations. Among other topics, adolescents were asked whom they disliked (rejection) and liked (acceptance), for which they could nominate an unlimited number

Table 1. Number of participants for each study variable

	Peer nominations		Antisocial behavior assessments	
Participants	T1	T2	T1	T4
Total	1,704	3,334	2,206	1,653
Number who are regular TRAILS participants	1,065	1,007	2,206	1,653
Number for whom genotype data are available	616	603	1,238	1,118[a]

Note. Missing data on predictor variables and covariate were imputed by using multiple imputation. T1 = Time 1; T2 = Time 2; T4 = Time 4; TRAILS = TRacking Adolescents' Individual Lives Survey.

[a]Number of participants on which analyses are based (availability of outcome measure and genotype information).

of classmates. The nominations received for being disliked were divided by the total number of children in the class—that is, the maximum number of nominations possible. The same procedure was applied to nominations received for being liked. These proportion scores take class size into account and range from 0 to 1, with higher scores indicating more rejection/acceptance. This procedure is commonly cited and a reliable way to treat peer nominations (cf. Bukowski & Hoza, 1989).

Genotyping for serotonin transporter polymorphism 5-HTTLPR. A subsample of TRAILS was genotyped at T3 of the study. DNA was extracted from blood samples (n = 1,190) or buccal swabs (n = 275) (Cytobrush; CooperSurgical, Trumbull, CT) by using a manual salting-out procedure as described by Miller, Dykes, and Polesky (1988). The *5-HTTLPR* polymorphism in the promoter region of *SLC6A4* (*5-HTT, SERT*) gene was genotyped by simple sequence-length analysis for 1,414 individuals. The length of the *5-HTTLPR* allele was determined by direct analysis on an automated capillary sequencer (ABI3730; Applied Biosystems, Nieuwerkerk aan den IJsel, The Netherlands). Genotyping was done at the research lab for multifactorial diseases within the Human Genetics Department of the Radboud University Nijmegen Medical Centre in Nijmegen, the Netherlands. The single-nucleotide substitution (A to G) present in the *HTTLPR* long (L) allele (rs25531) was genotyped by using a custom-made TaqMan assay (Applied Biosystems). Because the Lg polymorphism represents low serotonin expression comparable to the short (S) allele, S and Lg alleles were recoded S′ and La was recoded L′. We excluded all individuals who did not have full Dutch ancestry (n = 157) and one member of each sibling pair (n = 12) from the sample. As a result, serotonin transporter polymorphism data were available from 1,245 individuals (53% female).

Missing Data Treatment

The subsequent regression analyses are based on cases for which genetic information was available after accounting for sibship and ethnicity. We compared participants who took part in collection of genetic material to those from whom no such data are available on all study measures. Those with genetic data present showed lower levels of antisocial behavior at T1 ($M_{present}$ = 0.30, $SD_{present}$ = 0.32 vs. M_{absent} = 0.34, SD_{absent} = 0.38; t[2204] = 2.15, p = .03), were better liked at T1 ($M_{present}$ = 0.30, $SD_{present}$ = 0.15 vs. M_{absent} = 0.25, SD_{absent} = 0.16; t[1702] = −5.53, $p < .001$), and were less disliked at T1 ($M_{present}$ = 0.11, $SD_{present}$ = 0.12 vs. M_{absent} = 0.16, SD_{absent} = 0.16; t[1702] = 6.50, $p < .001$). No differences were observed with regard to like and dislike nominations received at T2 or antisocial behavior at T4.

Of those participants who provided genetic data, peer nominations at T1 were available for 616 and at T2 for 592. Antisocial behavior at T4 was assessed from 1,118 participants who also had genotype information. An overview of data availability for the measures used in this study is provided in Table 1.

To make full use of available data, we imputed data on peer rejection and acceptance and the covariate. This strategy was chosen given that the amount of missing data for antisocial behavior at T1 was negligible (1%) and peer nominations were missing at random (by design). Based on data presence for *5-HTTLPR,* we used the multiple imputation procedure *mi impute* in Stata 12 and based the imputation on a multivariate regression model composed of all variables included in subsequent models, as well as further auxiliary variables thought to increase the precision of imputed values (e.g., socioeconomic status, antisocial behavior at T2 and T3). Imputation models also included the dependent variable; however, following recommendations on treatment of imputed data, the subsequent analyses included only cases with data present on the outcome measure (Van Hippel, 2009). This procedure meant that analyses were conducted on 1,118 adolescents (genetic information but no T4 antisocial behavior, $n = 127$). A total of 25 imputed data sets were created, and analyses were conducted by using the *mi estimate* and *mibeta* macros in Stata 12 (White, Royston, & Wood, 2011). Descriptive statistics are presented for cases with data on the respective measure, but all subsequent analyses are based on imputed data. We note results obtained with complete cases wherever these differ meaningfully.

Analytic Strategy

We estimated two sets of regression models. The first set examined the effects of *5-HTTLPR* and peer rejection and acceptance in preadolescence (T1) on antisocial behavior in late adolescence (T4). The second set examined the effects of *5-HTTLPR* and peer rejection and acceptance in early adolescence (T2) on later antisocial behavior. Both sets were computed in three steps: (a) control variables (T1 antisocial behavior, gender) only, (b) additional estimation of main effects (peer rejection, peer acceptance, and genotype), and (c) additional estimation of interaction effect to identify whether the G $\times$ E explained variance above and beyond the main effects. Significant interaction effects were followed up by modeling the prediction of antisocial behavior at T4 by peer rejection (and peer acceptance, respectively) separately for each genotype variant. Finally, we examined gender-specific G $\times$ E effects by computing models that included a G $\times$ E $\times$ gender term.

Results

Descriptive Statistics

Descriptive statistics of all study variables are presented in Table 2. Allele frequencies for *5-HTTLPR* were in Hardy–Weinberg equilibrium ($p = .95$). We first examined genotype differences for environmental measures. Significantly different exposure to environmental measures suggests a gene–environment correlation and needs to be ruled out prior to examining G × E effects. No significant associations were found, carriers of the long–long, short–long, and short–short variants of *5-HTTLPR* did not differ in their experience of peer rejection in preadolescence, $F(2, 613) = 0.87$, $p = .42$, or early adolescence, $F(2, 589) = 0.29$, $p = .75$. Similarly, *5-HTTLPR* variant carriers did not differ with regard to peer acceptance in preadolescence, $F(2, 613) = 0.06$, $p = .94$, or early adolescence, $F(2, 600) = 1.03$, $p = .36$. A comparison of *5-HTTLPR* carrier mean levels of antisocial behavior at T1 did not yield a significant effect either, $F(2, 1235) = 0.35$, $p = .70$. We also tested for gender differences on environmental and outcome measures and found that girls reported lower antisocial behavior both at T1 ($t = -15.53$, $p < .001$) and T4 ($t = -9.25$, $p < .001$). Girls were further less likely to be rejected in preadolescence ($t = -7.07$, $p < .001$) and early adolescence ($t = -2.22$, $p = .03$) and more liked in preadolescence ($t = 2.33$, $p = .02$). No gender differences in peer acceptance were found for early adolescence.

Pairwise correlations between study variables are presented in Table 3. Antisocial behavior was considerably stable over time, as shown by

Table 2. Descriptive statistics of study measures

	M	*SD*	Range
Antisocial behavior T4	0.08	0.16	0–1.77
Antisocial behavior T1	0.32	0.35	0–2.84
Peer rejection T1	.14	.15	0–0.90
Peer rejection T2	.12	.13	0–0.84
Peer acceptance T1	.27	.16	0–0.80
Peer acceptance T2	.55	.20	0–1.00
	Long–long	Long–short	Short–short
5-HTTLPR	26.1%	50.4%	23.5%

Note. Descriptive statistics are based on cases for which data were available. T1 = Time 1; T2 = Time 2; T4 = Time 4.

Table 3. Pairwise correlations between study measures

		1	2	3	4	5	6
1	Antisocial behavior T4						
2	Antisocial behavior T1	.31***					
3	Peer rejection T1	.16**	.20***				
4	Peer rejection T2	.04	.04	.28***			
5	Peer acceptance T1	−.06	−.08	−.41***	−.22***		
6	Peer acceptance T2	.04	.03	−.22***	−.63***	.25***	
7	*5-HTTLPR*	.06	−.01	−.01	.02	−.01	.03

Note. Pairwise correlations are based on imputed data. T1 = Time 1; T2 = Time 2; T4 = Time 4.

*$p < .05$.

**$p < .01$.

***$p < .001$.

a significant association between assessments at T1 and T4. Antisocial behavior at T4 was also linked to higher levels of peer rejection in preadolescence but not early adolescence. The same was true for antisocial behavior at T1. Neither measure of antisocial behavior was associated with peer acceptance, although we found moderate to strong negative associations between peer rejection and acceptance at both times and also considerable stability of peer rejection and peer acceptance from preadolescence to early adolescence.

Regression Models

We began by computing regressions in which only gender and antisocial behavior at T1 functioned as predictors of antisocial behavior at T4 (Step 1). These models were equivalent in both sets of regression analyses reported next. Antisocial behavior in preadolescence significantly predicted antisocial behavior later, as did gender, with boys being at greater risk.

Peer Rejection and Acceptance

T1 × 5-HTTLPR. Results for this model are presented in Table 4. *5-HTTLPR* was associated with antisocial behavior at T4, even after accounting for baseline antisocial behavior. No prediction was found for peer acceptance, and the prediction of peer rejection just missed

Table 4. Regression model for prediction of late adolescent antisocial behavior by rejection and acceptance in preadolescence and *5-HTTLPR* polymorphism

	Antisocial behavior T4					
	Step 1		Step 2		Step 3	
	B	β	*B*	β	*B*	β
Gender (0 = female, 1 = male)	.08	.18***	.08	.18***	.08	.18
ASBQ (T1)	.18	.25***	.17	.23***	.17	.23
Peer rejection (T1)			.02	.09	−.02	−.08
Peer acceptance (T1)			.01	.01	−.01	−.06
5-HTTLPR (0 = LL, 1 = LS, 2 = SS)			.02	.06*	.02	.07*
5-HTTLPR * Peer rejection (T1)					.04	.21*
5-HTTLPR * Peer acceptance (T1)					.01	.08
R^2		.12		.14		.15

Note. Regression models are based on imputed data. LL = long–long; LS = long–short; SS = short–short; T1 = Time 1; T2 = Time 2; T4 = Time 4.

*$p < .05$.

**$p < .01$.

***$p < .001$.

statistical significance ($p = .09$). In the third step, the interactions between peer rejection in preadolescence and *5-HTTLPR,* as well as between peer acceptance in preadolescence and *5-HTTLPR,* were added, yielding a significant interaction effect for peer rejection × *5-HTTLPR*. We also computed separate regression models for peer rejection and acceptance, which confirmed these results. That is, no prediction of antisocial behavior by peer acceptance was found. In contrast, peer rejection significantly predicted antisocial behavior in the second step ($\beta = .10$, $p = .01$) as well as in interaction with *5-HTTLPR* in the third step ($\beta = .19$, $p = .01$). We followed this up and computed simple slopes to examine the prediction of antisocial behavior at T4 by peer rejection separately for genotype variants, controlling again for all other variables in the model. Whereas the prediction of antisocial behavior by peer rejection at T1 was significant for carriers of the short–short version of *5-HTTLPR* ($\beta = .07$, $p = .002$), this association was smaller in effect size and just significant for carriers of the short–long ($\beta = .03$, $p = .04$) and not significant for

carriers of the long–long variant of the genotype ($\beta = -.02$, $p = .47$) (see Figure 1). All effects were marginally stronger when only complete cases were used for analyses. Finally, we examined whether this G × E differed by gender but found no support (*5-HTTLPR* × peer rejection T1 × gender: $\beta = -.01$, $p = .96$).

T2 × 5-HTTLPR. Results for this model are presented in Table 5. Neither peer rejection nor acceptance in early adolescence were predictive of antisocial behavior at T4, but a significant effect of *5-HTTLPR* was yielded again. This effect remained when the interaction terms (genotype × peer rejection and genotype × peer acceptance) were added to the model. However, no significant interaction effects were found. All results obtained from complete case analyses were similar to the ones obtained with imputed cases.

Discussion

Our study is one of the first to examine the interplay of *5-HTTLPR* with peer rejection and acceptance in the prediction of late adolescent antisocial behavior. In line with recent studies on aspects of peer relationships (Benjet, Thompson, & Gotlib, 2010; Kretschmer, Dijkstra, Ormel,

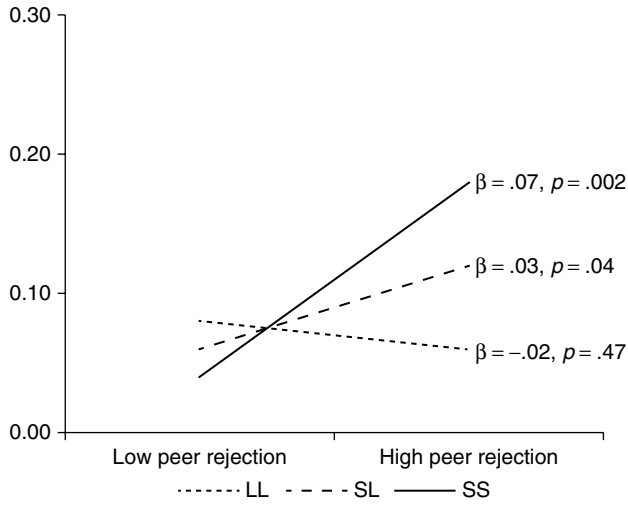

Figure 1. Prediction of antisocial behavior at Time 4 (T4) (square-root-transformed scale) by peer rejection for long–long (LL), short–long (SL), and short–short (SS) variants of *5-HTTLPR*. Low and high rejection represent 1 *SD* from the mean. Values are based on imputed data.

Table 5. Regression model for prediction of late adolescent antisocial behavior by rejection and acceptance in early adolescence and 5-HTTLPR polymorphism

	Antisocial behavior T4					
	Step 1		Step 2		Step 3	
	B	β	*B*	β	*B*	β
Gender (0 = female, 1 = male)	.08	.18***	.08	.18***	.08	.18***
ASBQ (T1)	.18	.25***	.18	.24***	.18	.24***
Peer rejection (T2)			.01	.05	.01	.05
Peer acceptance (T2)			.01	.06	−.01	−.04
5-HTTLPR (0 = LL, 1 = LS, 2 = SS)			.02	.06*	.02	.06*
5-HTTLPR * Peer rejection (T2)					.01	.02
5-HTTLPR * Peer acceptance (T2)					.02	.13
R^2		.12		.13		.14

Note. Regression models are based on imputed data. LL = long–long; LS = long–short; SS = short–short; T1 = Time 1; T2 = Time 2; T4 = Time 4.

*$p < .05$.

**$p < .01$.

***$p < .001$.

Verhulst, & Veenstra, 2013; Sugden et al., 2010) and similar outcome measures (Latendresse et al., 2011; Lee, 2011), we showed that variation in particular genotypes partly affects vulnerability to negative (though not positive) peer exposure. Moreover, the current study set out to explore whether taking a developmental perspective in examining the environment in G × E studies might provide a more thorough understanding of the bio-social interplay associated with behavioral development.

When examined separately, our results showed a significant prediction of late adolescent antisocial behavior by peer rejection in preadolescence but not early adolescence. For peer rejection in preadolescence, this association was especially pronounced for carriers of the short–short variant, which is in line with our hypothesis as well as the findings in previous G×E studies on this polymorphism (Douglas et al., 2011; Li & Lee, 2010).

A G×E can function in two ways: If carrying a particular genotypic variant not only increases the risk for maladjustment under negative conditions but also elevates the positive effect of nourishing environments on development, this interplay suggests a differential susceptibility mechanism

(Ellis et al., 2011). In contrast, if carrying a specific genotypic variant only increases the risk posed by negative environments (e.g., maltreatment, peer rejection) but levels of maladjustment do not vary in the absence thereof (or presence of positive environment), a dual-risk or differential vulnerability model is present.

We were fortunate to be able to test which of these models applied to our data and found support for the latter, given that the interaction between peer acceptance and genotype was not predictive of adolescent antisocial behavior. However, short–short variant carriers were particular vulnerable to peer rejection. This mechanism might be based (partly) on prolonged amygdala reactivity following social rejection (Lau et al., 2011). As suggested by Munafò et al. (2008), carriers of the *5-HTTLPR* short allele show amygdala hyperreactions when presented with negative emotional stimuli and need significantly more time than do carriers of other variants to disengage from such stimuli. Thus, carriers of the short allele, particularly the short–short homozygous variant, may be more likely to show prolonged amygdala activity after being exposed to peer rejection. The amygdala is strongly related to the neurotransmitter systems that are implicated in behavior, including forms of problem behavior (DeLisi, Umphress, & Vaughn, 2009). Moreover, amygdala activity and dysfunction have been related to psychopathy (Blair, 2007). Psychopathy describes a developmental disorder that is characterized by low levels of guilt, high levels of impulsivity and narcissism, and poor behavioral control. Psychopathy, particularly its callous-unemotional component, and antisocial behavior overlap considerably (Frick & White, 2008), which supports the assumption that amygdala functioning is implicated in antisocial behavior and that this association may be affected by serotonin transporter polymorphism. Although some associations have been reported between amygdala impairment and antisocial behavior (e.g., Raine & Yang, 2006), more research, particularly studies that employ neurobiological measures, are needed in order to better understand how amygdala activity translates into antisocial behavior.

It is curious that no association was found between peer rejection in early adolescence and late adolescent antisocial behavior, but a few interpretations are tenable. For instance, young adolescents might engage more often with peers outside of their immediate classroom environment. At an age when adolescents spend most of their spare time with self-chosen peers and friends, peer relations in the classroom might count less and difficulties in the classroom context may not have a long reach. In other words, adolescents at T2 may simply be less affected by what their classmates think about them because most of their social life happens outside of the

classroom. In addition, individuals who engage in antisocial behavior in late adolescence might already be on the track to do so by the second time that peer rejection was assessed (13.6 years). If this is true, being rejected by classroom peers may not pose as much of a risk anymore. Finally, whereas assessments of peer rejection in preadolescence were conducted in primary school settings—that is, taken in classrooms that should have been relatively stable for the years prior to T1—the second assessment took place in the early years of secondary school. Thus, the level of peer rejection at T1 might represent an ongoing and repeated experience that started much earlier and may have carried the risk for long-term negative consequences (e.g., antisocial behavior). In contrast, T2 assessments were conducted in a relatively new environment and from a different set of peers.

Ellis and colleagues (2011) called for an examination into whether G × E effects hold for different time points and developmental stages. The design of our study—relatively simple environmental assessments carried out at different time points that span chronologically proximal yet developmentally distinct stages in life—enabled us to to so. Fascinatingly, we found the predicted associations and genetic moderation when rejection was assessed in preadolescence but not when assessed in early adolescence. This result may be an indication for developmental variation with regard to the gene–environment interplay. Thus, particular negative environments might pose a risk only when experienced at a particular point in life. In addition, individual genetic vulnerability may also change, and not finding a significant effect of the interaction between *5-HTTLPR* and early adolescent rejection may suggest developmental variation in the G × E interplay. Our interpretation is tentative and requires further empirical support. That is, the interaction effect found for preadolescent rejection may not be a developmentally specific phenomenon but simply suffer from nonreplicability.

Limitations and Future Directions

Our results need to be interpreted with several limitations in mind. First, we focused on *5-HTTLPR* because of its role in predicting externalizing forms of behavior and as moderator of associations between environmental risk and maladjustment, but antisocial behavior is a multifaceted and polygenic phenomenon. Selecting *5-HTTLPR* as candidate gene for this study was driven by hypotheses regarding this polymorphism's implication in constructs similar to the one examined here (antisocial behavior) and because *5-HTTLPR* has been discussed as a susceptibility gene (Belsky & Pluess, 2009). While our results were more in favor of a differential vulnerability model, genetic

susceptibility may be better captured by using cumulative plasticity indices that consist of several polymorphisms (Belsky & Pluess, 2009). In addition to additive genetic vulnerability or susceptibility, interactions between different genes are likely, and many more environmental factors contribute to this form of maladjustment. Examining characteristics of peers themselves (e.g., antisocial behavior) in addition to characteristics of the relationship can shed further light on the role of genetic moderation of peer context. For instance, Beaver et al. (2011) showed that affiliating with delinquent peers is substantially genetically influenced; hence, a next step may be to explore the role that particular candidate genes may play in that regard.

Second, although our analyses benefited from a longitudinal and multireporter design, we employed self-report measures of antisocial behavior, which are not free of bias. Since young people may overreport or underreport their involvement, official data or peer reports would be suitable instruments to increase the quality of such data. However, despite the shortcomings of self-reports, Thornberry and Krohn (2000) suggested that self-reports are a valid way to assess adolescent delinquency. In addition, notwithstanding their value as a more objective measure than self-reports, we lost a substantial amount of data because peer rejection could not be assessed for every TRAILS participant. Finally, the participants in our study showed very low levels of antisocial behavior, particularly at T4, reducing the amount of variance that could be explained in this measure. This pattern might affect the representativity of the data. Further, the reference to the previous year (compared to lifetime at T1) might have contributed to this low average level of antisocial behavior. However, these limitations are likely to underestimate rather than overestimate the effects we yielded. In addition to using self-reports, we employed an overall measure of antisocial behavior, but different patterns might emerge for aggressive and nonaggressive forms. Aggressive behavior was rare in our sample, and subscales of aggressive and nonaggressive behavior showed considerable lower reliability than the combined measure. However, more detailed and varied assessments of aggressive behavior may circumvent this problem.

Most importantly, our findings need to be replicated in an independent sample. Many G$\times$E effects have been found only in particular samples or within a particular range of environmental exposure, but interaction effects need to be replicated to substantiate findings (Asherson & Price, 2012). Whereas it would have been satisfying to present a replication of results, our study design is unique in incorporating peer nominations from different ages, longitudinal measures, and a sufficient sample size to detect genetic main and interaction effects.

In sum, despite its limitations, the current study provided insight into the interplay of peer relation quality and *5-HTTLPR* in the prediction of adolescent antisocial behavior. We showed that variation in vulnerability to preadolescent peer rejection is partly due to genetic differences. This also means that genetic effects are expressed particularly under adverse environmental conditions—posing a double risk for some adolescents. We did not find such genetic moderation for peer acceptance, which supports a differential vulnerability rather than a differential susceptibility model. Whereas modifying and adjusting genetic factors, even if it were possible, is not ethical, findings like ours also point out that changing the environment may be particularly important for some young people. In other words, if certain environmental risks, such as peer rejection in preadolescence, are especially harmful for some children (those that carry specific genetic variants), efforts should go into studying ways to prevent or intervene in these adverse environments to ensure healthy development.

References

Agnew, R. (2003). An integrated theory of the adolescent peak in offending. *Youth & Society, 34,* 263–299.

Asherson, P., & Price, T. S. (2012). Commentary: Replication, replication, replication: The continued need to substantiate G×E effects in child psychopathology—A response to Laucht et al. (2012). *Journal of Child Psychology and Psychiatry, 53,* 360–362.

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.

Beaver, K. M., Gibson, C. L., Turner, M. G., DeLisi, M., Vaughn, M. G., & Holand, A. (2011). Stability of delinquent peer associations: A biosocial test of Warr's sticky-friends hypothesis. *Crime & Delinquency, 57,* 907–927.

Beaver, K. M., Vaughn, M. G., Wright, J. P., & DeLisi, M. (2012). An interaction between perceived stress and 5-HTTLPR genotype in the prediction of stable depressive symptomatology. *American Journal of Orthopsychiatry, 82,* 260–266.

Beevers, C. G., Wells, T. T., Ellis, A. J., & McGeary, J. E. (2009). Association of the serotonin transporter gene promoter region (5-HTTLPR) polymorphism with biased attention for emotional stimuli. *Journal of Abnormal Psychology, 118,* 670–681.

Belsky, J., & Pluess, M. (2009). Beyond diathesis-stress: Differential susceptibility to environmental influence. *Psychological Bulletin, 135,* 885–908.

Benjet, C., Thompson, R. J., & Gotlib, I. H. (2010). 5-HTTLPR moderates the effect of relational peer victimization on depressive symptoms in adolescent girls. *Journal of Child Psychology and Psychiatry, 51,* 173–179.

Berndt, T. J. (1982). The features and effects of friendship in early adolescence. *Child Development, 53,* 1447–1460.

Blair, R. J. R. (2007). The amygdala and ventromedial prefrontal cortex in morality and psychopathy. *Trends in Cognitive Science, 11,* 387–392.

Brendgen, M. (2012). Genetics and peer relations: A review. *Journal of Research on Adolescence, 22,* 419–437.

Brendgen, M., Boivin, M., Vitaro, F., Girard, A., Dionne, G., & Pérusse, E. (2008). Gene–environment interaction between peer victimization and child aggression. *Development and Psychopathology, 20,* 455–471.

Bukowski, W. M., & Hoza, B. (1989). Popularity and friendship: Issues in theory, measurement, and outcomes. In T. J. Berndt & G. W. Ladd (Eds.), *Peer relations in child development* (pp. 15–45). New York: Wiley.

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

Caspi, A., McClay, J., Moffitt, T. E., Mill, J., Martin, J., Craig, I. W., . . . Poulton, R. (2002). Role of genotype in the cycle of violence in maltreated children. *Science, 297,* 851–854.

Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A., Craig, I. W., Harrington, H., . . . Poulton, R. (2003). Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science, 301,* 386–389.

Coie, J. D., & Kupersmidt, J. B. (1983). A behavioral analysis of emerging social status in boys' groups. *Child Development, 54,* 1400–1416.

DeLisi, M., Umphress, Z. R., & Vaughn, M. G. (2009). The criminology of the amygdala. *Criminal Justice and Behavior, 36,* 1241–1252.

De Winter, A., Oldehinkel, A. J., Veenstra, R., Brunnekreef, J. A., Verhulst, F. C., & Ormel, J. (2005). Evaluation of non-response bias in mental health determinants and outcomes in a large sample of pre-adolescents. *European Journal of Epidemiology, 20,* 173–181.

Dijkstra, J. K., Lindenberg, S., Verhulst, F. C., Ormel, J., & Veenstra, R. (2009). The relation between popularity and antisocial behaviors: Moderating effects of athletic abilities, physical attractiveness, and prosociality. *Journal of Research on Adolescence*, 19, 401–413.

Dodge, K. A. (1983). Behavioral antecedents of peer social status. *Child Development, 54,* 1386–1389.

Dodge, K. A., Coie, J. D., & Lynam, D. (2006). Aggression and antisocial behavior in youth. In W. Damon (Series Ed.), R. M. Lerner & N. Eisenberg (Vol. Eds.), *Handbook of child psychology: Vol. 3. Social, emotional, and personality development* (6th ed., pp. 719–788). New York: Wiley.

Dodge, K. A., Lansford, J. E., Burks, V. S., Bates, J. E., Pettit, G. S., Fontaine, R., . . . Price, J. M. (2003). Peer rejection and social information-processing factors in the development of aggressive behavior problems in children. *Child Development, 74,* 374–393.

Dodge, K. A., & Pettit, G. S. (2003). A biopsychosocial model of the development of chronic conduct problems in adolescence. *Developmental Psychology, 39,* 349–371.

Douglas, K., Chan, G., Gelernter, J., Arias, A. J., Anton, R. F., Poling, J. . . . Kranzler, H. R. (2011). 5-HTTLPR as a potential moderator of the effects of adverse childhood experiences on risk of antisocial personality disorder. *Psychiatric Genetics, 21,* 240–248.

Eisenberger, N. I., Lieberman, M. D., & Williams, K. D. (2003). Does rejection hurt? An fMRI study of social exclusion. *Science, 203,* 290–292.

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

Feinn, R., Nellissery, M., & Kranzler, H. R. (2005). Meta-analysis of the association of a functional serotonin transporter promoter polymorphism with alcohol dependence. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics, 133,* 79–84.

Frick, P. J., & White, S. F. (2008). The importance of callous-unemotional traits for developmental models of aggressive and antisocial behavior. *Journal of Child Psychology and Psychiatry, 49,* 359–375.

Furman, D. J., Hamilton, J. P., Joormann, J., & Gotlib, I. H. (2011). Altered timing of amygdala activation during sad mood elaboration as a function of 5-HTTLPR. *Social Cognitive and Affective Neuroscience, 6,* 270–276.

Giordano, P. C. (1995). The wider circle of friends in adolescence. *American Journal of Sociology, 101,* 661–697.

Glenn, A. L. (2011). The other allele: Exploring the long allele of the serotonin transporter gene as a potential risk factor for psychopathy: A review of the parallels in findings. *Neuroscience and Biobehavioral Reviews, 35,* 612–620.

Hariri, A. R., Mattay, V. S., Tessitore, A., Kolachana, B., Fera, F., Goldman, D., . . . Weinberger, D. R. (2002). Serotonin transporter genetic variation and the response of the human amygdala. *Science, 297,* 400–403.

Huisman, M., Oldehinkel, A. J., De Winter, A., Minderaa, R. B., De Bildt, A., Huizink, A. C., . . . Ormel, J. (2008). Cohort profile: The Dutch 'TRacking Adolescents' Individual Lives Survey'; TRAILS. *International Journal of Epidemiology, 37,* 1227–1235.

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.

Kupersmidt, J. B., & Coie, J. D. (1990). Preadolescent peer status, aggression, and school adjustment as predictors of externalizing problems in adolescence. *Child Development, 61,* 1350–1362.

Ladd, G. W., & Burgess, K. B. (2001). Do relational risks and protective factors moderate the linkages between childhood aggression and early psychological and school adjustment? *Child Development, 72,* 1579–1601.

Laird, R. D., Jordan, K., 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 problems. *Development and Psychopathology, 13,* 337–354.

Latendresse, S. J., Bates, J. E., Goodnight, J. A., Lansford, J. E., Budde, J. P., Goate, A., . . . Dick, D. M. (2011). Differential susceptibility to adolescent externalizing trajectories: Examining the interplay between *CHRM2* and peer group antisocial behavior. *Child Development, 82,* 1797–1814.

Lau, J. Y. F., Guyer, A., Tone, E. B., Jenness, J., Parrish, J. M., Pine, D. S., & Nelson, E. E. (2011). Neural responses to peer rejection in anxious adolescents: Contributions from the amygdala-hippocampal complex. *International Journal of Behavioral Development, 36,* 36–44.

Lee, S. S. (2011). Deviant peer affiliation and antisocial behavior: Interaction with monoamine oxidase A (MAOA) genotype. *Journal of Abnormal Child Psychology, 39,* 321–332.

Li, J. J., & Lee, S. S. (2010). Latent class analysis of antisocial behavior: Interaction of serotonin transporter genotype and maltreatment. *Journal of Abnormal Child Psychology, 38,* 789–801.

Mason, D. A., & Frick, P. J. (1994). The heritability of antisocial behavior: A meta-analysis of twin and adoption studies. *Journal of Psychopathology and Behavioral Assessment, 16,* 301–322.

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

Miller, S. A., Dykes, D. D., & Polesky, H. F. (1988). A simple salting out procedure for extracting DNA from human nucleated cells. *Nucleic Acids Research, 16,* 1215.

Moffitt, T. E., & Silva, P. A. (1988). Self-reported delinquency, neuro-psychological deficit, and history of attention deficit disorder. *Journal of Abnormal Child Psychology, 16,* 553–569.

Munafò, M. R., Brown, S. M., & Hariri, A. R. (2008). Serotonin transporter (5-HTTLPR) genotype and amygdala activation: A meta-analysis. *Biological Psychiatry, 63,* 852–857.

Nederhof, E., Jorg, F., Raven, D., Veenstra, R., Verhulst, F. C., Ormel, J., & Oldehinkel, A. J. (2012). Benefits of extensive recruitment effort persist

during follow-ups and are consistent across age group and survey method. The TRAILS study. *BMC Medical Research Methodology, 12,* 93.

Noskova, T., Pivac, N., Nedic, G., Kazantseva, A., Gaysina, D., Faskhutdinova, G., . . . Muck Seler, D. (2008). Ethnic differences in the serotonin transporter polymorphism (5-HTTLPR) in several European populations. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 32,* 1735–1739.

O'Neil, R., Welsh, M., Parke, R. D., Wang, S., & Strand, C. (1997). A longitudinal assessment of the academic correlates of early peer acceptance and rejection. *Journal of Clinical Child Psychology, 26,* 290–303.

Paaver, M., Nordquist, N., Parik, J., Harro, M., Oreland, L., & Harro, J. (2007). Platelet MAO activity and the 5-HTT gene promoter polymorphism are associated with impulsivity and cognitive style in visual information processing. *Psychopharmacology, 194,* 545–554.

Parker, J. G., & Asher, S. R. (1987). Peer acceptance and later personal adjustment: Are low-accepted children "at risk"? *Psychological Bulletin, 102,* 357–389.

Parker, J. G., & Asher S. R. (1993). Friendship and friendship quality in middle childhood: Links with peer group acceptance and feelings of loneliness and social dissatisfaction. *Developmental Psychology, 29,* 611–621.

Raine, A. (2002). Biosocial studies of antisocial and violent behavior in children and adults: A review. *Journal of Abnormal Child Psychology, 30,* 313–326.

Raine, A., & Yang, Y. (2006). Neural foundations to moral reasoning and antisocial behavior. *Social Cognition and Affective Neuroscience, 1,* 203–213.

Retz, W., Retz-Junginger, P., Supprian, T., Thome, J., & Rösler, M. (2004). Association of serotonin transporter promoter gene polymorphism with violence: Relation with personality disorders, impulsivity, and childhood ADHD psychopathology. *Behavioral Sciences & the Law, 22,* 415–425.

Rhee, S. H., & Waldman, I. D. (2002). Genetic and environmental influences on antisocial behavior: A meta-analysis of twin and adoption studies. *Psychological Bulletin, 128,* 490–529.

Rose-Krasnor, L. (1997). The nature of social competence: A theoretical review. *Social Development, 6,* 111–135.

Rosenthal, N. E., Mazzanti, C. M., Barnett, R. L., Hardin, T. A., Turner, E. H., Lam, G. K., . . . Goldman, D. (1998). Role of serotonin transporter promoter repeat length polymorphism (*5-HTTLPR*) in seasonality and seasonal affective disorder. *Molecular Psychiatry, 3,* 175–177.

Rubin, K. H., Bukowski, W., & Parker, J. G. (2006). Peer interactions, relationships, and groups. In W. Damon (Vol. Ed.), R. M. Lerner & N. Eisenberg (Vol. Eds.), *Handbook of child psychology: Vol. 3, Social, emotional, and personality development* (6th ed., pp. 571–645). New York: Wiley.

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

Sakai, J. T., Boardman, J. D., Gelhorn, H. L., Smolen, A., Corley, R. P., Huizinga, D., . . . Stallings, M. C. (2010). Using trajectory analyses to refine phenotype for genetic association: Conduct problems and the serotonin transporter (5-HTTLPR). *Psychiatric Genetics, 20,* 199–206.

Schinka, J. A., Busch, R. M., & Robichaux-Keene, N. (2004). A meta-analysis of the association between the serotonin transporter gene polymorphism (5-HTTLPR) and trait anxiety. *Molecular Psychiatry, 9,* 197–202.

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

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.

Sugden, K., Arseneault, L., Harrington, H., Moffitt, T. E., Williams, B., & Caspi, A. (2010). Serotonin transporter gene moderates the development of emotional problems among children following bullying victimization. *Journal of the American Academy of Child & Adolescent Psychiatry, 49,* 830–840.

Thornberry, T. P., & Krohn, M. D. (2000). The self-report method of measuring delinquency and crime. In D. Duffee (Ed.), *Measurement and analysis of crime and justice: Criminal justice 2000* (pp. 33–83). Washington, DC: Office of Justice Programs, US Department of Justice.

Van Hippel, P. T. (2009). How to impute squares, interactions, and other transformed variables. *Sociological Methodology, 39,* 265–291.

Vaske, J., Newsome, J., & Wright, J. P. (2012). Interaction of serotonin transporter linked polymorphic region and childhood neglect on criminal behavior and substance use for males and females. *Development and Psychopathology, 24,* 181–193.

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

Veenstra, R., Lindenberg, S., Oldehinkel, A. J., De Winter, A. F., Verhulst, F. C., & Ormel, J. (2005). Bullying and victimization in elementary schools: A comparison of bullies, victims, bully/victims, and uninvolved preadolescents. *Developmental Psychology, 41,* 672–682.

White, I. R., Royston, P., & Wood, A. M. (2011). Multiple imputation using chained equations: Issues and guidance for practice. *Statistics in Medicine,* 30, 377–399.

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