Developmental Psychology
2016, Vol. 52, No. 11, 1893–1903

0012-1649/16/$12.00 http://dx.doi.org/10.1037/dev0000166

Relations Between Three Dopaminergic System Genes, School Attachment, and Adolescent Delinquency

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Both environmental factors and genetic variation, particularly in genes responsible for the dopaminergic system such as *DRD4*, *DRD2*, and *DAT1 (SLC6A3)*, affect adolescent delinquency. The school context, despite its developmental importance, has been overlooked in gene-environment research. Using data from the National Institute of Child Health and Human Development Study of Early Child Care and Youth Development (NICHD ECCYD), this study examined key interactions between school attachment and (a) each of the *DRD4*, *DRD2*, and *DAT1 (SLC6A3)* genotypes; and (b) a polygenic score. Results indicate that there is a main effect of school attachment, unlike genetic variation, on delinquency. Interestingly, there are important interactive effects of school attachment and dopaminergic genotypes on delinquency. Carriers of the *DRD2*-A1 allele were differentially affected by both positive and negative school environments, whereas *DAT1*-10R carriers fared the same as 9R homozygotes in poorer and moderate school environments, but fared disproportionately better in more positive environments. Contrary to expectations, youth without the *DRD4*-7R allele were particularly affected by the school environment. These findings contribute to the literature considering the roles of both context and genes in delinquency research, and inform our understanding of the individual-level traits that influence sensitivity to particular contexts.

Keywords: dopamine, genetics, adolescence, delinquency, school attachment

It is well-understood that both genetic and environmental factors work together to shape development (Mullineaux & DiLalla, 2015) and to affect adolescent delinquency (Dmitrieva & Espel, 2014; Turkheimer, 2000). Researchers acknowledge that specific genetic variants may interact with particular environments to affect delinquency (Caspi et al., 2003; Moffitt, 2005). Equally difficult as identifying "which gene" and "what mechanism" is the challenge of focusing on the "environment" (see Salvatore & Dick, 2015). Studies of such gene-environment (G×E) interactions have typically focused on the family, peer, or neighborhood contexts. Surprisingly, prior G×E delinquency research has largely ignored the school context, despite the fact that school is essential for promoting positive youth development and reducing delinquency involvement (Brookmeyer Fanti, & Henrich, 2006; Hirschi, 1969; Li et al., 2011; Thornberry, 1987). Not all school contexts are the same, however, and equally important, not all contexts influence adolescents in the same manner. The dopamine D4 (*DRD4*) receptor is important to consider in regards to susceptibility to delinquency and context not only because *DRD4* variation has been found to be associated with delinquency (Beaver et al., 2007; Boutwell & Beaver, 2008), but also because it is implicated in various G×E interactions for adolescent delinquency (see Bakermans-Kranenburg & van IJzendoorn, 2006; Barnes & Jacobs, 2013; Beaver, Gibson, DeLisi, Vaughn, & Wright, 2012; Janssens et al., 2015; Sheese, Voelker, Rothbart, & Posner, 2007). Despite the strong links between school context and delinquency, studies have yet to examine whether the school context is more influential for individuals with the *DRD4*-7R allele.

Schools, Genetic Variation, and Delinquency

Although G×E studies have largely focused on the influence of the home and neighborhood contexts on delinquency, other contexts become increasingly important in the development of prosocial behaviors during adolescence. Youth spend more time in school than in any other environment (Roeser, Eccles, & Sameroff, 2000), thus it is unsurprising that school becomes particularly important for promoting the skills, competencies, and values that enable adolescents to successfully transition into adulthood and promote prosocial behaviors (Hirschfield & Gasper, 2011; Thornberry, 1987; Wang & Eccles, 2013; Wang & Holcombe, 2010). Yet despite its salience to positive youth development, schools have received far less attention than family or friends in G×E studies of delinquency.

One customary method of analyzing the school context is through the youth's perceptions of the school. How a youth feels about his or her school is critical for development during adolescence. School attachment is characterized by close affective relationships with the school and is an important component of a variety of developmental theories (Catalano, Haggerty, Oesterle, Fleming, & Hawkins, 2004; Hirschi, 1969). Youth who are more

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attached to school are less likely to engage in a variety of delinquent behaviors (Hawkins, Doueck, & Lishner, 1988), including adolescent alcohol use (Henry & Slater, 2007), cigarette smoking, marijuana use (Dornbusch, Erickson, Laird, & Wong, 2001), and many antisocial behaviors (for reviews see Loukas, Roalson, & Herrera, 2010; Monahan, Oesterle, & Hawkins, 2010).

Due to the abundant evidence linking school attachment to adolescent externalizing behaviors, G×E studies are beginning to examine the school context as an influential, promotive environment for reducing adolescent delinquency. Beaver (2011) found that the association between genetics and delinquency was stronger if youth were less attached to school. However, because the authors used sibling pairs to quantify genetic influences, the particular genes involved in the G×E were unclear. To our knowledge, only two studies by Guo, Roettger, and Cai (2008) and Boardman et al. (2014) have examined how specific genes interact with school attachment to affect adolescent delinquency, and both focused on genes affecting the dopaminergic system. Before summarizing their results, however, a brief explanation of the dopaminergic system and the key genes is necessary.

The Dopaminergic System and Delinquency

Dopamine is a neurotransmitter that regulates brain activity, specifically the modulation of responses to rewards and punishments (Cherek & Pietras, 2003; Killcross, 2003). Activation of the dopaminergic pathway increases physiological arousal and intense feelings of well-being or pleasure (Heath, 1964), and can produce greater cravings for pleasurable and exciting experiences (Dmitrieva & Espel, 2014). Dopaminergic functioning is associated with a variety of behaviors during adolescence, including substance abuse (Boutwell & Beaver, 2008) and delinquency (Burt & Mikolajewski, 2008). Several genes that regulate the dopaminergic system are also associated with adolescent delinquency. For example, the dopamine transporter gene *DAT1* (*SLC6A3*) codes for the dopamine transporter protein that limits dopamine receptor activation (Bannon & Whitty, 1995). *DAT1* variation has been found to be associated with adolescent delinquency (Guo, Roettger, & Shih, 2007). Similarly, variants of *DRD2*, or the dopamine receptor D2 Taqman1A polymorphism, that encode the production of the dopamine receptor D2 have been found to be associated with delinquency (Guo, Roettger, & Cai, 2008). Finally, the dopamine D4 receptor (*DRD4*) gene has received considerable attention in the behavioral genetics field because it is associated with dopaminergic transmission, especially in the prefrontal cortex (Asghari et al., 1995). *DRD4* is highly polymorphic with a range of two to 11 tandem repeats in the protein-coding region (Ding et al., 2002). The presence of the 7-repeat (7R) allele has been associated with a blunted response to dopamine (Asghari et al., 1995; Wang et al., 2004). Individuals with the 7R allele tend to engage in behaviors that enable them to achieve the levels of dopamine necessary for activation, such as sensation seeking (Benjamin et al., 1996; Ebstein et al., 1996), impulsivity (Congdon, Lesch, & Canli, 2008), and risk taking (Dreber et al., 2009; Roussos, Giakoumaki, & Bitsios, 2009). It is thus unsurprising that *DRD4* has been linked to a variety of maladaptive behaviors during adolescence, including antisocial behavior (Beaver et al., 2012), attention deficit hyperactivity disorder (ADHD; Grady et al., 2003; Li, Sham, Owen, & He, 2006), addiction (Olsson et al., 2011), and psychopathy (Wu & Barnes, 2013).

DRD4 genetic variation, however, is inconsistently related to maladaptive adolescent behaviors. For example, whereas some studies find that *DRD4* allelic variation is associated with aggression (Fresan et al., 2007), others do not find an association (Vassos, Collier, & Fazel, 2014) or find the reverse pattern (Janssens et al., 2015). Studies of *DRD4*'s relation to adolescent delinquency are also inconsistent. Although several studies using the National Longitudinal Study of Adolescent Health (Add Health) dataset found that *DRD4* variation was directly related to adolescent delinquency (Beaver et al., 2007; Boutwell & Beaver, 2008), Beaver and colleagues found that youth with a *DRD4*-7R genotype are more likely to *desist* from delinquency (Beaver, Wright, DeLisi, & Vaughn, 2008). Two subsequent studies, one using the Tracking Adolescents' Individual Lives Survey (TRAILS) dataset (Kretschmer, Dijkstra, Ormel, Verhulst, & Veenstra, 2013) and the other using the Mannheim Study of Children at Risk (Hohmann et al., 2009), also did not find that *DRD4* variation was directly related to adolescent delinquency.

The seemingly contradictory findings with *DRD4* may indicate that carrying the *DRD4*-7R allele is not universally favorable or adverse, rather its effects likely depend on the context studied (see Grady et al., 2013; Janssens et al., 2015; Kretschmer et al., 2013). For instance, Kretschmer et al., (2013), using the peer environment, found that genetic variation in *DRD4* was associated with increased risk in the presence of negative environmental conditions, but also lowered risk in more favorable conditions. This aligns with the differential susceptibility hypothesis, positing that individuals with "sensitive" genotypes are expected to be disproportionately affected by the environment in a "for better or for worse" fashion (Belsky & Pluess, 2009). Based on their genotype, certain individuals may function more poorly in negative contexts, but may function disproportionately better in positive contexts (Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van Ijzendoorn, 2011). Indeed, children with the 7R allele but who experience more positive parenting or neighborhood conditions display less externalizing behavior (Bakermans-Kranenburg & van IJzendoorn, 2006), sensation seeking (Sheese, Voelker, Rothbart, & Posner, 2007), and propensity for violence (Barnes & Jacobs, 2013; Beaver et al., 2012) than their non-7R counterparts. These findings suggest that the 7R allele may serve as a "context-susceptibility" or a "plasticity" trait. Across these studies, the youth's experience with the parenting or neighborhood environment moderates the relation between the *DRD4*-7R allele and delinquency.

Dopaminergic Genes and the School Environment

Just as a G×E interaction may be specific to a particular developmental *period* (see Belsky & Pluess, 2009), a G×E interaction may be unique to a particular developmental *context*. In line with ecological theory (Bronfenbrenner, 1979; Bronfenbrenner & Morris, 1998), a variety of contexts likely alter how genetic variation affects development (Belsky & Pluess, 2009). Considering school's increasing importance during adolescence, it is important to examine whether this particular context during this particular developmental period also moderates the relation between dopaminergic system genes and delinquency. Indeed, a few

studies have begun this examination, particularly focusing on *DRD2* because of its association with externalizing problem behaviors (Beaver et al., 2007). Guo et al. (2008) found that the effects of *DRD2* variants on serious delinquency were conditional on school attachment. Consistent with these findings, Boardman et al. (2014) found that *DRD2* and *DAT1* variants were associated with serious delinquency if youth are not attached to school. Guo et al. (2008) and Boardman et al. (2014) found similar results using two nationally representative data sets. Therefore, there is compelling evidence for the G×E interaction between dopaminergic genes and school attachment predicting delinquency. However, two important questions remain. First, despite its salience in the delinquency literature, to our knowledge, no studies have examined whether *DRD4* variants interact with school attachment to affect delinquency. Findings are thus far limited to *DRD2* and *DAT1*. Although both *DRD2* and *DRD4* are implicated in the development of antisocial behaviors (Rowe, 2002), *DRD2* and *DRD4* may not have consistent main effects on antisocial phenotypes (Beaver et al., 2007). Indeed, other studies have found that variation in *DRD4*, but not *DRD2*, is related to delinquency (Boutwell & Beaver, 2008). However, studies have not yet considered whether *DRD4* variants affect the relation between school attachment and delinquency.

Second, there is reason to believe that the relation between school attachment and adolescent delinquency may vary by sex. Previous research indicates that school attachment is only related to male delinquency (Cernkovich & Giordano, 1992; Freidenfelt Liljeberg, Eklund, Fritz, & Klinteberg, 2011; Rosenbaum & Lasley, 1990). However, some studies find that school attachment also affects female delinquency (Hart & Mueller, 2013; Torstensson, 1990) or does not differ by sex (Dornbusch et al., 2001). Similarly, there is some evidence that G×E interactions may also vary by sex. Studies have found that *DRD4* variation is related to delinquency but only for male adolescents (Dmitrieva, Chen, Greenberger, Ogunseitan, & Ding, 2011). Taking such findings into account, it is important to consider sex when evaluating the associations between genetic susceptibility, school context, and adolescent delinquency.

The present study examined how the interaction between the school context and *DRD4, DRD2,* and *DAT1* variants may predict adolescent delinquency. Based on prior research on school attachment and delinquency, we expected that adolescents who were less attached to school would engage in more delinquency than those who were more attached to school. In line with the differential susceptibility hypothesis and prior work examining the relation between school attachment, genetic variants, and delinquency, we expected individuals with the *DRD4*-7R allele to be more sensitive to the effects of the school context. That is, adolescents who were less attached to their school and who have the 7R allele would engage in more delinquency than those who do not have the 7R allele. Based on findings in the literature (Boardman et al., 2014; Zhang et al., 2015), we also expected individuals with the *DRD2*-A1 allele and those with the *DAT1*-10R allele to be more sensitive to the effects of the school context. Considering the mixed findings on sex differences in the link between delinquency and school attachment, we also examined whether the interactions between school attachment, genotype, and delinquency varied by sex. Based on limited prior research (Freidenfelt Liljeberg et al., 2011), we expected that school attachment may be a better determinant of delinquency for males than for females. Finally, we tested the interaction between the school context and a polygenic score, expecting to find that as the score increased, the effects of both positive and negative school attachment would increase.

Method

Participants

Participants were enrolled in the National Institute of Child Health and Development Study of Early Child Care and Youth Development (NICHD SECCYD), a longitudinal study conducted in 10 research sites across the United States (for details on recruitment and selection procedures see NICHD Early Child Care Research Network, 2005). Youth self-identified their races, and 83% of the adolescents identified as White/Caucasian, 10% as Black/African American, and 7.1% as other.

Measures

Demographics. Mother's education was used as a proxy for socioeconomic status. When the youth were 15-years-old, mothers reported the highest level of education they had achieved. Approximately 10.2% had not received a high school diploma, 21.1% had received a high school diploma, 33.4% attended college but did not receive a degree, and 35.4% had received at least a college diploma. Mother's education was included as a categorical predictor in all models with less than a high school diploma as the reference group. Race was also included as a categorical predictor in all models with White youth as the comparison group.

Genotype. DNA extraction and genotyping was performed at the Genome Core Facility in the Huck Institute of the Life Sciences at the Pennsylvania State University under the direction of Deborah S. Grove, Director for Genetic Analysis. Buccal mucosa cells were self-collected from participants with a cotton swab. We used three candidate genes implicated in the functioning of the dopaminergic neurotransmitter system (Belsky & Pluess, 2009). The assay for genotyping the *DRD4* VNTR was based on methods developed by Sander et al. (1997) and modified by Anchordoquy, McGeary, Liu, Krauter, and Smolen (2003). The Genomics Core Facility modified it further as follows: 1 x Taq Gold Buffer, 2.25 mM final concentration of MgCl2, 10% DMSO, 0.2 mM dNTPs, 0.1 mM deazo GTP, 0.75 uM primers, 40 ng of DNA and 1 U of Taq Gold (Applied Biosystems, Foster City CA) in a volume of 12 μl. The primer sequences were: forward, 5′-AGGACCCTCATGGCCTTG 3′ (fluorescently labeled), and reverse, 5′-GCGACTACGTGGTCTAC TCG-3′. This method results in PCR products of (in bp): 379, 427, 475, 523, 571, 619, 667, 715, 763, and 811 (i.e., corresponding to the 2–10 repeat alleles, respectively). After amplification all VNTR fluorescent-labeled products were analyzed using the 3730XL DNA Analyzer (Applied Biosystems, Foster City, CA). One half μl of LIZ-500 standard and 10 μls of formamide were added to 1 μl of the PCR and products were separated on a 50 cm capillary array. Genotypes were identified using Genotyper software v4.0 (Applied Biosystems, Foster City, CA). All samples were genotyped twice, and approximately 5% could not be genotyped in the subsample. In line with Belsky et al. (2015), higher values were assigned to hypothesized plasticity variants of the markers. *DRD4* genotype was dichotomously coded based on the

number of 7-repeat alleles, such that *DRD4*-7R$^+$ included those who were either homozygous or heterozygous for the 7-repeat allele (coded 1; $n = 176$) and *DRD4*-7R$^-$ included those without a single copy of the 7-repeat allele (coded 0; $n = 458$). As in similar work (e.g., Berry, McCartney, Petrill, Deater-Deckard, & Blair, 2014), repeat alleles greater than 7 were coded as 7$^-$ based on prior work indicating that higher number repeats (e.g., 10-repeat) display functional properties that are more similar to those with lower number of repeats (e.g., 2-repeat) than to those with 7-repeats (Jovanovic, Guan, & Van Tol, 1999). Results were reanalyzed using a sample of just individuals possessing at least one copy of the 7-repeat allele (coded 1: $n = 161$) versus those without a single copy of the 7-repeat allele, and results did not change.) For *DRD2* (polymorphism in *ANKK1*; rs1800497), individuals with the A1 allele were coded as 1 ($N = 257$), and A2/A2 homozygotes were coded 0 ($N = 433$). For *DAT1* (40-bp VNTR; *SLC6A3*), individuals homozygous for 9R were coded 0 ($N = 50$), heterozygous 10R/9R coded 1 ($N = 218$), and homozygous for 10R coded 2 ($N = 382$). Similar to Belsky et al. (2015), a small number (approximately 5%) of individuals who did not receive final genotypes of 9/9, 9/10, or 10/10 were dropped from the analysis of *DAT1*.

School attachment. At age 15, participants completed the "What My School is Like" questionnaire. This measure was based on a similar measure in the "New Hope Study" and the Adolescent Health dataset (see Cernkovich & Giordano, 1992; Crosnoe, Johnson, & Elder, 2004; Flexon, 2015), however, responses were modified into a 4-point scale. Using a 4-point scale ranging from *not at all true* to *very true*, participants answered 19 questions pertaining to their attachment to school (e.g., "*I feel close to others at my school*" and "*I am happy to be at my school;*" $M = 3.32$, $SD = .55$). The scale was internally consistent ($\alpha = .75$).

Delinquency. Participants self-reported engaging in delinquent behaviors using the 11-item Delinquent Behavior subscale of the Youth Self-Report (YSR; Achenbach & Rescorla, 2001) at age 15. Participants were asked to rate how well each item describes him or her within the last 6 months. All items (e.g., "*I destroy things belonging to others*") were answered using a 3-point scale (0 = *not true*, 1 = *somewhat or sometimes true*, 2 = *very true or often true*). We used the delinquent behavior *t*-score, which is composed of items pertaining to antisocial behaviors such as cheating, truancy, stealing, alcohol, and drug use ($M = 53.98$, $SD = 6.12$). *T* scores could range from 50–100, with higher scores indicating higher levels of delinquent behavior. The subscale had adequate internal consistency ($\alpha = .70$).

Plan of Analysis

To begin, we examined whether males and females differed on key variables. We then assessed whether delinquency and school attachment varied by genotype, within each sex. Regression analyses were then conducted in four steps. First, to examine whether genetic variation was related to delinquency, delinquency was regressed on each genotype, adjusting for mother's education, race, site, and sex. Second, to examine whether the relation between genotype and delinquency varied by sex, delinquency was regressed on the interaction between genotype and sex, adjusting for the same set of covariates. Third, delinquency was regressed on school attachment to identify a potential main effect of school attachment, adjusting for covariates. Fourth, to examine whether the relation between school attachment and delinquency varied by genotype, delinquency was regressed on the interaction between school attachment and genotype, adjusting for the same set of covariates. Considering school attachment may be a better determinant of delinquency for males than for females, we regressed delinquency on the three-way interaction between genotype, sex, and school attachment. Finally, based on results from the independent tests of the genotypes, a polygenic score was tested. To calculate the polygenic score, based on results from the first set of models, *DRD4* was reverse-coded (i.e., 7R$^-$ coded 1), *DRD2* was coded dichotomously into individuals with the A1 allele coded as 1, and *DAT1* was recoded dichotomously into individuals with at least one 10R allele coded as 1. A polygenic score was calculated by weighting each genotype by the strength of its independent interaction term, then summing the values (see Harrison & Bookheimer, 2016). The interaction between the polygenic score and school attachment was tested, adjusting for the same set of covariates.

Results

Sample Characteristics

The *DRD4* groups ($\chi^2 = 1.94$, $p = .585$), *DRD2* groups ($\chi^2 = 5.16$, $p = .161$), and *DAT1* groups ($\chi^2 = 5.49$, $p = .482$) did not differ on mother's education status. Males and females did not differ on *DRD4* genotype ($\chi^2 = 0.10$, $p = .756$), *DRD2* genotype ($\chi^2 = 0.93$, $p = .336$), or *DAT1* genotype ($\chi^2 = 2.95$, $p = .229$). Males and females both engaged in the same level of delinquency, $t(690) = -1.424$, $p = .155$. Females were more attached to school, $t(690) = -2.826$, $p = .005$. Within each sex, neither delinquency nor school attachment differed by genotype (see Table 1).

Predicting Delinquency

First, delinquency was regressed on each dopaminergic gene, adjusting for mother's education, race, site, and sex. Consistent with Dmitrieva, Chen, Greenberger, Ogunseitan, and Ding (2011) among others (e.g., Chhangur et al., 2015), there was no main genetic effect on delinquency (Tables 2–4). Second, to examine whether the relation between each dopaminergic gene and delinquency varied by sex, delinquency was regressed on the interaction between each dopaminergic gene and sex, adjusting for the same set of covariates. None of the interactions were significant, indicating that the relation between these genetic markers and delinquency did not vary by youth sex.

Third, delinquency was regressed on school attachment, adjusting for the same covariates, to examine whether school attachment was related to delinquency. Results indicate a main effect of school attachment on delinquency ($b = -3.75$, $SE = 0.41$, $p < .001$). Fourth, delinquency was regressed on the interaction between school attachment and each genotype, adjusting for the same set of covariates. Results indicate a significant interaction between *DRD4*-7R status and school attachment ($F = 27.43$, $p < .001$; Table 2; Figure 1). The slope for those without the 7R allele ($b = -4.39$, $SE = 0.50$) differed from the slope for those with the 7R allele ($b = -1.97$, $SE = 0.77$; $dydx = 2.41$, $p = .008$). At 2 *SD* below the mean on school attachment, youth with the 7R

Table 1
Descriptive Statistics by Sex and Genotype

Variable	Males							Females						
		DRD4		*DRD2*		*DAT1*			*DRD4*		*DRD2*		*DAT1*	
	All	7R$^-$ (N = 228)	7R$^+$ (N = 84)	A2/A2 (N = 206)	A1 (N = 132)	9R/9R (N = 25)	10R/9R (N = 115)	10R/10R (N = 174)	All	7R$^-$ (N = 230)	7R$^+$ (N = 92)	A2/A2 (N = 227)	A1 (N = 125)	9R/9R (N = 25)	10R/9R (N = 103)	10R/10R (N = 208)
Delinquency *M* (*SD*)	53.65 (5.76)	53.84 (6.12)	53.64 (5.23)	53.31 (5.54)	54.24 (6.16)	53.88 (6.09)	54.27 (6.89)	53.39 (5.12)	54.31 (6.45)	54.12 (6.17)	54.56 (6.76)	54.25 (6.26)	54.12 (.57)	56.16 (9.81)	53.90 6.30)	54.14 (5.74)
School attachment *M* (*SD*)	3.26 (.61)**	3.27 (.61)	3.18 (.62)	3.29 (.62)	3.18 (.61)	3.27 (.51)	3.20 (.63)	3.28 (.62)	3.37 (.55)	3.37 (.47)	3.37 (.50)	3.38 (.50)	3.37 (.44)	3.53 (.49)	3.37 (.45)	3.36 (.50)

$^*p < .05$. $^{**}p < .01$. $^{***}p < .001$.

engaged in significantly less delinquency than those without the 7R allele (*dydx* = −2.66, *SE* = 1.11, 95% CI [−4.84, −0.47], *p* = .017). At 1 *SD* below the mean on school attachment, youth with the 7R engaged in somewhat less delinquency than those without the 7R allele (*dydx* = −1.31, *SE* = 0.70, 95% CI [−2.69, 0.07], *p* = .064). At the mean on school attachment, youth engaged in the same amount of delinquency regardless of DRD4 allele (*dydx* = 0.47, *SE* = 0.51, 95% CI [−0.96, 1.06], *p* = .927). At 1 *SD* above the mean, youth with the 7R allele engaged in more delinquency than those without the 7R allele (*dydx* = 1.40, *SE* = 0.74, 95% CI [−0.06, 2.85], *p* = .060), and this difference increased at 2 *SD* above the mean (*dydx* = 2.75, *SE* = 1.16, 95% CI [0.46, 5.03], *p* = .018). That is, compared with youth with the 7R allele, youth *without* the 7R allele were more affected by both positive and negative school contexts.

The interaction between *DRD2* and school attachment was significant ($F = 28.52$, $p < .001$; Figure 2; Table 3>). The slope for A1 carriers ($b = -5.12$, $SE = 0.68$) was significantly different from that for A2/A2 homozygotes ($b = -2.78$, $SE = 0.51$; *dydx* = −2.34, *p* = .006), indicating that the former were more affected by the school context. Youth with the A1 allele engaged in more delinquency than A2/A2 heterozygotes at both 2 *SD* (*dydx* = 2.78, *SE* = 1.03, 95% CI [0.75, 4.80], *p* = .007) and 1 *SD* below the mean on school attachment (*dydx* = 1.47, *SE* = 0.65, 95% CI [0.20, 2.74], *p* = .023). At the mean level of school attachment, youth engaged in the same amount of delinquency regardless of genotype (*dydx* = 0.16, *SE* = 0.46, 95% CI [−0.75, 1.07], *p* = .728). At 1 *SD* above the mean of school attachment, carriers of the A1 allele engaged in the same amount of delinquency as A2/A2 homozygotes (*dydx* = −1.15, *SE* = 0.67, 95% CI [−2.47, 0.17], *p* = .089), yet significantly less at 2 *SD* above the mean (*dydx* = −2.46, *SE* = 1.07, 95% CI [−4.55, −0.36], *p* = .022). In short, compared with A2/A2 homozygous youth, youth with the A1 allele were significantly more affected by both positive and negative attachment to school.

The interaction between *DAT1* and school attachment was also significant ($F = 16.44$, $p < .001$; Figure 3; Table 4). The slope for those with the 10R/10R ($b = -3.55$, $p < .001$) and the slope for those with 10R/9R ($b = -.4.42$, $p < .001$) were significantly different from zero, whereas the slope for 9R/9R was not ($b = -2.38$, $p = .139$). However, the slopes were not significantly different from each other (see Table 4). At 2 *SD* below the mean on school attachment, youth with the 10R/10R allele (*dydx* = −0.39, *SE* = 2.20, 95% CI [−4.72, 3.94], *p* = .861) and youth with the 10R/9R allele (*dydx* = 0.50, *SE* = 2.29, 95% CI [−3.99, 4.50], *p* = .827) engaged in the same amount of delinquency as those with the 9R/9R genotype. Similarly, at 1 *SD* below the mean on school attachment, youth with the 10R/10R allele (*dydx* = −1.04, *SE* = 1.39, 95% CI [−3.77, 1.68], *p* = .453) and youth with the 10R/9R allele (*dydx* = −0.65, *SE* = 1.44, 95% CI [−3.74, 2.18], *p* = .654) engaged in the same amount of delinquency as those with the 9R/9R genotype. However, at the mean on school attachment, youth with the 10R/10R allele (*dydx* = −1.70, *SE* = 0.88, 95% CI [−3.43, 0.30], *p* = .054) and youth with the 10R/9R allele (*dydx* = −1.79, *SE* = 0.92, 95% CI [−3.61, 0.02], *p* = .053) engaged in less delinquency than those with the 9R/9R genotype (though *p* values are close to .05). Similarly, at 1 *SD* above the mean on school attachment, youth with the 10R/10R allele (*dydx* = −2.35, *SE* = 1.18, 95% CI

Table 2
Multiple Regression Analysis of Delinquency on School Attachment, Sex, and DRD4 Genotype

Variable	Model 1 *b* (*SE*)	Model 2 *b* (*SE*)	Model 3 *b* (*SE*)	Model 4 *b* (*SE*)	Model 5 *b* (*SE*)
Genotype					
DRD4–7R[A]	.02 (.54)	−.30 (.78)		.05 (.51)	−.29 (.75)
Sex[B]	.62 (.48)	.45 (.57)	1.12* (.44)	.93* (.46)	.72 (.54)
School Attachment			−3.74*** (.41)	−4.39*** (.50)	−4.57*** (.62)
Genotype × Sex		.62 (1.08)			.72 (1.03)
Genotype × School Attachment				2.41** (.91)	2.67* (1.19)
Sex × School Attachment					.57 (1.02)
Genotype × Sex × School Attachment					−.88 (1.85)
Constant	51.16*** (2.25)	51.20*** (2.26)	52.12*** (2.39)	50.04*** (2.14)	50.11*** (2.14)
R^2	.03	.04	.15	.15	.15
R^2_{adjusted}	.02	.01	.13	.13	.12

Note. Standard errors in parentheses. Mother's education, ethnicity, and site were included as controls.
[A] DRD4–7R allele coded 1. [B] Female coded 1.
* $p < .05$. ** $p < .01$. *** $p < .001$.

[−4.68, −0.03], $p = .047$) and youth with the 10R/9R allele (*dydx* = −2.94, *SE* = 1.26, 95% CI [−5.40, −0.47], $p = .002$) engaged in less delinquency than youth with the 9R/9R allele. That is, compared with youth without the 10R allele, youth with the 10R allele were more positively influenced by positive school attachment.

We then tested whether the relation between genotype, school attachment, and delinquency varied by sex by regressing delinquency on the three-way interaction between *DRD4* genotype, sex, and school attachment. We repeated analyses for *DRD2* and *DAT1*. None of the interactions between genotype, school attachment, and sex reached significance. This finding suggested that the relation between genotype, school attachment, and delinquency was similar for both sexes.

Finally, we regressed delinquency on the interaction between the polygenic score and school attachment, adjusting for the same covariates as in previous models. Results indicate that the model fit the data ($F = 6.12$, $p < .001$; $R^2 = .159$, $R^2_{adjusted} = .133$) and that the interaction was significant ($b = -0.857$, $SE = 0.25$, 95% CI [−1.35, −0.36], $p = .001$; $f_{omnibus} = 26.30$, $p < .001$; Figure 4). As the polygenic score increased, the effects of both positive and negative school attachment increased.

Reanalysis: Whites Only

Due to concerns about the multiethnic sample, all analyses were rerun focusing exclusively on the White subsample. This resulted in only very modest changes to the coefficients and did not change any results. Specifically, the G×E interactions all remained consistent.

Discussion

During adolescence, youth spend more time in school than in any other context, and youth who are more attached to school tend to engage in less delinquency. Yet genetics research has yet to uncover why the school context, either positive or negative, may be more influential for some adolescents. Despite the importance of school on reducing adolescent delinquency and *DRD4*'s prominence in the general gene-by-environment interaction literature,

Table 3
Multiple Regression Analysis of Delinquency on School Attachment, Sex, and DRD2 Genotype

Variable	Model 1 *b* (*SE*)	Model 2 *b* (*SE*)	Model 3 *b* (*SE*)	Model 4 *b* (*SE*)
Genotype				
DRD2[A]	.26 (.49)	.83 (.68)	.16 (.46)	.16 (.66)
Sex[B]	.67 (.47)	1.11 (.60)	1.09* (.45)	1.12* (.57)
School attachment			−2.78*** (.51)	−2.64*** (.65)
DRD2[A] × Sex		−1.14 (.97)		−.07 (.93)
DRD2[A] × School Attachment			−2.34*** (.84)	−2.67* (1.06)
Sex × School Attachment				−.36 (1.02)
DRD2[A] × Sex × School Attachment				.99 (1.78)
Constant	51.20*** (2.17)	50.74*** (2.16)	49.84*** (2.05)	49.86*** (2.06)
R^2	.05	.04	.15	.15
R^2_{adjusted}	.02	.02	.13	.12

Note. Standard errors in parentheses. Mother's education, ethnicity, and site were included as controls.
[A] DRD2 A1 allele Coded 1. [B] Female coded 1.
* $p < .05$. ** $p < .01$. *** $p < .001$.

Table 4
Multiple Regression Analysis of Delinquency on School Attachment, Sex, and DAT1 Genotype

Variable	Model 1 *b* (*SE*)	Model 2 *b* (*SE*)	Model 3 *b* (*SE*)	Model 4 *b* (*SE*)
Genotype				
DAT1 10R/9R[A]	−1.35 (.97)	−.14 (1.36)	−1.79 (.92)	−.53 (1.29)
DAT1 10R/10R[A]	−1.68 (.93)	−1.23 (1.32)	−1.70 (.88)	−1.01 (1.24)
Sex[B]	.65 (.49)	2.03 (1.73)	.97* (.47)	2.84 (1.70)
School attachment			−2.38 (1.60)	−2.37 (2.32)
DAT1 10R/9R[A] × Sex		−2.50 (1.93)		−2.82 (1.89)
DAT1 10R/10R[A] × Sex		−.94 (1.85)		−1.59 (1.81)
DAT1 10R/9R[A] × School Attachment			−2.04 (1.76)	−2.20 (2.48)
DAT1 10R/10R[A] × School Attachment			−1.17 (1.69)	−1.09 (2.42)
DAT1 10R/9R[A] × Sex × School Attachment				1.79 (3.67)
DAT1 10R/10R[A] × Sex × School Attachment				.68 (3.48)
Constant	52.74*** (2.29)	52.01*** (2.45)	51.98*** (2.18)	51.06*** (2.36)
R^2	.05	.05	.15	.16
$R^2_{adjusted}$	.02	.02	.13	.12

Note. Standard errors in parentheses. Mother's education, ethnicity, and site were included as controls.
[A] DAT1 9R/9R homozygotes coded 0. [B] Female coded 1.
* $p < .05$. ** $p < .01$. *** $p < .001$.

the present study is the first to examine whether *DRD4* variants affect the relation between school attachment and adolescent delinquency. Because dopaminergic gene variation has been related to differential susceptibility to a number of different environments (such as home environments and neighborhoods), we expected similar results for school environments. Consistent with other studies of school attachment (e.g., Boardman et al., 2014; Guo et al., 2008), in general, youth who were more attached to school reported less delinquency than those who were less attached to school. To our surprise, we found that youth *without* the 7R allele were particularly affected by the school environment. Results therefore indicate that school attachment does not affect reported delinquency for adolescents with the *DRD4*-7R allele. These adolescents engaged in similar amounts of delinquency regardless of how attached they were to their school. It is unclear why adolescents with a *DRD4*-7R genotype are unaffected by school attachment, yet these findings align with those of Kretschmer et al. (2013) who found that the association between peer victimization and delinquency was not significant for 7R allele carriers. It is possible that the hypothesized "plasticity" of this dopamine variant is only expressed at particular developmental stages (Grady et al., 2013). By the age of participants in this study (15-years-old), environmental effects such as school attachment may have minimal impact, especially if other nonschool environmental factors, not measured in the current study, predominate. Clearly, further work is necessary to uncover the "window" of the hypothesized *DRD4*-7R "plasticity," and what factors contribute to differential sensitivity of individuals with a *DRD4*-7R allele.

Findings with *DRD2* are more consistent with expectations, such that carriers of the A1 allele are differentially affected by both positive and negative school environments. These results are consistent with Zhang et al. (2015) who found that A1 carriers are more susceptible to negative parenting, but fare better under more positive parenting conditions. Finally, results for the *DAT1* indicate that 10R carriers fare the same as 9R homozygotes in poorer and moderate school environments, but

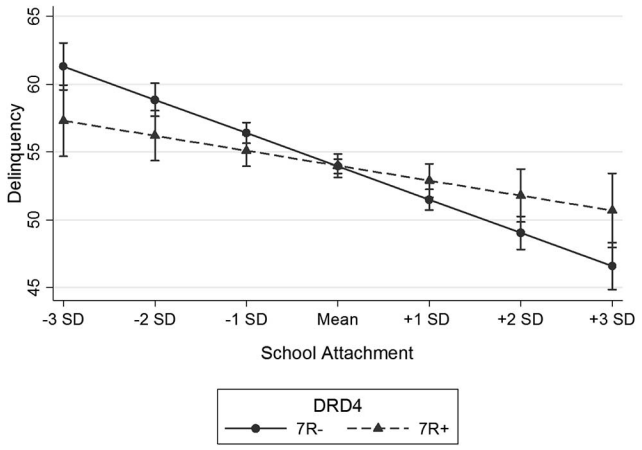

Figure 1. Two-way interaction between school attachment and DRD4 genotype predicting delinquency.

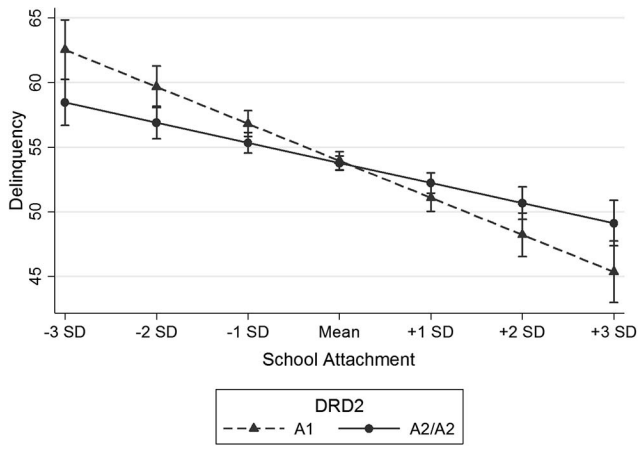

Figure 2. Two-way interaction between school attachment and DRD2 genotype predicting delinquency.

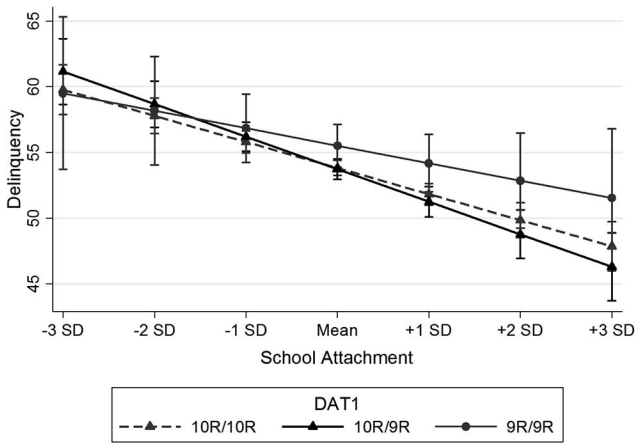

Figure 3. Two-way interaction between school attachment and DAT1 genotype predicting delinquency.

fare disproportionately better in more positive environments. Results for *DAT1* are therefore somewhat consistent with Boardman et al. (2014) who found that *DAT1* genotype was a protective factor among those with the strongest attachment to their schools. However, the results of this study did not indicate that they fare more poorly in more negative school environments. Thus, results of this study are the first to indicate that the direction, degree, and form of the dopaminergic gene by school environment interaction may vary for each of these three dopaminergic system genes.

Several other findings are noteworthy. To our knowledge, this is one of the first studies to examine whether school attachment varied by genetic variation (see Boardman et al., 2014; Guo et al., 2008), and the first to focus on *DRD4,* particularly comparing it with other related genes. The *DRD4*-7R allele is related to impulsivity, hyperactivity, and inattention (Congdon et al., 2008; Grady et al., 2003; Li et al., 2006) that are related to academic failure and classroom disruption (Frick et al., 1991). If individuals with the 7R allele experience these symptoms and behavioral problems, it is likely that they would experience less school bonding, and consequently engage in more delinquency. However, we did not find any evidence that individuals with the *DRD4*-7R allele experience significantly lower levels of school attachment or engage in more delinquency. This suggests that our findings are not merely due to genetic differences in school attachment. Youth with and without the 7R allele are equally attached to school. Second, our use of a distinct, large dataset contributes to the finding in the literature that genetic effects are not deterministic for delinquency (Bakermans-Kranenburg & van Ijzendoorn, 2006; Beaver et al., 2012). Third, although we found that females were more attached to their schools, the relation between school attachment and delinquency did not differ by sex. In addition, the interaction between genotype and delinquency did not differ by sex. These findings indicate that the way genetic variation and school attachment interact to influence delinquency is consistent for males and females. This emphasizes that school attachment is important for affecting delinquency involvement for both sexes.

This study extends previous research in several important ways. First, we contribute to the growing body of G×E literature that considers environments beyond peers, family, and neighborhoods. Only a handful of studies have considered the school context. Considering these results are not entirely consistent with findings with other contexts, further research on this particular context is clearly important. Regardless, this study points to a promising avenue of research on gene-by-school interactions. Second, this study extends the limited extant literature by considering a large, multisite dataset. Prior research on G×E interactions incorporating school context has used data from the National Longitudinal Study of Adolescent to Adult Health (Beaver, 2011; Guo et al., 2008) and the NYSFS (Boardman et al., 2014). Although these large, longitudinal data sets are well-suited to answer questions pertaining to G×E interactions and school settings, it is important that studies replicate questions using alternative data sets, such as reported here. Third, this study examined a constellation of genetic polymorphisms specifically associated with dopamine receptors and transporters. Given that researchers are increasingly examining additive or cumulative genetic plasticity (e.g., Belsky & Beaver, 2011) and the complex genetic/environmental nature of delinquency, further work should investigate additional genetic polymorphisms and their possible interactions (see Boutwell et al., 2014).

While this study extends our understanding on the gene by school environment interaction, several limitations of this study should be noted. First, the measures included in this study were obtained from youth self-reports. School attachment is inherently subjective, and reports of school environment and delinquency are subject to reporter bias. It would be advantageous for future research to incorporate a broader multimethod, multiinformant approach. Prior research has found more objective indicators of school environment, such as school size (Chen & Vazsonyi, 2013), to be related to school behavior and delinquency. Therefore, additional objective indicators of school environment should be considered in future analyses. It is important to note, however, that objective quality measures of the school environment may mask the individual's experiences. Therefore, subjective measures of school context, such as school attachment, may be able to better capture the effect of school on delinquent behavior.

A second important limitation is that despite the large sample size, our sample was also highly educated, predominantly White, and

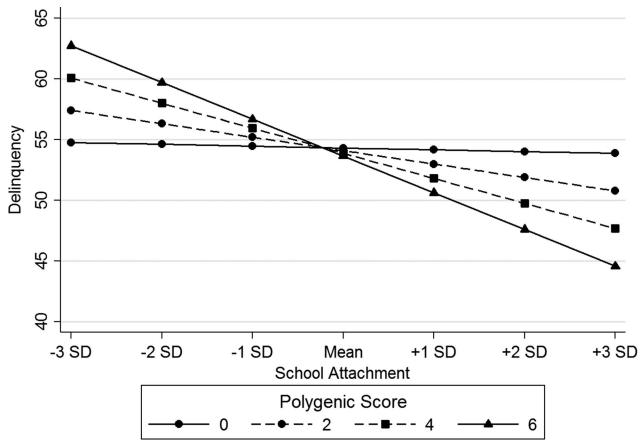

Figure 4. Two-way interaction between school attachment and polygenic score predicting delinquency.

engaged in low levels of delinquency. Although results were consistent when parallel analyses were conducted using only the White youth, the results of this study may be more generalizable to White youth, particularly those from highly educated families who engage in low levels of offending. Therefore, it is essential that future studies utilize samples diverse in ethnic composition, socioeconomic status, and offending histories. An important note is that we relied on the traditional $p < .05$ threshold for statistical significance in each model. Because we conducted four separate tests of G×E for each genotype, a single model for a main effect of school attachment, and then repeated all analyses using only the White youth subsample, the conservative Bonferroni adjustment for the all of the models and parameters tested would have been $p = .05/(2^*3^*4) + 3) = .0018$. This would have resulted in some nonsignificant findings, indicating a clear need for replication of the patterns reported here.

Despite these limitations, this study takes an important step in G×E research by focusing on the significance of the school context for reducing adolescent delinquency. Results indicate that the interaction between school context and genetic factors is important to consider when examining mechanisms of adolescent delinquency. Examining the effects of either school attachment or genetic variation for delinquency in isolation is likely to yield inaccurate results and ignore an important G×E interaction. Our results reaffirm the finding that genetic effects on behavior are not deterministic, that is, the presence or absence of a gene variant alone would not be expected to result in lower school attachment or higher school delinquency. We found that individuals without the *DRD4-7R* allele or youth with the *DRD2*-A1 allele were more susceptible to both positive and negative school environments, whereas youth with the *DAT1*-10R allele are more positively influenced by positive school attachment. As expected given these associations, the polygenic score of these three dopaminergic genes also interacted with the amount of school attachment to affect delinquency involvement. Attachment to school serves as an important social bond that influences delinquent and antisocial behavior. Due to the salience of school during adolescence, it is important to recognize the individual-level traits that influence the degree to which school context will influence maladaptive behaviors.

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Received November 30, 2015
Revision received May 18, 2016
Accepted May 23, 2016 ■