

Delinquent Peer Group Formation: Evidence of a Gene × Environment Correlation

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ABSTRACT. Emerging evidence suggests that variants of specific genes may influence some youths to seek out or associate with antisocial peers. Using genotypic data ($N = 1,816$) from the National Longitudinal Study of Adolescent Health (J. R. Udry, 1998, 2003), the authors tested this possibility. They found that the 10R allele of the dopamine transporter (DAT1) gene was associated with self-reported delinquent peer affiliation for male adolescents from high-risk environments (β range = .13–.14) despite controlling for delinquent involvement, self-control, and drug and alcohol use. The authors discuss the importance of using a biosocial framework to examine issues related to adolescent development.

Keywords: behavioral genetics, biology, delinquent peers, genes

HOMOPHILY, noted McPherson, Smith-Lovin, and Cook (2001), represents a “basic organizing principle” of human action (p. 416). For a variety of reasons, individuals prefer to associate with others of similar talents, beliefs, characteristics, and behaviors. Empirical evidence of this fact is robust: Individuals tend to

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associate with others of the same race and ethnicity (Giordano, 2003; Hallinan & Williams, 1989; Moody, 2001; Way & Chen, 2000) and to associate with others similar in age and education (Marsden, 1988; McPherson et al.). Researchers studying spouse and friendship similarity have also detected significant levels of homophily. Galbaud du Fort, Boothroyd, Bland, Newman, and Kakuma (2002), for example, reported strong similarity between spouses in adult antisocial behavior (odds ratio of 20:1). Moreover, in analyses of monozygotic (MZ) and dizygotic (DZ) twins, Rushton and Bons (2005) found that the friends and spouses of MZ twins are significantly more alike than are the spouses and friends of DZ twins. Further analyses revealed that 34% of the variance in spouse and friendship selection was attributable to genetic influences.

Homophily has also been detected in a range of criminological studies (Cairns & Cairns, 1994; Glueck & Glueck, 1950; Warr, 2002). One of the strongest and most robust correlates to crime and delinquency is associating with delinquent friends (Akers, 1998; Haynie, 2001, 2002; Matsueda & Anderson, 1998; Warr, 1996, 2002). Measures of delinquent peers have been found to predict a wide range of antisocial, criminal, and drug-using behaviors (Warr, 1998, 2002). The link between antisocial peers and misconduct is so well established and so consistently replicated that Warr (2002) contended, "Few, if any, empirical regularities in criminology have been documented as often or over as long a period of time as the association between delinquency and delinquent friends" (p. 40).

Recognizing that peers are an important correlate of misbehavior, researchers have focused much of criminological research on whether peers cause criminal conduct or whether they are a byproduct of preexisting antisocial tendencies (Akers, 1998; Elliott & Menard, 1996; Gottfredson & Hirschi, 1990; Warr, 2002; Warr & Stafford, 1991). Grounded in the theoretical tradition of differential association and social learning theory, the social causation perspective maintains that delinquent friends transfer and imprint antisocial values and delinquent behaviors directly onto their peers (Akers). These behaviors and values are subsequently mimicked, practiced, refined, and ultimately adopted by members of the antisocial peer network (Warr & Stafford). In contrast, control theories contend that certain crime-producing traits may differentially lead some youths to select themselves into antisocial peer networks (Gottfredson & Hirschi). Advocates of self-selection argue that individuals seek out friends who share similar interests, similar personalities, and similar behavioral proclivities (Galbaud du Fort et al., 2002).

These two competing perspectives have framed the debate about the correlation between delinquent peers and behavior (Elliott & Menard, 1996). However, as Walsh (2002; see also Scarr & McCartney, 1983) noted, "We all seek environments that are compatible with our genetic dispositions" (p. 173). Walsh's observation leads to an equally plausible, yet unexplored, possibility—that selection and acceptance into delinquent peer groups is influenced by one's genotype. Using data from the National Longitudinal Study of Adolescent Health (Add Health; Udry, 1998, 2003) that includes genotypic information, we explored the

possibility that variants of the dopamine transporter gene (DAT1) are associated with assortment into delinquent peer networks.

Gene × Environment Correlations

Criminologists have largely ignored the possibility that many correlates of delinquency may have genetic underpinnings. Some researchers, however, contend that this oversight is a serious mistake (Rowe, 1994; Rowe & Rodgers, 1997; Scarr, 1992; Scarr & McCartney, 1983; Walsh, 2002). Research from divergent disciplines reveals, for example, that adolescents' social lives are not completely divorced from their genetic makeup (Cleveland, Wiebe, & Rowe, 2005; DiLalla, 2002; Iervolino et al., 2002; Moffitt, 2005; Rushton & Bons, 2005; Scarr; Scarr & McCartney). As several theorists have asserted, an individual's genotype may be partially responsible for shaping, structuring, and selecting environments that allow optimum gene expression (Scarr; Scarr & McCartney). In the language of behavioral genetics, the phenomenon by which a genotype is intimately intertwined with its environment is referred to as a *gene × environment correlation* (rGE; Rutter, 2006; Rutter et al., 1997; Rutter & Silberg, 2002; Walsh, 2002).

rGEs offer an important explanation for why people's personality or temperament often correlates with the environment in which they find themselves. Most personality factors, such as neuroticism, are partially heritable, and many disorders, such as attention deficit hyperactivity disorder (ADHD), are primarily genetic in origin (Barkley, 2005). People with certain personality traits, such as a penchant for thrill seeking, are apt to place themselves in dangerous or risky situations, such as skydiving classes. Conversely, an individual with a cautious disposition would probably pass up the opportunity to jump out of an airplane in favor of a less hazardous and more mundane activity. In this case, the genes that influence these personality characteristics are also the genes that aid in the creation of the environment and the experiences that transpire within the environment—precisely what behavioral geneticists mean by rGEs.

In general, there are three main types of rGEs—passive, evocative, and active—each of which accounts for a unique but overlapping process through which genetic factors influence or otherwise mold the environment (Caspi & Moffitt, 1995; Moffitt, 2005; Rutter, 2006; Rutter & Silberg, 2002; Scarr & McCartney, 1983). *Passive rGEs* build on the fact that parents pass along two different elements to their offspring: genes and an environment. First, children receive half of their genes from each parent. Second, children are born into environments that are partially a reflection of their parents' genetic makeup. As a result, a child's genetic propensities are often correlated with the environment into which they are born.

Evocative rGEs reflect the fact that people elicit certain responses from the environment based, in part, on their unique genotype (Caspi & Moffitt, 1995). A person with one genotype may evoke one type of response from the environment,

whereas another person, with a different genotype, may evoke a completely different response. Evocative rGEs are best summarized by stating that certain genetically influenced traits elicit particular responses from the environment, and these responses are correlated with the person's genotype.

Active rGEs arise from individuals actively seeking out and selecting environments or niches that are compatible with their personalities, attitudes, and behaviors (Caspi & Moffitt, 1995; Harris, 1998; Moffitt, 2005; Rutter, 2006; Rutter et al., 1997; Scarr & McCartney, 1983; Walsh, 2002). Active rGEs have the potential to explain why some adolescents associate with delinquent peers (Cleveland et al., 2005; Walsh). For some adolescents, especially those with a proclivity to engage in mischief, antisocial friendship groups may be particularly appealing. Other youths, however, particularly those who are not inclined to become involved in delinquency, may veer away from deviant peers and select more prosocial youths to befriend. According to the logic of active rGEs, choice, or human agency, is the key reason why individuals select themselves into certain environments—environments that are conducive to genetic expression (DiLalla, 2002; Rutter; Rutter & Silberg, 2002; Scarr & McCartney; Walsh).

Research on rGEs and Delinquent Peers

Although criminological researchers have not yet explored how rGEs may be related to the formation of antisocial peer networks, three studies have examined genetic contributors to antisocial friendship networks. In the first study, Iervolino et al. (2002) used two genetically sensitive data sets to examine the environmental and genetic influences on adolescent peer group socialization. The first sample included siblings from the Nonshared Environment in Adolescent Development (NEAD) study. The second sample consisted of 81 adoptive sibling pairs and 99 nonadoptive sibling pairs from the Colorado Adoption Project (CAP). Iervolino et al. measured a number of dimensions of peer-group preference (e.g., peer college orientation, peer popularity), but only one—peer delinquency—was applicable to the role rGEs play in delinquent peer affiliation. For both samples, peer delinquency was measured with self-reported questionnaires that indexed the friends' rebelliousness and drug-taking behaviors.

Iervolino et al. (2002) used traditional model-fitting techniques to decompose the proportion of variance in peer delinquency that was accounted for by genetic factors and by nonshared and shared environments. They calculated the models separately for both samples. For the NEAD sample, the results revealed that genetic factors accounted for virtually none of the variance in peer delinquency (3%). Shared and nonshared environmental influences, however, accounted for 20% and 77% of the variance in peer delinquency, respectively. Different results were gleaned for the CAP sample. Genetic factors accounted for 65% of the variance in peer delinquency, whereas the nonshared environment explained 35% of the variance; shared environment had no effect on peer delinquency. The

two samples produced divergent results and thus suggest the need for additional research to determine the importance of genetic factors in the formation of anti-social peer groups.

Cleveland et al. (2005) posed a similar research question when they sought to uncover the genetic and environmental sources of substance-abusing friends. Cleveland et al. used data from the Add Health study (Udry, 1998, 2003) that consisted of sibling pairs of different genetic relatedness (analytic sample $N = 1,036$ sibling pairs). Based on the results from biometric models, Cleveland et al. “found strong support for genetic influences on adolescents’ exposure to friends’ substance use, but no support for the social influence of families” (p. 164). Specifically, the shared environment had no effect on associating with delinquent peers, whereas genetic factors accounted for 64% of the variation in delinquent peer affiliation. Nonshared environments explained the remaining 36% of the variation.

In the most recent study, Kendler et al. (2007) analyzed a sample of male twins from the Virginia Twin Registry to estimate genetic influences on peer group deviance. Analysis of the data revealed that genetic factors accounted for 30–50% of the variance in delinquent friends, whereas the nonshared environment accounted for most of the remaining variance. Collectively, the results of these three studies hint at the possibility that rGEs may be able to shed some light on why certain youths choose to befriend antisocial peers.

DAT1 and Peer Group Selection

DAT1 is a particularly promising genetic polymorphism that may be associated with the formation of delinquent peer networks. One variant of the DAT1 gene—the 10-repeat allele (10R)—has been the focus of much empirical research (Comings et al., 2001; Rowe et al., 1998; Swanson et al., 2000). Findings from these studies suggest that the 10R allele is associated with the development of ADHD, pathological gambling, generalized anxiety disorder, depression, and other maladaptive outcomes (Comings et al.; Gill, Daly, Heron, Hawi, & Fitzgerald, 1997; Rowe et al.; Swanson et al.). DAT1 also has been linked to externalizing problem behaviors in young children, hinting at the possibility that DAT1 could also influence selection into antisocial environments (Young et al., 2002).

Present Study

For more than 20 years, scholars have hypothesized that genes are partially responsible for why some youths self-select into antisocial peer groups, whereas other adolescents seek out more prosocial friendship networks (Scarr, 1992; Scarr & McCartney, 1983; Walsh, 2002). Until recently, it was not possible to empirically assess the validity of this proposition. To our knowledge, the present research represents the first attempt to examine the correlation between a measured gene (i.e., DAT1) and antisocial friendship formation.

Method

Sample

The data for this study come from the Add Health study (Udry, 1998, 2003), the largest, nationally representative sample of adolescents in Grades 7–12 (Harris et al., 2003). The Add Health study used a multistage stratified sampling procedure to obtain a random sample of 80 high schools and 52 middle schools (Harris et al.). Students enrolled at these schools were interviewed, resulting in more than 90,000 completed questionnaires. More detailed information about the students and their families was collected from a subsample of adolescents who were reinterviewed in their homes (Harris et al.). This subsample was chosen by using school rosters to stratify the original school-based sample by grade level and by sex. In total, 20,745 adolescents were chosen and eventually agreed to participate in the Wave 1 in-home interview. One parent (typically the mother) also completed a survey during the Wave 1 in-home interview to provide additional information about his or her child, including neighborhood conditions, economic conditions, and family dynamics. Wave 1 data collection efforts began in 1994, when most of the Add Health participants were 11–19 years old. The second wave of data was collected in 1996. The third and final wave of data was collected between 2001 and 2002, when most of the respondents were 18–26 years old.

At Wave 3, respondents who had a sibling or cotwin participating in the Add Health study (Udry, 1998, 2003) were asked to supply a sample of their DNA for genotyping. The genetic subsample includes 2,574 monozygotic (MZ) twins, dizygotic (DZ) twins, and siblings. However, to provide conservative parameter estimates, 1 twin from each MZ twin pair was removed from the sample (Habersstick et al., 2005). With this selection criterion, and after deleting missing cases, we were left with a final analytical sample size of $N = 1,816$.

Measures

Delinquent peers. Past researchers using the Add Health (Udry, 1998, 2003) data have used a three-item scale that indexes an adolescent's delinquent peer network (Beaver & Wright, 2005; Bellair, Roscigno, & McNulty, 2003). At Wave 1, respondents reported how many of their three closest friends smoke at least one cigarette per day, drink alcohol once a month, and smoke pot at least once a month. The sum of these responses formed a measure of delinquent peers ($\alpha = .76$). We transformed the delinquent peers measure into a standardized scale because of severe skewedness. Higher scores on the delinquent peers measure indicate more involvement and contact with antisocial friends.

The delinquent peers measure includes only questions pertaining to those friends who use drugs, alcohol, and tobacco. An optimal measure would have included items that measure the spectrum of peers' involvement in delinquent

activities. However, prior researchers using this scale have established its predictive validity; the pattern of correlations observed with the delinquent peers measure is similar to those using alternative measures of antisocial peers (Beaver & Wright, 2005; Bellair et al., 2003). In a recent analysis of the Add Health (Udry, 1998, 2003) data, for example, the three-item delinquent peers measure was the strongest predictor of delinquent involvement in an ordinary least squares equation that controlled for a number of biological, psychological, and sociological variables (Beaver & Wright). Moreover, findings generated from using such a limited measure of antisocial peers provide for a more conservative examination of the effect that DAT1 has on selection into friendship networks.

DAT1. Five genetic polymorphisms were genotyped and included in the Add Health (Udry, 1998, 2003) genetic sample. We focused attention on one genetic polymorphism: the DAT1 gene. (For a more extensive review of the other polymorphisms available in the Add Health data, see Add Health Biomarker Team, n.d.; Hopfer et al., 2005.) DAT1 has a 40-base pair (bp) variable number of tandem repeats that can be repeated 3–11 times. Carriers of the 10R allele (480 bp) of DAT1 are significantly more susceptible to a range of psychological and behavioral problems (Comings et al., 2001; Gill et al., 1997).

Prior researchers have examined the effect of the 9-repeat (9R) allele and the 10R allele on a range of different behavioral and psychological outcomes. The 10R allele is considered the risk allele, because the 10R allele (in comparison with the 9R allele) confers an increased susceptibility of developing a range of maladaptive outcomes (Gill et al., 1997; Rowe et al., 1998; Rowe et al., 2001). More than 96% of the Add Health (Udry, 1998, 2003) sample had one of the following three combinations of alleles: (a) two 9R alleles, (b) two 10R alleles, or (c) one 9R allele and one 10R allele. In line with previous research using the Add Health data, participants who had an allele other than a 9R or a 10R were removed from the data set (Hopfer et al., 2005).

We recoded the three allele combinations as 0, 1, or 2 to reflect the presence of zero, one, or two 10R alleles. The distribution of DAT1 alleles was 5.3% for zero, 35.1% for one, and 59.6% for two 10R alleles, which is comparable to those found in other samples. Hardy–Weinberg equilibrium was fulfilled, $\chi^2(1, N = 1,816) = 0.0239, p > .05$.

Family risk. We created three measures of family risk that indexed various dimensions of the mother–offspring relationship. The first scale, Maternal attachment, assessed the emotional closeness of the mother and her adolescent. We used two items reported on by the adolescent during Wave 1 to create the Maternal attachment scale ($\alpha = .64$). Specifically, adolescents reported how close they felt to their mother and how much they thought their mother cared about them. We summed responses to these two items, with higher scores indicating more maternal attachment. Haynie (2001) and Schreck, Fisher, and Miller (2004) have used this measure previously.

The second scale, Maternal involvement, determined the extent to which mothers engaged in a variety of activities with their child. Researchers have used a similar scale when analyzing the Add Health (Udry, 1998, 2003) data (e.g., Crosnoe & Elder, 2004). During Wave 1 interviews, adolescents considered 10 activities, such as shopping; playing a sport; going to a movie, play, or sporting event; talking about a personal problem; and working on a project for school. They indicated which activities they had completed with their mother in the past 4 weeks. Those activities that the adolescent responded to affirmatively were assigned a value of 1; otherwise they were coded as 0 ($\alpha = .55$). Adolescents at Wave 1 also reported on five items concerning maternal disengagement. This scale ($\alpha = .84$) tapped whether adolescents perceived their mother as cold and withdrawn. Adolescents, for example, were asked whether they are satisfied with the way their mother communicates with them.

We then recoded all of the scales such that higher scores represented less maternal attachment, less maternal involvement, and more maternal disengagement (i.e., higher scores indicated more family risk). Because extant research has revealed that global measures of family risk are consistent predictors of criminogenic outcomes (Caspi et al., 2002; Wright & Cullen, 2001), we created a composite family risk measure based on factor analysis of the items comprising the three scales. The analysis indicated that the pattern of correlations could be accounted for by a single factor. We saved regression factor scores to create a factor measuring family risk. We then dichotomized the family risk factor score by splitting it at the mean; we coded values below or equal to the mean as 0 and values above the mean as 1. We classified respondents with a score of 0 as living in low-risk families and those with a score of 1 as living in high-risk families.

Control variables. Given the strong relation between age and antisocial outcomes (Hirschi & Gottfredson, 1983), we included a variable measuring the respondent's age in years at Wave 1. For two reasons, we also included the variable of criminal father as a dichotomous item tapping whether the respondent's biological father was ever incarcerated for a crime. First, prior research has revealed that antisocial behaviors and socially taxing traits are genetically influenced (Arseneault et al., 2003; Reiss, Neiderhiser, Hetherington, & Plomin, 2000; Wright & Beaver, 2005). By including a measure of fathers' antisocial history, we partially controlled for passive rGEs. Second, criminal parents tend to engage in ineffective childrearing practices, such as using harsh disciplinary tactics and withdrawing from their child's life, which are predictive of delinquency (Farrington, 2003; Loeber & Stouthamer-Loeber, 1986; Patterson, 1982; Sampson & Laub, 1993). The measure of paternal criminal behavior thus likely captured genetic and environmental risk factors predictive of adolescent offending behavior (Moffitt, 2005).

To help rule out the possibility that any relation between DAT1 and delinquent peers is due to the effects of a confounding variable, we also included three

additional scales: a delinquency scale, a low self-control scale, and a drug and alcohol use index. To create the Wave 1 delinquency scale, we used questions that asked respondents to indicate how many times in the past year they had engaged in 15 different delinquent activities. These items tapped into a variety of antisocial behaviors, including lying, fighting, and stealing. The coding scheme for these items was as follows: 0 = *never*, 1 = *one or two times*, 2 = *three or four times*, 3 = *five or more times*. We then summed responses to these questions to form the Wave 1 delinquency scale ($\alpha = .78$).

Following prior researchers' procedures (Perrone, Sullivan, Pratt, & Margaryan, 2004), we constructed a five-item low self-control scale. During Wave 1 interviews, participants answered questions that tapped whether they had (a) problems paying attention, (b) problems keeping their mind focused, (c) trouble with their teachers, or (d) trouble finishing their homework. We summed responses to these questions to form the Wave 1 low self-control scale ($\alpha = .63$).

Last, we constructed a measure that indexed adolescents' involvement in drug and alcohol use. At Wave 1, respondents reported how frequently in the past 12 months they had consumed alcohol and on how many days in the past month they had smoked marijuana. We summed these items to form the drug and alcohol use index.

Results

We calculated a series of ordinary least squares regression equations to examine the direct effect that DAT1 had on associating with delinquent friends, with a net of appropriate control variables. Because the allelic combinations of certain genetic polymorphisms vary considerably across different racial and ethnic groups (Kang, Palmatier, & Kidd, 1999; Mountain & Risch, 2004; Sarich & Miele, 2004; Shields et al., 2005), we also calculated all of the models separately by race. Respondents self-identified their racial status, and we included only those respondents who indicated that they were non-Hispanic White or Black. We also estimated all of the models by gender to explore the possibility that DAT1 has differential effects on male and female adolescents.

As shown in Table 1, we regressed the delinquent peers measure on DAT1, age, and criminal father for the full sample and separately by gender and by race. Across all models, age was the most consistent predictor of associating with delinquent peers; all of the coefficients were statistically significant and positive. However, most important was the association between DAT1 and the delinquent peers scale. DAT1 was significantly predictive of antisocial friends only for the subsample of male adolescents; male adolescents with a greater number of risk alleles were more likely to report having heavier involvement with deviant peers, providing initial evidence of an rGE. Because DAT1 and delinquent friends were not related for the full sample, for female adolescents, for African Americans, or for Caucasians, we confined our subsequent analyses to data from the subsample of male adolescents.

TABLE 1. Direct Effects of the Dopamine Transporter (DAT1) Gene on Delinquent Peers for the Full Sample ($N = 1,816$), by Gender ($n = 849$ male adolescents; $n = 967$ female adolescents) and by Race ($n = 1,464$ White adolescents; $n = 352$ Black adolescents)

Variable	Full sample ^a			Male adolescents ^b			Female adolescents ^c			White adolescents ^d			Black adolescents ^e		
	<i>b</i>	β	<i>SE</i>	<i>b</i>	β	<i>SE</i>	<i>b</i>	β	<i>SE</i>	<i>b</i>	β	<i>SE</i>	<i>b</i>	β	<i>SE</i>
DAT1	.05	.03	.04	.17	.10*	.06	-.06	-.03	.05	.05	.03	.04	.12	.07	.09
Age	.17	.29*	.01	.18	.29*	.02	.17	.29*	.02	.18	.29*	.02	.14	.26*	.03
Criminal father	.10	.04	.06	.01	.00	.10	.18	.07*	.08	.14	.05	.07	.05	.02	.12

^a $R^2 = .09$. ^b $R^2 = .10$. ^c $R^2 = .09$. ^d $R^2 = .09$. ^e $R^2 = .07$.

*Significant at the .05 level, two-tailed.

Of particular interest was whether the effect of the DAT1 variable was conditioned by different family risk levels. To examine this possibility, we calculated a separate ordinary least squares regression equation for male adolescents who received a score of 0 on the family risk scale (i.e., low family risk) and male adolescents who received a score of 1 on the family risk scale (i.e., high family risk). Table 2 presents the results for the low- and high-risk groups for White male adolescents, Black male adolescents, and both groups combined. In all of the analyses on low-risk males, age was the only statistically significant predictor. For high-risk males, however, the DAT1 variable was significantly related to the delinquent peers scale in all three statistical models. This finding suggests that, for male adolescents, the effect of DAT1 was conditioned by the family environment. Taken together, the Add Health (Udry, 1998, 2003) data revealed a robust relation between DAT1 and affiliating with delinquent peers for both White and Black male adolescents.

The possibility still exists, however, that the relation between DAT1 and delinquent peers may be spurious. To isolate the effects of DAT1, we recalculated the models and controlled for three potentially confounding variables measured at Wave 1: delinquent involvement (Model 1), low self-control (Model 2), and drug and alcohol abuse (Model 3). Model 4 considers all three factors simultaneously. To preserve degrees of freedom, we estimated the models for the entire sample of high-risk male adolescents. As Table 3 shows, DAT1 continued to be statistically

TABLE 2. Effect of the Dopamine Transporter (DAT1) Gene on Delinquent Peers by Risk Classification for Male Adolescents ($N = 814$), White Male Adolescents ($n = 666$), and Black Male Adolescents ($n = 148$)

Variable	Low-risk family				High-risk family			
	<i>b</i>	β	<i>SE</i>	R^2	<i>b</i>	β	<i>SE</i>	R^2
Male adolescents				.11				.08
DAT1	.11	.07	.07		.26	.15*	.09	
Age	.18	.32*	.03		.15	.24*	.03	
Criminal father	.01	.00	.12		.09	.03	.15	
White male adolescents				.10				.07
DAT1	.11	.07	.08		.21	.12*	.10	
Age	.18	.31*	.03		.15	.24*	.04	
Criminal father	-.02	-.01	.15		.14	.05	.16	
Black male adolescents				.14				.15
DAT1	.10	.07	.14		.55	.29*	.25	
Age	.19	.36*	.05		.18	.26	.09	
Criminal father	.14	.06	.22		-.49	-.12	.56	

*Significant at the .05 level, two-tailed.

TABLE 3. Additional Statistical Specifications to Test for Spuriousness Between the Dopamine Transporter (DAT1) Gene and Delinquent Peers (High-Risk Male Adolescents; $N = 341$)

Variable	Model 1 ^a		Model 2 ^b		Model 3 ^c		Model 4 ^d	
	<i>b</i>	<i>SE</i>	<i>b</i>	<i>SE</i>	<i>b</i>	<i>SE</i>	<i>b</i>	<i>SE</i>
DAT1	.23	.13*	.25	.14*	.22	.13*	.21	.13*
Age	.16	.24*	.15	.24*	.12	.19*	.14	.22*
Criminal father	.01	.00	.09	.15	-.03	-.01	-.10	-.03
Delinquency	.07	.39*					.04	.24*
Low self-control			.05	.14*			.03	.09
Drug and alcohol use					.06	.42*	.05	.31*

^a $R^2 = .23$. ^b $R^2 = .10$. ^c $R^2 = .26$. ^d $R^2 = .32$.

*Significant at the .05 level, two-tailed

associated with having delinquent peers (β range = .13–.14) despite strong relations with delinquency ($\beta = .39$), low self-control ($\beta = .14$), drug and alcohol use ($\beta = .42$), and the combination of factors.

Discussion

We sought to test whether genetic factors are partially involved in the formation of antisocial peer groups during adolescence. To answer this question, we used data from the Add Health study (Udry, 1998, 2003) that contained information about adolescents' social life, peer groups, relationships with family members, and a specific measured gene. These data thus provide an exceptional opportunity to examine whether genetic polymorphisms, net of the effects of social measures, predict selection into antisocial peer groups. The results of the multivariate models reveal an rGE, where male adolescents who possessed the 10R allele of DAT1 were significantly more likely to associate with delinquent peers than were male adolescents who possessed the 9R allele. To our knowledge, this is the first study to detect an rGE between a measured genetic polymorphism and an antisocial environment. Note, however, that the rGE between DAT1 and delinquent peer affiliation was not invariant across all environments or genders. The association between DAT1 and the delinquent peers scale was generally confined to male youths who lived in high-risk families. These families were characterized as lacking maternal warmth and affection and as having disengaged mothers. For adolescent males residing in low-risk families—that is, families with high levels of maternal support and involvement—the DAT1 polymorphism did not exert a statistically significant effect on associating with antisocial friends. Findings for female adolescents were consistently nonsignificant regardless of the risk level of their family.

We are left to hypothesize why we observed the effect of DAT1 only in high-risk families of male adolescents. We offer two potential reasons. First, during adolescence, parents have the ability to monitor and to control their teenage sons, at least to some degree. Some parents may take advantage of this opportunity and place limits on their adolescent's social life by instilling curfews, restricting dating, and steering their son into the appropriate prosocial peer groups. These types of parents are in a good position to intervene in their adolescent's life if they realize that their child is not obeying the family rules or if their son is beginning to drift toward the wrong crowd. In comparison, other parents are relatively cold, withdrawn, and combative toward their son. These families lack structure; discipline and punishment is irregular, erratic, and harsh (Gottfredson & Hirschi, 1990; Loeber & Stouthamer-Loeber, 1986; Patterson, 1982). Adolescent males growing up in families with these characteristics would be classified in the high-risk family category in our analyses. In both the high- and the low-risk family environments, the 10R allele of DAT1 may increase an adolescent male's propensity to associate with delinquent peers, but parents from low-risk families may be able to mitigate these genetic tendencies.

A second, slightly different interpretation is that genetic expression of the 10R allele of DAT1 differs by family environments. In low-risk families, the 10R allele may remain inactive. In high-risk families, however, the 10R allele may be triggered by risk factors such as constant stress and the general lack of support in these families.

In either case, our results reinforce findings from other studies revealing the close interplay between genetic influences and environmental forces. For example, in a landmark study, Caspi et al. (2002) found empirical support for an rGE in the etiology of antisocial behavior. Using the Dunedin Longitudinal Study, they found that the low-functioning alleles of the monoamine oxidase A (MAOA) polymorphism were related to an increase in antisocial conduct for individuals with a history of childhood maltreatment. In the absence of such maltreatment, MAOA was not significantly related to crime or delinquency. On the basis of our findings, a similar process may be at work in the formation of delinquent and drug-using peer groups.

Our research draws attention to the linkages between genotypes and social environments. Only genetically informative data sets, such as the one used in our analysis, can shed light on how genes and the environments are related. This is not to say that our research was free of any limitations; rather, we note at least two shortcomings. First, the measure of delinquent peers available in the Add Health study (Udry, 1998, 2003) tapped only the drug-using behaviors of the respondents' friends. We could not collect detailed information about serious violent acts committed by the adolescent's peer group. We recommend that in follow-up studies, researchers replicate our analyses using a different data set, with measures of antisocial peers that index more diverse and serious forms of delinquency. The second limitation of our study was that only a subsample of Add Health participants was genotyped. Although prior research analyzing the Add Health data has shown that the distribution of genes is similar to those found in other samples (Hopfer et al., 2005), we are cognizant of the possibility that our findings may not be generalizable to all adolescents. It is notable, however, that our sample size ($N = 1,816$) was much larger than has typically been available in genetic research.

AUTHOR NOTES

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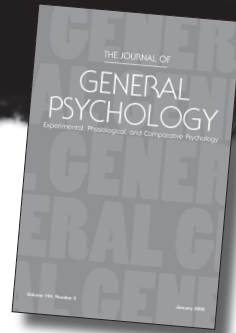
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