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# Gene–Environment Interplay and Delinquent Involvement

## Evidence of Direct, Indirect, and Interactive Effects

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Behavioral genetic research has revealed that biogenic factors play a role in the development of antisocial behaviors. Much of this research has also explicated the way in which the environment and genes may combine to create different phenotypes. The authors draw heavily from this literature and use data from the National Longitudinal Study of Adolescent Health to examine genetic and environmental effects on adolescent delinquent involvement. The results of the multivariate models reveal that genetic factors have a direct effect on youthful misconduct. Most important, however, is that genetic factors interact with delinquent peers and with low self-control to predict variation in delinquency. Analysis of the Add Health data also provide evidence suggesting that there is a shared genetic pathway to delinquent involvement, to antisocial peer group formation, and to the development of low self-control.

**Keywords:** *Add Health; delinquency; delinquent peers; gene-environment interplay; genetics; self-control; twins*

Results garnered from behavioral genetic studies have revealed substantial genetic effects on delinquency and conduct problems, with the highest estimates sometimes exceeding .85 (Arseneault et al., 2003; Miles & Carey, 1997; Rowe, 1983, 1986; Rowe & Farrington, 1997;

Rowe & Osgood, 1984; Slutske et al., 1997). Moreover, comprehensive literature reviews and meta-analyses converge to show that that delinquency is approximately 50% heritable with the remaining variance explained by nonshared environmental influences (Harris, 1998, 2006; Mason & Frick, 1994; Miles & Carey, 1997; Moffitt, 2005; Rhee & Waldman, 2002; Rowe, 1994, 2002). Behavioral genetic studies have consistently documented genetic effects on various phenotypes. With the mapping of the human genome and advances in genetic technology, however, researchers have moved toward examining *measured* genetic polymorphisms and their relationship to the development of antisocial behaviors (Caspi et al., 2002; Clark & Grunstein, 2000; Hamer & Copeland, 1998; Morley & Hall, 2003; Niehoff, 1999; Ridley, 1999; Rowe, 2002). Findings gleaned from these studies have provided quantitative evidence showing that specific genes are implicated in antisocial behaviors (Caspi et al., 2002; Foley et al., 2004).

Genes that are related to the production, transportation, and degradation of neurotransmitters (e.g., dopamine and serotonin) are potentially important causes of antisocial personality traits and behavioral disorders (Morley & Hall, 2003; Rowe, 2002). For example, certain variants of a gene responsible for the transportation of dopamine (DAT1) have been found to be associated with tobacco use, pathological gambling, and generalized anxiety disorder (Comings et al., 2001; Rowe et al., 1998; Timberlake et al., 2006), whereas certain alleles of a dopamine receptor gene (DRD4) have been found to increase the risk of ADHD (Faraone, Doyle, Mick, & Diederman, 2001; Rowe, 2002). A number of additional empirical studies have also found evidence linking genes to criminal, deviant, and aberrant conduct (Caspi et al., 2002; Foley et al., 2004; Ridley, 1999; Rowe et al., 2001; Rutter, 2006). Taken together, behavioral and molecular genetic studies find that adolescent delinquency is at least *partially* due to genetic influences.

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## Gene-Environment Interplay and Delinquency

Although genetic factors have an influence on most phenotypes, including delinquency, most phenotypes are not created by a single gene or even a small number of genes (Rutter, 2006). Instead, there is good reason to believe that phenotypic variation is due to a multifactorial arrangement of environmental influences and genetic effects acting independently and interactively. The most current genetic research, for example, uses complex research designs that are able to probe the close interplay between genes and the environment (Moffitt, 2005; Moffitt, Caspi, & Rutter, 2005; Ridley, 2003; Rutter & Silberg, 2002). Gene-environment interplay refers to the ways in which genetic influences interlock with environmental forces to bring about measurable phenotypic differences. There are two main types of gene-environment interplay: gene  $\times$  environment interactions ( $G \times E$ ) and gene  $\times$  environment correlations (rGE; Caspi & Moffitt, 1995; Rutter & Silberg, 2002; Scarr & McCartney, 1983; Walsh, 2002a).

$G \times E$ s are grounded in empirical research revealing that personality traits, temperaments, and other individual differences—many of which are partially under genetic influence—affect the way in which people respond to environmental stimuli (Caspi & Moffitt, 1995; Moffitt, 2005; Rutter, 2006). Two people embedded in the same environment, for instance, may react to it in divergent ways because of their varying genetic makeup.  $G \times E$ s can also potentially explain why shared environmental influences (e.g., the effects of the family) have relatively little effect on the development of personality and later-life outcomes. In one of the first studies to document this type of association, Caspi et al. (2002) analyzed data from the Dunedin Multidisciplinary Health and Development Study to determine whether a gene that codes for the enzyme MAOA was associated with antisocial behavior. Those with the low-functioning MAOA allele were significantly more likely to become involved in criminal behavior than people who possessed the high-functioning allele; however, this genetic effect was only found for people who were maltreated as children. Those participants who were not maltreated, but possessed the MAOA “risk” allele, were no more likely to be criminals than people who lacked the “risk” allele and were not maltreated. In this case, the MAOA gene exerted an effect on antisocial behavior only when it was paired with a “risky” environment (cf. Foley et al., 2004; Haberstick et al., 2005; Kim-Cohen et al., 2006).

Researchers have also devised additional methods for uncovering  $G \times E$ s in the creation of delinquent involvement (Beaver & Wright, 2005; Jaffee et al., 2005; Kendler et al., 1995; van Lier et al., 2007). One of the more

noteworthy strategies used to test for  $G \times E$ s was employed by Jaffee et al. (2005; see also van Lier et al., 2007). They investigated a  $G \times E$  between genetic risk and physical maltreatment in the prediction of conduct problems using data from the Environmental Risk (E-Risk) Longitudinal Twin Study. A unique aspect of their research was the way that they measured genetic risk. One twin from each twin pair was selected as the target twin and their sibling was included as the co-twin. Each co-twin's score on the dichotomous measure of conduct disorder was determined. A continuum of genetic risk for the target twin was then created by examining the co-twin's conduct disorder status in combination with their twin status (i.e., monozygotic or dizygotic). In multivariate models, Jaffee et al. (2005) found a significant main effect for genetic risk ( $\beta = .27$ ), a significant main effect for physical maltreatment ( $\beta = .15$ ), and a significant interaction effect between these two measures ( $\beta = .11$ ).

Another type of gene-environment interplay—rGEs—however, speaks to the possibility that genetic factors may be partially responsible for the way in which people select, modify, and create their own environments—in other words, a person's genotype and their environment are often correlated (Scarr & McCartney, 1983). The logic of rGEs, according to Walsh, “avers that genotypes and the environments are not random with respect to one another” (Walsh, 2002a, p. 39). Instead, research indicates that social environments are partially a construction and a reflection of each person's genotype (Beaver & Wright, 2007; DiLalla, 2002; Moffitt, 2005; Rowe & Rodgers, 1997). People actively seek out environments that are compatible with their genetic predispositions—singers join the choir, athletes compete in sporting events, and delinquents seek out other delinquents to befriend (Beaver, Wright, & DeLisi, 2008; Caspi & Moffitt, 1995; Cleveland, Wiebe, & Rowe, 2005; DiLalla, 2002; Rutter, 2006; Scarr & McCartney, 1983; Walsh, 2002a, 2002b). rGEs thus reflect the genetic underpinnings to varying social environments.

Relatively little empirical research has been devoted to examining whether rGEs are related to antisocial behaviors, but there is emerging evidence underscoring the importance of rGEs. For instance, Iervolino et al. (2002) used data from the Nonshared Environment in Adolescent Development (NEAD) study and the Colorado Adoption Project (CAP) to estimate the environmental and genetic influences on delinquent peer group formation. Analysis of the NEAD sample revealed that the shared and nonshared environments accounted for 97% of the variance in antisocial peer group formation, whereas genetic factors exerted a negligible effect (3%). A strikingly different set of results were reported when the

CAP sample were analyzed. For the CAP sample, 65% of the variance in peer delinquency was accounted for by genetic influences. The remaining 35% of the variance was attributed to the nonshared environment. Similarly, Cleveland et al. (2005) analyzed a sample of twins from the Add Health data to uncover the genetic and environmental sources to substance-abusing friends. Their analysis of the Add Health data revealed “strong support for genetic influences on adolescents’ exposure to friends’ substance use, but no support for the social influence of families when we used a behavioral-genetic design” (p. 164). Specifically, 64% of the variance in their measure of delinquent peers was accounted for by genetic factors, while the remaining 36% of the variance was attributable to the nonshared environment. Taken together, the results of these studies provide mixed evidence on the relative importance of genetic and environmental factors in the development of antisocial peer groups.

## The Current Study

That behavioral genetics research has begun to explore the effects of antisocial peers is important because delinquent peer association is among the quintessential correlates of delinquency. Decades of research have documented that a significant amount of delinquency transpires in the presence of co-offending peers or via involvement with gangs (Klein, 1995). As a result, scholars have focused on the outcomes of possessing or associating with delinquent peers or some other variant affiliated with the delinquent peer structure (Warr, 1996, 2002). The current study draws heavily from the behavioral genetic literature and examines four intertwined issues. First, we examine whether genetic factors have direct effects on delinquency. Second, because of the importance of gene-environment interplay (Rutter, 2006), we also explore the possibility that genetic effects may interact with one criminogenic environment—delinquent peers—to promote delinquency. Third, given that one of the most tested and most empirically supported criminological theories is Gottfredson and Hirschi’s (1990) general theory of crime, which asserts that an individual’s level of self-control primarily predicts involvement in delinquency and other antisocial behaviors, we also examine if genetic influences interact with levels of self-control to predict youthful offending. Fourth, a series of statistical models are estimated to discern whether variance in the measure of delinquent peers (i.e., an rGE) and variance in the measure of low self-control can be accounted for by genetic effects.

## Method

### Data

This study uses data from the National Longitudinal Study of Adolescent Health (Add Health). Add Health is a nationally representative sample of American adolescents in 7th through 12th grade (Udry, 2003). Three waves of data have been collected thus far. The first wave of data was collected in 1994, when respondents were between the ages of 11 and 19 years. More than 90,000 adolescents completed a self-report survey that was administered at school. A random subsample of youths was also selected to participate in a follow-up interview conducted in their home. More than 20,700 adolescents took part in the Wave I in-home survey and answered detailed questions about their peer groups, their involvement in risky and delinquent behaviors, their social relationships, and about other health-related topics. In addition, approximately 17,700 of their primary caregivers (usually the mother) also were interviewed and asked questions about their child (Harris et al., 2003).

The second wave of data was collected in 1995-1996 and the third round of questionnaires was administered in 2001-2002. Of all the respondents participating in the Wave I in-home survey, 14,738 were reinterviewed at Wave II. Information pertaining to the adolescent's delinquent behaviors, their personalities, and their social life was garnered from the Wave II battery of questions. At Wave III, when the respondents were 18 to 26 years old, follow-up interviews were conducted for 15,197 of the original Add Health participants.

One of the unique features of the Add Health data is that a subsample of adolescent sibling pairs is embedded within the larger sample of respondents (Harris et al., 2003). During Wave I interviews, adolescents were asked to indicate whether they had a co-twin or if they lived with a half-sibling, with an unrelated sibling (e.g., a stepsibling), or with a cousin. If they responded affirmatively, and their sibling was between the ages of 11 and 20 years old, then their sibling was added to the sample. In addition, a probability sample of full siblings was also selected to participate in the study (Jacobson & Rowe, 1999). Altogether, 3,139 adolescent pairs ( $N = 6,278$ ) were included in the sample. For reasons to be detailed momentarily, the final analytical sample was restricted to 452 DZ (dizygotic) opposite- and same-sex twin pairs and 289 MZ (monozygotic) twin pairs.<sup>1</sup>

### Measures

*Delinquency.* Similar to prior research using the Add Health data (Beaver & Wright, 2005), we developed a delinquency scale at Wave II to

**Table 1**  
**Descriptive Statistics for Add Health**  
**Sample Variables and Scales**

Variable	Mean	Standard Deviation	Percentage
Genetic risk	0.81	0.74	
Delinquency at Wave I	4.05	5.15	
Delinquency at Wave II	2.47	3.72	
Delinquent peers at Wave I	2.49	2.66	
Delinquent peers at Wave II	2.85	2.82	
Low self-control at Wave I	6.23	3.14	
Low self-control at Wave II	5.92	2.99	
Age at Wave I	16.09	1.62	
Gender			
Male			51
Female			49
Race			
White			63
Non-White			37

use as the main dependent variable in the analyses. During Wave II interviews, Add Health respondents were presented with a list of 15 different delinquent and criminal acts and were asked to indicate how frequently in the past year they had engaged in each behavior. Items comprising the delinquency scale included vandalism, physical fighting, stealing, lying, joyriding, breaking and entering, and drug use, among others. Responses to each question were coded as 0 = *never*, 1 = *1 or 2 times*, 2 = *3 or 4 times*, and 3 = *5 or more times*. The Wave II delinquency scale was created by summing together responses to each of these 15 items ( $\alpha = .78$ ). Higher scores on the delinquency scale indicate more delinquent involvement. Table 1 contains the means and standard deviations for all of the variables and scales used in the analyses.

*Genetic risk.* Prior researchers examining the genetic influences on conduct disorder (Jaffee et al., 2005), aggression (van Lier et al., 2007), and depression (Kendler et al., 1995) have developed a unique way to measure genetic risk (Moffitt, 2005). We created a genetic risk scale by following the same procedure outlined by Jaffee et al. (2005) and Kendler et al. (1995). First, a *target twin* was randomly chosen from each twin pair and their twin was identified as the *co-twin*.<sup>2</sup> Next, each of the co-twin's delinquency scale

scores was converted into a dichotomous variable. Co-twins who scored at the 90th percentile or higher on the delinquency scale were assigned a value of "1"; co-twins who scored below the 90th percentile on the delinquency scale were assigned a value of "0." A value of 1 on the dichotomous delinquency variable therefore indexed the co-twins who were the most heavily involved in delinquent activities.<sup>3</sup>

Behavior genetic studies have revealed significant genetic effects on adolescent delinquency and other problem behaviors (Rowe, 1983, 1986; Rowe & Farrington, 1997; Rowe & Osgood, 1984; Slutske et al., 1997; Walsh, 2002a). Thus, we would expect the delinquency scale available in the Add Health data to be at least partially influenced by genetic factors. We examined this possibility by calculating biometric models (i.e., ACE models) to determine whether genetic factors explained part of the variance in the delinquency scale. The results of this analysis revealed that 49% of the variance in the dichotomous delinquency scale was attributable to genetic factors, with the remaining 51% to the nonshared environment and measurement error.

Given that the delinquency scale available in the Add Health is influenced by genetic factors, then each target twin's genetic risk can be modeled as a function of their zygosity and of their co-twin's score on the dichotomous delinquency scale (Jaffee et al., 2005; Kendler et al., 1995). By using the zygosity of the twin pair along with the co-twin's delinquency status (i.e., whether they scored above or below the 90th percentile), a reliable and valid genetic risk scale can be constructed. MZ twins share 100% of their DNA. For disorders that are genetically driven, MZ twins should resemble each other more similarly because they share all the genes for that particular phenotype. DZ twins, on the other hand, share only 50% of their genes. So if a DZ twin displayed a particular phenotype (e.g., delinquency), their co-twin's genetic risk of displaying that same phenotype would be high. The same logic can be applied to MZ twins, but the genetic risk for one twin would be higher (when compared with DZ twins) because MZ twins share 100% of their DNA, not 50% such as DZ twins.

Using this logic, MZ target twins would be at greatest genetic risk if their co-twin was assigned a score of "1" on the dichotomous delinquency variable.<sup>4</sup> The group with the second highest genetic risk would be DZ target twins whose co-twin was assigned a score of "1" on the dichotomous delinquency variable. DZ target twins with a co-twin who had a score of "0" on the dichotomous delinquency variable were at the third highest genetic risk. Finally, MZ target twins whose co-twin was assigned a score of "0" on the dichotomous delinquency variable were at the lowest genetic risk. Values on the genetic risk scale ranged from 0-3, with higher scores

indicating greater genetic risk (Jaffee et al., 2005). Specifically, genetic risk was coded such that 0 = MZ target twins whose co-twin was assigned a score of 0 on the dichotomous delinquency variable; 1 = DZ target twins whose co-twin was assigned a score of 0 on the dichotomous delinquency variable; 2 = DZ target twins whose co-twin was assigned a score of 1 on the dichotomous delinquency variable; 3 = MZ target twins whose co-twin was assigned a score of 1 on the dichotomous delinquency variable.

It is important to point out that modeling genetic risk using this strategy does *not* confound environmental effects with genetic effects as long as the assumption of equal environments is fulfilled. Given that research has found support of equal environments between MZ and DZ twins (Kendler, Neale, Kessler, Heath, & Eaves, 1992), the only difference that exists between MZ twin pairs and DZ twin pairs is the amount of shared genetic material; environmental effects are constant. As a result, the only reason that MZ twin pairs should be more similar to each other than DZ twin pairs is because MZ twin pairs share 100% of their DNA, whereas DZ twin pairs share 50% of their DNA. For example, MZ twin pairs and DZ twin pairs reside in the same household, have the same parents, usually attend the same schools, and live in the same neighborhood. Thus, the only difference that would exist between MZ twins and DZ twins is the amount of shared genetic material—and it is this difference in genetic overlap that the genetic risk scale is tapping (Moffitt, 2005; van Lier et al., 2007).

*Delinquent peers.* An empirical regularity within criminology is that associating with delinquent peers is one of the strongest correlates antisocial behaviors (Akers, 1998; Warr, 1996, 2002). The Add Health data contain a number of items that index delinquent friends. In line with past research analyzing the Add Health data, we include a three-item measure of delinquent friends at Wave I and an identical three-item measure at Wave II (Beaver & Wright, 2005). At both waves, respondents were asked how many of their three best friends smoked pot at least once a month, smoked at least one cigarette each day, and drank alcohol at least once a month. Answers to these questions were coded as follows: 0 = *no friends*, 1 = *1 friend*, 2 = *2 friends*, and 3 = *3 friends*. The three items were then added together to form the Wave I delinquent peers scale ( $\alpha = .75$ ) and the Wave II delinquent peers scale ( $\alpha = .78$ ).

*Low self-control.* Measures of low self-control have been found to be among the most powerful and most consistent predictors of crime and delinquency. As a result, research that fails to include a low self-control scale may

be misspecified (Pratt & Cullen, 2000). We follow the lead of prior researchers examining Gottfredson and Hirschi's (1990) theory with the Add Health data and include a five-item low self-control scale at Wave I and an identical five-item scale at Wave II (Beaver, 2008; Perrone, Sullivan, Pratt, & Margaryan, 2004). Respondents were asked a series of questions regarding their ability to keep their mind focused, whether they had problems paying attention in school, whether they had trouble finishing their homework, and whether they had trouble getting along with other of their teachers. Responses to these items were then added together to form the low self-control scales with higher scores reflecting less self-control (Wave I  $\alpha = .63$ ; Wave II  $\alpha = .67$ ).

*Control variables.* To account for the confounding effects of certain demographic measures, we include three different control variables in the analyses. Specifically, we employ a continuous measure of the respondent's age (measured in years), as well as dichotomous dummy variables for race (0 = *White*, 1 = *non-White*) and for gender (0 = *female*, 1 = *male*).

## Plan of Analysis

Our analysis proceeded in a series of interrelated steps. First, a number of Ordinary Least Squares (OLS) regression models using the Wave II delinquency scale as the dependent variable were estimated. The first model included the genetic risk measure, the Wave II delinquent peers scale, the Wave II low self-control scale, and the control variables. The next two models included a  $G \times E$  measure by interacting the genetic risk measure with the delinquent peers measure and by interacting the genetic risk measure with the low self-control measure. To reduce problems associated with collinearity when estimating multiplicative interactions, all the scales were mean centered prior to constructing the interaction terms (Jaccard, Turrisi, & Wan, 1990).

These models provided initial information about genetic effects and  $G \times E$  effects on delinquency, but they were based on cross-sectional data. To test the robustness of the findings, we replicated the models using lagged measures of delinquent peers (measured at Wave I) and of low self-control (measured at Wave I). The  $G \times E$  terms therefore were calculated by multiplying the genetic risk score by the Wave I delinquent peers scale and by the Wave I low self-control scale.

Finally, we examined whether the genetic risk measure had indirect effects on delinquency by using the two delinquent peers scales (Waves I and II) and the two low self-control scales (Waves I and II) as dependent variables in four

**Table 2**  
**Genetic Effects and Gene × Environment Effects on**  
**Delinquency at Wave II Using Wave II Predictor Variables**

	Model 1		Model 2		Model 3	
	<i>b</i>	$\beta$	<i>b</i>	$\beta$	<i>b</i>	$\beta$
Genetic risk	.93 (.19)	.19*	.86 (.19)	.18*	.91 (.19)	.19*
Delinquent peers	.44 (.06)	.33*	.42 (.06)	.32*	.44 (.06)	.34*
Low self-control	.27 (.05)	.21*	.28 (.05)	.22*	.25 (.05)	.20*
Gender	.19 (.29)	.03	.19 (.28)	.03	.21 (.28)	.03
Race	.58 (.30)	.08*	.60 (.30)	.08*	.58 (.30)	.08
Age	-.26 (.10)	-.11*	-.25 (.09)	-.10*	-.26 (.10)	-.11*
Genetic risk × delinquent peers			.15 (.06)	.10*		
Genetic risk × low self-control					.16 (.07)	.09*
$R^2$	.25		.26		.26	

Note: Standard errors in parentheses.

\* $p < .05$ , two-tailed.

separate OLS regression equations. The models that employed the delinquent peers measures as the dependent variables tested for rGEs because they examined whether genetic factors were implicated in the structuring of an environment (i.e., delinquent peers). The models that employed the low self-control scales as the dependent variables provided information about whether genetic factors associated with delinquency were the same genetic factors that were associated with the development of low self-control.

## Results

We begin our analysis by predicting scores on the continuous Wave II delinquency scale with the genetic risk measure and with predictor variables

that were measured at Wave II. Table 2 presents the results of these cross-sectional equations. Model 1 contains the findings for the main effects models and shows that delinquent peers and low self-control exert significant positive effects on the delinquency scale. Of particular importance, however, is that the coefficient for the measure of genetic risk is positive and statistically significant. Thus, delinquent involvement appears to be at least partially influenced by genetic influences.

Thus far we have only examined whether the measure of genetic risk has a direct effect on delinquency. Prior research has demonstrated that genetic effects are often strongest when they are coupled to certain criminogenic environments—that is,  $G \times E$  (Caspi et al., 2002; Jaffee et al., 2005; Moffitt, 2005; Rutter, 2006; Walsh, 2002a). Model 2 explores this possibility by examining whether the measure of genetic risk interacts with delinquent peers to predict variation in the delinquency scale. As shown, the  $G \times E$  coefficient is positive and statistically significant, indicating that genetic risk interacts with delinquent peers to predict delinquency.

The final model in Table 2—Model 3—estimates the effect that the genetic risk  $\times$  low self-control interaction term has on the delinquency scale. Similar to Model 2, the interaction between genetic risk and low self-control is positive and statistically significant. Thus, those respondents with high levels of genetic risk *and* with low levels of self-control have an increased risk of engaging in delinquency.

The models estimated in Table 2 provide initial evidence suggesting that genetic factors and environmental forces have significant direct effects on delinquency. At the same time, the models also revealed substantial support in favor of the view that genes and the environment interact to promote delinquent involvement. However, the models that have been estimated up until this point have largely been based on cross-sectional data. To provide a more conservative test we next estimate models by using a lagged measure of delinquent peers and a lagged measure of low self-control (both measured at Wave I) to predict scores on the Wave II delinquency scale.

Table 3 contains the results of these analyses. As shown in Model 1, and in line with the models estimated in Table 2, the measure of genetic risk, the delinquent peers scale, and the measure of low self-control all have positive and statistically significant effects on the delinquency scale. Model 2 shows a similar pattern of results, wherein the genetic risk  $\times$  delinquent peers interaction coefficient maintains a positive and marginally significant ( $p = .051$ ) relationship with the Wave II delinquency scale. Finally, Model 3 reveals that the interaction between genetic risk and low

**Table 3**  
**Genetic Effects and Gene × Environment Effects on**  
**Delinquency at Wave II Using Wave I Predictor Variables**

	Model 1		Model 2		Model 3	
	<i>b</i>	$\beta$	<i>b</i>	$\beta$	<i>b</i>	$\beta$
Genetic risk	1.13 (.19)	.24*	1.05 (.20)	.22*	1.09 (.19)	.23*
Delinquent peers	.34 (.06)	.25*	.33 (.06)	.24*	.34 (.06)	.25*
Low self-control	.11 (.05)	.10*	.11 (.05)	.10*	.09 (.05)	.08
Gender	.47 (.28)	.06	.46 (.28)	.06	.48 (.28)	.07
Race	.22 (.22)	.03	.24 (.24)	.03	.25 (.25)	.03
Age	-.30 (.09)	-.13*	-.29 (.09)	-.13*	-.31 (.09)	-.14*
Genetic risk × delinquent peers			.13 (.07)	.08 <sup>†</sup>		
Genetic risk × low self-control					.12 (.05)	.09*
<i>R</i> <sup>2</sup>	.17		.18		.18	

Note: Standard errors in parentheses.

\* $p < .05$ , two-tailed. <sup>†</sup> $p = .051$ .

self-control is also statistically significant. Taken together, the findings garnered from the longitudinal models closely parallel those found in the cross-sectional models.

The results reported in Tables 2 and 3 showed that the measure of genetic risk had direct and interactive effects on delinquent involvement. There is also reason to believe that genetic factors may also have indirect effects on delinquency that operate through the environment and through different personality traits (Jaffee et al., 2005; Moffitt, 2005; Rutter, 2006). We examine this possibility in Table 4 by employing the delinquent peers scales (measured at Wave I and at Wave II) and the low self-control scales (measured at Wave I and at Wave II) as dependent variables in four separate regression equations. The first model shows that the measure of genetic risk has a positive and statistically significant effect on the Wave I delinquent peers scale. In other words, respondents with a genetic risk for delinquency also possess a liability to associate with delinquent friends. Similar results were found

**Table 4**  
**Testing for Gene-Environment Interplay**

	Dependent Variables							
	Wave I Delinquent Peers		Wave II Delinquent Peers		Wave I Low Self- Control		Wave II Low Self- Control	
	<i>b</i>	$\beta$	<i>b</i>	$\beta$	<i>b</i>	$\beta$	<i>b</i>	$\beta$
Genetic risk	.52 (.14)	.15*	.56 (.15)	.15*	.76 (.17)	.18*	.43 (.18)	.11*
Gender	.01 (.21)	.00	.56 (.22)	.10*	.22 (.26)	.03	.40 (.26)	.07
Race	-.71 (.22)	-.13*	-.68 (.23)	-.12*	-.52 (.28)	-.08	-.46 (.27)	-.08
Age	.45 (.07)	.27*	.44 (.07)	.25*	.04 (.08)	.02	-.01 (.09)	-.00
$R^2$	.10		.10		.04		.02	

\* $p < .05$ , two-tailed.

in the next model when the Wave II delinquent peers scale was entered into the analysis as the dependent variable. These results provide evidence of an rGE, where genetic risk factors are related to delinquent peer affiliation.

The last two equations in Table 4 reveal the results of the models using the low self-control scales as dependent variables. In both these equations, the genetic risk measure is positive and statistically significant. Thus, these findings provide empirical evidence that some of the genetic factors that are related to delinquency are some of the same genetic factors that are related to self-control. Stated differently, analysis of the Add Health data suggests that there is a shared genetic pathway to delinquency and to low self-control.

## Discussion

We used data from the National Longitudinal Study of Adolescent Health to determine whether delinquent involvement was partially created by genetic influences. To do so, we followed prior researchers (Jaffee et al., 2005; Kendler et al., 1995; van Lier et al., 2007) and constructed a genetic risk scale that was a function of each twin's zygosity (i.e., monozygotic or dizygotic) and of their co-twin's delinquent involvement. Using this

measurement strategy, we were able to assess the direct, indirect, and interactive effects that genes had on youthful misconduct. The measure of genetic risk had relatively strong and consistent direct effects on the Wave II delinquency scale, even after controlling for delinquent peers, low self-control, gender, race, and age.

In line with the behavioral genetic literature, we also examined whether the genetic risk scale would interact with measures of the social environment to predict variation in the delinquency scale. The results of these  $G \times E$  models revealed a significant interaction between genetic risk and delinquent peers. This  $G \times E$  was consistent—it was observed using a cross-sectional measure of delinquent peers (Wave II) and a measure of delinquent peers that was lagged 2 years (Wave I). In this case, genetic liability to delinquency was amplified for persons who affiliated with delinquent peers. To our knowledge, this is the first time that a  $G \times E$  has been detected for a measure of antisocial friends (Shanahan & Hofer, 2005).

Similarly, we also found evidence that the genetic risk scale interacted with the Wave I and the Wave II low self-control scales. This is a particularly interesting finding because low self-control has been found to be one of the strongest predictors of delinquency (Pratt & Cullen, 2000). But as our research shows, the effect of low self-control may be more pronounced when paired with certain genetic risk factors. As for now, these findings add to a growing body of literature showing that individual differences in antisocial behavior are the result of a synergistic association between genes and the environment (Rutter, 2006).

We also examined whether the genetic risk scale had indirect effects on delinquency. For these models, the genetic risk scale was used as a predictor variable of the delinquent peers scales (Waves I and II) and the low self-control scales (Waves I and II). The results of these models revealed that the genetic risk scale was a statistically significant predictor of the Wave I and Wave II delinquent peers scales, providing empirical documentation of an rGE in the construction of antisocial friendship networks (Cleveland et al., 2005; Scarr & McCartney, 1983; Walsh, 2002b). Our analysis also revealed that the measure of genetic risk had a significant effect on the two low self-control scales. Overall, the results of the multivariate models showed the importance of genes not only in the etiology of delinquency, but also in the creation of delinquent peer groups and in the development of low self-control.

With these findings in mind, it is important to touch on two limitations of the current study. First, our measure of genetic risk only permitted us to assess the relative effects that genes have on delinquent involvement; it did not allow us to discern which particular genes are implicated in the genesis

of delinquency. Although some researchers have uncovered genetic polymorphisms that relate to offending behaviors (Caspi et al., 2002), much more research is needed to identify the precise genes that contribute to anti-social misconduct. Even so, behavioral geneticists generally agree that most phenotypes are created by hundreds of genes working in concert together, making it difficult to determine which genes are related to offending behaviors (Rutter, 2006). Often the effects of one gene on a phenotype are very small, making it difficult to detect a statistically significant effect of one particular gene (Moffitt, 2005; Rutter, 2006). The use of a global scale of genetic risk—like the one used in this study—captures the effects of multiple genes because it aggregates the influence of many genetic polymorphisms into one summary measure.

Second, the measure of low self-control and the measure of delinquent peers are limited in scope. The delinquent peers scales, for example, only contain items that index the drug-using behaviors of the respondent's friends. Admittedly, we would have preferred to use a measure that included items pertaining to a broader range of delinquent activities. In addition, the Grasmick, Tittle, Bursik, & Arneklev, (1993) low self-control scale was not available in the Add Health data. Even so, there is empirical research showing that the Grasmick et al. scale is not the only way—or even the best way—to measure levels of self-control (DeLisi, Hochstetler, & Murphy, 2003; Marcus, 2003; Pratt & Cullen, 2000). Nonetheless, it is important to point out that fallibility in the low self-control scale and the delinquent peers scale would actually make it much more difficult to detect statistically significant interactive effects (Hanushek & Jackson, 1977).

Analysis of the Add Health data revealed strong genetic underpinnings to delinquency, to delinquent peers, and to low self-control. What does this mean for theories of crime causation? We explore two different theoretical implications. First, most criminological theories ignore the possibility that crime and delinquency are influenced by genes (Gottfredson & Hirschi, 1990). However, there is now a considerable amount of research—including the findings reported in this study—calling that assumption into question (Harris, 1998; Rowe, 1994; Wright & Beaver, 2005). This is not to say that all the extant theories of crime causation are wrong and should be abandoned; instead we suggest that social theorists follow the work of Walsh (2002a) and seek to integrate biological and genetic constructs into the dominant criminological theories. Gottfredson and Hirschi's (1990) theory on the development of low self-control is a prime example. They argue that low self-control is determined through parental management techniques. Recent

work by Wright and Beaver (2005) reveals that low self-control is probably determined more by genetic factors than by parental socialization tactics. The general theory could easily be modified regarding the causes of self-control to include biological and genetic factors.

Social learning theory could also be reformulated to include biological and genetic constructs. According to social learning theory, adolescents learn to engage in delinquent activities by mimicking the behaviors of antisocial role models. But how does observing an antisocial role model translate into learning to be a delinquent? One potential answer comes from the results of genetic literature showing that certain genes are related to cognitive functioning and learning (Clark & Grunstein, 2000; Hamer & Copeland, 1998; Ridley, 1999). Social learning theory could incorporate findings from molecular genetic research to shed some light on why some people learn to become criminal, whereas other people do not. This type of theoretical modification would begin to link criminology with other disciplines, such as biology, genetics, and neuroscience and would likely result in much more powerful explanations of criminal involvement (Wilson, 1998).

The second, and perhaps most noteworthy, implication of the current research is that the measure of genetic risk was a statistically significant predictor of delinquent involvement, delinquent peers, and low self-control. Although criminologists recognize that antisocial friends and low self-control are among the most robust correlates of crime and delinquency, there is very little agreement over how these criminogenic influences ultimately lead to delinquency (Akers, 1998; Gottfredson & Hirschi, 1990). Some theorists advocate a social causation perspective, whereas others advocate a self-selection perspective. Our study provides a third explanation. Genetically speaking, part of the reason that antisocial friends and low self-control are two of the most robust correlates of delinquency is because the genetic factors that are associated with delinquency overlap with the genetic factors that are related to delinquent peers and to low self-control. The results of our analysis support this view and point to the importance of using a biosocial approach when examining the correlates of crime and delinquency.

## Notes

1. We recalculated all the multivariate models using only same-sex DZ twin pairs. The substantive findings remained unchanged.

2. Jaffee et al. (2005) included each twin pair in the sample twice: One time "twin A" was the *target twin* and "twin B" was the *co-twin* and the other time "twin A" was the *co-twin* and

“twin B” was the *target twin*. Unlike Jaffee et al. (2005), we only included each twin pair in the sample once to allow each of the observations to be independent. As Jaffee et al. (2005) point out, including each twin pair in the sample only once would not bias the parameter estimates and would allow for the estimation of models using ordinary least squares (OLS) regression. Jaffee et al. used OLS regression with Huber/White variance estimates to correct for nonindependence in their observations. We also recalculated all the models with each twin pair entered into the data twice and estimated the models with the Huber/White variance estimator. The pattern of results garnered from these models was very similar to those revealed with OLS. As a result, we only present the findings for the OLS models where each twin pair is represented only once in the data.

3. Jaffee et al. (2005) used items extracted from a conduct disorder scale to determine which of the children would meet the *Diagnostic and Statistical Manual of Mental Disorders—4th edition (DSM-IV)* criteria for a clinical diagnosis of conduct disorder. Thus instead of dividing scores at the 90th percentile, Jaffee et al. (2005) determined which of the co-twins would meet the minimum requirements needed to be diagnosed with conduct disorder and assigned them a score of “1”; co-twins who were not classified as having conduct disorder were assigned a value of “0.” In their sample, 8.5% of co-twins were diagnosed with conduct disorder. In our final analytic sample, a little less than 10% of the sample of co-twins was assigned a value of “1.” Thus there is a very close correspondence between the percentage of co-twins classified as conduct disordered in Jaffee et al.’s (2005) study and the percentage of co-twins scoring in the 90th percentile on the delinquency scale in our study (see also van Lier et al., 2007).

4. The rationale for why this would be the case is because MZ twins share 100% of their DNA. Disorders and behaviors that are partially created by genetic factors should covary between MZ twins. Research, for example, has found concordance rates for many types of diseases and disorders, such as schizophrenia and depression, to be much higher for MZ twins than for DZ twins (Clark & Grunstein, 2000; Hamer & Copeland, 1998). One of the main reasons for the high concordance rates between MZ twins is because genetically speaking, they are the same person. The same logic holds true for the modeling of genetic risk. We would expect MZ twins to have the highest odds of becoming heavily involved in delinquency if their co-twin scored in the 90th percentile or above on the delinquency scale.

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