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Evidence of a Gene X Environment Interaction in the Creation of Victimization

Results From a Longitudinal Sample of Adolescents

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A large body of research has revealed that aggressive personality traits and violent criminal behaviors are influenced by genetic factors. Surprisingly, however, no research has been devoted to investigating the potential genetic origins to adolescent victimization. In the current study, the authors address this gap in the literature by using data from the National Longitudinal Study of Adolescent Health (Add Health) to examine whether different variants of the dopamine D2 receptor gene (*DRD2*) are related to victimization, net of the effects of environmental measures. The results of the multivariate models revealed a significant gene X environment interaction in the creation of victimization for White males. Specifically, *DRD2* interacted with delinquent peers to predict victimization. The authors discuss the implications of these findings.

Keywords: *biosocial; gene X environment interaction; DRD2; victimization*

A burgeoning line of behavioral genetic research has revealed that aggressive personality traits and violent criminal behaviors are influenced by genetic factors (Miles & Carey, 1997; Rutter, 2006; J. P. Wright & Beaver, 2005). The consistency and robustness of these genetic effects are quite impressive. Studies using diverse methodologies, with different samples of respondents, collected in different

countries, and at different time periods have converged to show that genetic influences account for between 40% and 80% of the variation in measures of antisocial outcomes (Arseneault et al., 2003; Mason & Frick, 1994; Miles & Carey, 1997; Moffitt, 2005; Raine, 1993; Reiss, Neiderhiser, Hetherington, & Plomin, 2000; Rhee & Waldman, 2002). As Rowe (2002) notes, “a broad range of evidence suggests that criminal disposition is heritable, including studies of psychiatric disorders, crime-linked personality traits, and crime itself, both as criminal acts recorded by the courts and police and as self-reported ones” (p. 39).

With the mapping of the human genome, researchers have moved away from simply calculating heritability estimates and have recently begun to examine whether *measured* genetic polymorphisms are associated with involvement in crime and delinquency (Caspi et al., 2002; Foley et al., 2004; Haberstick et al., 2005; Kim-Cohen et al., 2006). Results garnered from this body of research have shown that different variants of certain genes can alter the likelihood of engaging in aggression and violence. For example, Caspi and his colleagues (2002) found that variants of the monoamine oxidase A gene (*MAOA*) were related to antisocial behaviors for individuals with a history of childhood maltreatment. Subsequent research has identified additional genetic polymorphisms that are tied to the emergence of antisocial behaviors and traits (Clark & Grunstein, 2000; Dick et al., 2006; Guo, Roettger, & Shih, 2007; Hamer & Copeland, 1998; Rowe, 2002). Together, the available behavioral genetic and molecular genetic evidence suggests that genes are implicated—to varying degrees—in the development of offending behaviors (Arseneault et al., 2003; Caspi et al., 2002; Hamer & Copeland, 1998; Moffitt, 2005; Niehoff, 1999; Rowe, 2002; Rutter, 2006; J. P. Wright & Beaver, 2005).

Behavioral geneticists have conducted a wealth of empirical work revealing a genetic basis to a wide range of behaviors. Surprisingly, however, very little research has been devoted to investigating the potential genetic origins of personal victimization. Indeed, we have been unable to locate any studies that estimate genetic effects on victimization or any studies that examine whether certain genes relate to victimization. The purpose of the current study is to address this gap in the literature. To do so, we use data from the National Longitudinal Study of Adolescent Health (Add Health) and examine whether different variants of the dopamine D2 receptor gene (*DRD2*) are related to adolescent victimization, net of the effects of environmental measures.

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The Genetic Origins of Victimization

Each year, somewhere between 30% and 50% of all American adolescents experience at least one victimization incident, making them the most victimized age group in the nation (Christiansen & Evans, 2005; Esbensen & Huizinga, 1991; Menard, 2002; Snyder & Sickmund, 1999). Although adolescent victimization is relatively common, the consequences associated with victimization should by no means be trivialized. Adolescent victims not only sustain substantial physical injuries from their perpetrators but also frequently incur emotional trauma that is carried forward throughout their lives (Graham & Juvonen, 2001; Menard, 2002). Subsequent adult offending, problems with drug abuse, and being diagnosed with a mental health problem are all much more common for people who were victimized as an adolescent (Menard, 2002). Understanding the causes of adolescent victimization therefore is important to help reduce youthful victimization and to help prevent the negative consequences that are associated with it (Christiansen & Evans, 2005).

Dominant explanations of criminal victimization, such as the routine activities theory, have centered on how environmental, situational, and lifestyle factors may differentially lead an individual to be victimized (Cohen & Felson, 1979; Felson, 2002, 2006; Miethe & Meier, 1994; Sherman, Gartin, & Buerger, 1989). These environmentally oriented theories have provided a great deal of information about the correlates of victimization. Even so, many questions about the etiology of victimization remain unanswered; chief among them is whether genetic factors affect the odds that an adolescent will be the victim of a crime. Although we do not know of any extant research that has examined this possibility, there are at least three different reasons to suspect that victimization may be partially created by genetic influences (Haynie & Piquero, 2006).

First, victimization is much more common among samples of criminal offenders than among samples of nonoffenders (Baron & Hartnagel, 1998; Esbensen & Huizinga, 1991; Lauritsen, Sampson, & Laub, 1991; R. T. Wright & Decker, 1994, 1997). The overrepresentation of offenders as victims points to the possibility that the risk factors that promote involvement in crime and delinquency may also be the same risk factors that facilitate personal victimization (Gottfredson, 1981; Haynie & Piquero, 2006). Empirical research has tended to support this view. For example, Haynie and Piquero (2006) examined the association between pubertal development and adolescent victimization. They hypothesized that because a link between the timing of puberty and delinquent involvement has been established (Beaver & Wright, 2005), puberty may also contribute to victimization during adolescence. Analysis of the Add Health data revealed that early maturing youths were more likely to be victimized. The results of Haynie and Piquero's work highlight the importance of identifying biological risk factors to crime and delinquency and then applying them to the study of victimization.

Genetic factors may also be influential in understanding victimization. For example, Hines and Saudino (2004) were interested in examining genetic influences on intimate partner aggression. They estimated a series of biometric statistical models revealing that the “correlation between the use and receipt of psychological aggression is mediated genetically” (Hines & Saudino, 2004, p. 711). In other words, Hines and Saudino’s analyses showed that there is a common genetic pathway that predisposes people both to using violence and to being the victim of such violence—the genes that are associated with an increased risk of offending behaviors are the same genes that are related to victimization. Although beneficial, their data did not include information about which genes may be important contributors to victimization.

Quantitative genetic research, however, can be used to help pinpoint particular genetic polymorphisms that may be related to victimization. Recently, there has been a rapid increase in the number of studies that have examined whether specific genes are associated with maladaptive behaviors and antisocial traits. One gene in particular, *DRD2*, has been the focus of a considerable amount of attention. This line of research has examined whether an association exists between *DRD2* and various maladaptive behaviors and aggressive traits. Findings gleaned from these studies have indicated that *DRD2* is associated with alcoholism, antisocial personality symptoms, novelty seeking, pathological gambling, and others (Bau, Almeida, & Hutz, 2000; Comings et al., 2001; Connor, Young, Lawford, Ritchie, & Noble, 2002; Noble et al., 1998; Ponce et al., 2003). Haynie and Piquero (2006) argued that because “puberty has been found to be related to various sundry negative outcomes (i.e., offending), it is quite likely that the patterns revealed in the research in puberty and negative outcomes might easily apply to victimization as well” (p. 4). Following this logic, we suggest that because *DRD2* has effects that cut across a range of different antisocial outcomes, *DRD2* may also contribute to adolescent victimization.

The second reason to believe that victimization may be genetically influenced is that victims are often not innocent bystanders who are attacked at random. Instead, many victims elicit negative responses from their perpetrators that eventually culminate in a physical assault or some other type of victimization experience (Luckenbill, 1977). For example, in his classic work on criminal homicides, Wolfgang (1957) noted that “in many crimes . . . the victim is often a major contributor to the criminal act” (p. 1). More current research has applied Wolfgang’s conceptualization of victim-precipitated homicides to other types of predatory and nonpredatory crimes (Goldstein, 1985; R. T. Wright & Decker, 1994). The common theme running through these works, however, is that crime victims often are active or semiactive participants in the construction of their victimization experiences.

In a similar vein, behavioral geneticists also recognize that individuals may evoke certain responses from their environments that ultimately lead to victimization. The difference, however, is that behavioral geneticists believe that each person’s unique genetic arrangement is responsible for generating responses from the environment (Moffitt, 2005; Rutter, 2006). Given that many personality traits, such as attention

deficit/hyperactivity disorder, low self-control, and an explosive temper, are genetically influenced, then genes are at least partially accountable for eliciting differential responses from the environment (Barkley, 1997; Moffitt, 2005; Price, Simonoff, Waldman, Asherson, & Plomin, 2001; Rowe, 2002; Rutter, 2006; Walsh, 2002; J. P. Wright & Beaver, 2005). Genes, in short, will make it more or less likely that a person will be victimized. To illustrate, a person with a bad temper is much more likely to elicit aggressive responses from people than someone who is relatively docile and levelheaded. Each time that the person with a bad temper elicits an aggressive response from his or her environment, the odds of becoming a victim of a physical assault increase. Compounded over the course of a lifetime, people with difficult temperaments have a great likelihood of being victimized—due in large part to the reactions that their personality traits generate. In the parlance of behavioral geneticists, when genes elicit responses from the environment this relationship is referred to as an evocative gene X environment correlation (Moffitt, 2005; Rutter, 2006; Walsh, 2002).

The third reason to suspect that genes may affect victimization comes from research that has advocated trait-based explanations of victimization (Haynie & Piquero, 2006; Schreck, 1999; Schreck, Wright, & Miller, 2002; Stewart, Elifson, & Sterk, 2004; Unnever & Cornell, 2003). Schreck and his colleagues (Schreck, 1999; Schreck et al., 2002; see also Stewart et al., 2004) have spearheaded this movement toward individualized trait-based theories and have advanced a theoretical framework that links low self-control with violent victimization. Specifically, they argue that individuals lacking self-control will be more likely to associate with delinquent peers, to have weak family and school bonds, and to engage in risky lifestyles. All of these factors, in turn, will increase the likelihood of being the victim of a violent crime. In addition to these indirect effects, Schreck et al. (2002) also argue that low self-control will directly increase the odds of being victimized. Empirical assessment of their conceptual model revealed that low self-control had statistically significant direct and indirect effects on victimization (Schreck et al., 2002).

It is clear that the personality trait of low self-control has an effect on which environments each person will experience. Those with low self-control will be more likely to seek out deviant friends and to live risky lives. This viewpoint is strikingly similar to what behavioral geneticists typically call active gene X environment correlations (Cleveland, Wiebe, & Rowe, 2005; DiLalla, 2002; Moffitt, 2005; Rutter, 2006; Scarr, 1992; Scarr & McCartney, 1983; Walsh, 2002). Active gene X environment correlations refer to the likelihood that individuals seek out and choose environments that are well matched with their genetic predispositions. The point is that genes are an important reason that people select one environment over another or why some environments may be more appealing to one person than to another (Cleveland et al., 2005; Dick et al., 2006; Rutter, 2006; Scarr, 1992; Scarr & McCartney, 1983). Moreover, the choice of environments, which are largely scripted by genetic tendencies, may play a pivotal role in the creation of adolescent victimization.

Gene X Environment Interactions

Although genes can have direct effects on different outcome measures, most genetic research highlights the importance of examining how genes interact with the environment in the creation of different behaviors (Caspi et al., 2002; Foley et al., 2004; Haberstick et al., 2005; Ridley, 2003; Rutter, 2006). There is no reason to believe that victimization would be an exception to this general rule. Indeed, a long line of research has demonstrated that different environmental conditions and different situational factors may have a very powerful effect on the likelihood of victimization (Christiansen & Evans, 2005; Cohen & Felson, 1979; Felson, 2002, 2006; Haynie & Piquero, 2006; Miethe & Meier, 1994; Schreck, 1999; Schreck et al., 2002; Schreck & Fisher, 2004; Schreck, Fisher, & Miller, 2004). At the same time, there is limited research suggesting that genes may also have an effect on criminal victimization (Hines & Saudino, 2004). In all likelihood, however, environments probably moderate the effects of genes and genes probably moderate the effects of environments (Ridley, 2003; Rutter, 2006). In line with this literature, there is good reason to believe that one of the promising avenues for identifying the causes of victimization is by examining the close interplay between nature and nurture.

Method: The Sample

This article uses data from the Add Health. The Add Health is a prospective, longitudinal, and nationally representative sample of 7th- through 12th-grade students (K. M. Harris et al., 2003; Udry, 1998). To obtain a nationally representative sample of adolescents, the Add Health study used multistage stratified sampling procedures to select 80 high schools and 52 middle and junior high schools for inclusion in the study. In 1994, students at these schools were asked to complete a self-report survey asking questions about their behaviors, their social life, their relationship patterns, and their home life. More than 90,000 students completed the Wave I in-school survey. To gain more detailed information about the adolescent's life, a subsample of respondents was randomly chosen to participate in the Wave I in-home component of the Add Health study ($N = 20,745$). The in-home part of the study consisted of interviewing both the adolescent and the adolescent's primary caregiver (usually the mother) about a variety of topics including sexual conduct, personal victimization, delinquent involvement, family life, neighborhood conditions, and others. In total, 20,745 adolescents and 17,700 of their primary caregivers agreed to participate in the Wave I in-home interview (K. M. Harris et al., 2003).

Since the time of the Wave I interviews, two additional waves of data have been collected. The second wave of data was gathered approximately 1 to 2 years after the first wave of interviews was administered. A third wave of data was collected during 2001-2002 when the respondents were between the ages of 18 and 26 years. Of the original Wave I respondents, 14,738 were reinterviewed at Wave II and 15,197 were

reinterviewed at Wave III (K. M. Harris et al., 2003). Overall, the Add Health study consists of three waves of data that span nearly 7 years of adolescent and early adulthood development.

A distinguishing aspect of the Add Health data is that at Wave III a subset of the original sample submitted their DNA for genetic typing and analysis. Only those respondents with a sibling or co-twin¹ who was also a participant of the Add Health study were eligible to be included in the DNA subsample. Using this sampling process, 3,787 respondents were asked to provide swabs of their buccal cells. Overall, 2,574 respondents eventually agreed to participate and were genotyped. The inclusion of both environmental and genetic measures in a nationally representative and longitudinal data set provides an excellent opportunity to examine the range of factors that contribute to victimization.

Measures

Dependent Variable: The Victimization Scale

The Add Health data contain a number of items that tap the adolescent's personal victimization experiences. We follow the lead of prior researchers working with the Add Health data and construct a Victimization scale using four different items (Haynie & Piquero, 2006).² Specifically, during Wave II interviews, respondents were asked how many times in the past year someone pulled a gun or knife on them, someone jumped them, someone shot them, and someone stabbed them. Responses to these items were coded as 0 = *never*, 1 = *once*, and 2 = *more than once*. These items were then summed together to form the Victimization scale (standardized $\alpha = .69$), with higher scores representing more experiences with victimization in the past year. See Appendix A for a detailed description of the variables and scales used in the analysis, and consult Appendix B for a correlation matrix.

Independent Variables

Genetic Polymorphism Measure: DRD2

To explain the coding scheme for *DRD2*, it is first necessary to present a brief introduction to genes. Almost all genes are composed of two different copies: One copy is inherited maternally, and one copy is inherited paternally. Most genes come in only one form—that is, all people have the same gene. But for a small percentage of all genes, there are at least two different forms of the gene available in the population. These alternate forms of a gene are referred to as alleles. When more than one allele is in existence, the gene is called a polymorphism (Rowe, 2002). Unlike genes that are constant across all people, genetic polymorphisms have the potential to vary from person to person; thus, they also have the potential to explain variation in behaviors, personalities, and other observable human characteristics (Rowe, 2002; Rutter, 2006).

Quantitative genetic research examines whether different alleles from certain genes are related to the development of various forms of psychopathology. Alleles that contribute to the risk of antisocial outcomes are called “risk alleles.” For the *DRD2* polymorphism, the extant literature has revealed that the A1 allele of the *DRD2* gene (when compared to the A2 allele) is the risk allele because the A1 allele has been found to relate to alcoholism, pathological gambling, and novelty seeking (Comings et al., 2001; Connor et al., 2002; Noble et al., 1998). In genetic studies, researchers often code the genetic polymorphism measures as a count index of the number of risk alleles that each person possesses. Each person has the potential to inherit zero risk alleles, one risk allele (from either the mother or the father), or two risk alleles (from both the mother and the father).

In the Add Health data, two variables—one corresponding to the maternal allele and one corresponding to the paternal allele—are provided for each of the genetic polymorphisms. For the *DRD2* gene, each of the variables (i.e., the maternal allele variable and the paternal allele variable) was recoded into dichotomous variables. Specifically, each variable was assigned a score of 1 if the A1 allele was present; if the A1 allele was not present, the variable was assigned a value of 0. The two variables were then summed together to form the *DRD2* Risk Allele scale. This scale measures how many risk alleles are present for each respondent: A score of 0 indicates no risk alleles, a score of 1 indicates one risk allele, and a score of 2 indicates two risk alleles. Hardy-Weinberg equilibrium was fulfilled for *DRD2* in the Add Health sample ($\chi^2 = 1.86$, $df = 1$, $p = .173$).

Social Factors

Maternal attachment. Empirical research examining the correlates to victimization has found that adolescents who are more attached to their parents have lower levels of victimization (Schreck et al., 2002; Schreck et al., 2004). To take this finding into account, we created a Maternal Attachment scale that assesses the emotional closeness of the mother and her adolescent. In line with prior research using the Add Health data (Haynie, 2001; Haynie & Piquero, 2006; Schreck et al., 2004), two items reported on by the adolescent during Wave I were included in the Maternal Attachment scale. Specifically, the adolescents were asked how close they felt to their mothers and how much they thought their mothers care about them. Responses to these two items were then summed together, with higher scores indicating more maternal attachment ($\alpha = .62$).

Maternal involvement. A Maternal Involvement scale was developed to determine the extent to which the adolescents engaged in a variety of activities with their mothers. During Wave I interviews, the adolescents were presented with a list of different 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 were

then asked to indicate 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 0. Similar to the measures used by Crosnoe and Elder (2004) and by Haynie and Piquero (2006), the Maternal Involvement scale was created from 10 different activities reported on by the adolescent ($\alpha = .55$).

Maternal disengagement. A Maternal Disengagement scale was also available in the Add Health data. This scale indexed whether the mother was cold, withdrawn, and relatively removed from the adolescent's life. Five different questions, reported on by the adolescent at Wave I, were used to create the Maternal Disengagement scale ($\alpha = .84$). Adolescents, for example, were asked whether they were satisfied with the way their mother communicates with them, whether their mother was warm and loving most of the time, and whether they were satisfied with their relationship with their mother. Higher scores on this scale indicate more maternal disengagement.

Parental permissiveness. A lack of parental monitoring has been found to increase the risk of adolescent victimization (Esbensen, Huizinga, & Menard, 1999). As a result, we include a Parental Permissiveness scale that measures how much autonomy the parent gives the adolescent. At Wave I, adolescents were asked whether their parents allow them to make their own decisions about curfews, television viewing, the foods they eat, the clothes they wear, the peers they associate with, and their bedtime. Responses to these items were coded dichotomously (0 = *no*, 1 = *yes*). The Parental Permissiveness scale is composed of seven different items, with higher scores reflecting more parental permissiveness ($\alpha = .62$).

Neighborhood disadvantage. Given that the odds of being a victim of a crime vary significantly across different neighborhoods (Lauritsen et al., 1991; Sampson, 1985; Villarreal & Silva, 2006), we include a measure of neighborhood disadvantage. The Neighborhood Disadvantage scale was created by summing together responses from five different questions asked during Wave I interviews. For example, respondents were asked whether they felt safe in their neighborhood and whether they knew most of the people in the neighborhood. This scale is coded such that higher scores indicate more problems in the neighborhood ($\alpha = .48$).³

Delinquent peers. There is evidence suggesting that a youth's peer group is a particularly important correlate to personal victimization (Haynie & Piquero, 2006; Schreck et al., 2004; Schreck & Fisher, 2004). Adolescents who are embedded within delinquent friendship networks are significantly more likely to be victimized than are adolescents with relatively prosocial peers (Schreck et al., 2004). To explore the possibility that associating with antisocial friends may increase the odds of being a victim of a crime, we include a Delinquent Peers scale in our analyses. We follow past researchers using the Add Health data and use a delinquent peers measure composed

of three items tapping the drug-using behaviors of the adolescent's peer group (Beaver & Wright, 2005; Bellair, Roscigno, & McNulty, 2003). Specifically, at Wave I, respondents were asked how many of their three best friends engage in each of the following: smoke at least one cigarette per day, smoke pot more than once per month, and drink alcohol at least once each month. Responses to these items were summed together to form the Delinquent Peers scale ($\alpha = .76$).

Control Variables

Race

Prior research using the Add Health data has found that victimization rates differ significantly depending on the respondent's race (Haynie & Piquero, 2006; Schreck et al., 2004). As a result, race is included as a dichotomous dummy variable (0 = *White* and 1 = *Black*) in our statistical models.

Age

To help rule out the possibility that any significant findings are confounded by the respondent's age, we include a variable measuring the age of the respondent (Haynie & Piquero, 2006; Schreck et al., 2004). Age is a continuous variable measured in years.

Gender

We also include a measure of the respondent's gender to control for potential differences in victimization between males and females (Haynie & Piquero, 2006; Schreck et al., 2004). Gender is coded as a dichotomous dummy variable (0 = *female*, 1 = *male*).

Contemporaneous Delinquency

Given the close association between criminal involvement and victimization (Esbensen & Huizinga, 1991; Lauritsen et al., 1991), and following the lead of other researchers analyzing victimization in the Add Health data (Haynie & Piquero, 2006), we include a Delinquency scale as a statistical control. Items tapping the respondent's involvement in delinquency were collected during Wave II interviews. The Wave II Delinquency scale was created by adding 13 different items that indexed the adolescent's misconduct in the past year ($\alpha = .78$). Answers to each question were coded as 0 = *never*, 1 = *one or two times*, 2 = *three or four times*, and 3 = *five or more times*. Higher scores on the Wave II Delinquency scale represent a greater involvement in lawbreaking behaviors.

Prior Victimization

Finally, we also developed a Victimization scale based on responses to questions at Wave I. The Wave I Victimization scale is very similar to the Wave II Victimization scale (the dependent variable). For example, respondents were asked how many times in the past 12 months someone shot them, stabbed them, pulled a gun or knife on them, jumped them, or physically fought them. The response set was as follows: 0 = *never*, 1 = *once*, and 2 = *more than once*. Responses to these five different items were then summed together, and higher scores indicate more victimization experiences (standardized $\alpha = .68$).

Plan of Analysis

The analysis for this article proceeds in a series of sequential steps. First, we present means for the variables/scales that will be used in the multivariate analysis. Descriptive information is calculated for the full sample, for the White subsample, and for the Black subsample. We have chosen to restrict our final analytical sample to Whites and Blacks for one main reason. Population genetic research has shown that the distribution of *DRD2* alleles varies significantly across different racial and ethnic groups (Gelernter, Kranzler, Cubells, Ichinose, & Nagatsu, 1998). Research that fails to control for race and ethnicity may detect false-positive relationships between the genetic polymorphism and the dependent variable (Cardon & Palmer, 2003). Geneticists refer to this type of spurious association as a population stratification effect (Cardon & Palmer, 2003; Rutter, 2006). To help control for population stratification, we divide the sample into two relatively homogenous groups of respondents: Whites and Blacks. We calculate a series of *t* tests to determine whether there are any significant differences between Whites and Blacks on the variables, including the *DRD2* measure. If the *DRD2* variable differs significantly between Whites and Blacks, then it is necessary to perform the multivariate analyses separately for Blacks and for Whites.

Second, we calculate a series of multivariate regression models with the Wave II Victimization scale as the dependent variable. Because the Victimization scale is severely positively skewed (skewness statistic = 4.52; mean = 0.23; standard deviation = 0.74), the dependent variable does not meet the assumption of normality needed to conduct ordinary least squares (OLS) regression analysis. Using OLS regression with skewed dependent variables may produce unstable parameter estimates, downwardly biased standard errors, and erroneous hypothesis tests for the coefficients (Gardner, Mulvey, & Shaw, 1995). As a result, and in line with prior victimization research analyzing the Add Health data (Haynie & Piquero, 2006; Schreck et al., 2004), we use negative binomial regression techniques to examine the predictors of adolescent victimization.⁴

Third, prior research has revealed that contact with antisocial peers significantly increases the likelihood of victimization (Haynie & Piquero, 2006; Lauritsen et al., 1991; Schreck et al., 2002; Schreck et al., 2004). To take this finding into account, and to test for gene X environment interactions in the etiology of victimization, we divided the sample into two groups based on their contact with delinquent peers. Respondents who scored low on the measure of delinquent peers (i.e., they had a score of either 0 or 1) were placed into the “low delinquent peers” group. Respondents who scored high on the measure of delinquent peers (i.e., they had a score of 2 or greater) were placed in the “high delinquent peers” group. Analyses are calculated separately for each group. Essentially, we examine whether the effect of *DRD2* is conditioned by different social contexts—in this case, the social context is exposure to delinquent friends. If the regression coefficients for *DRD2* are significantly different between the low delinquent peers group and the high delinquent peers group, then a gene X environment interaction will have been detected. If, on the other hand, there are no significant differences in coefficients between the two groups, then the data will not lend support in favor of a gene X environment interaction in the creation of victimization. All of the models are calculated for the full sample ($N = 1,521$), the White subsample ($n = 1,226$; $n = 648$ White females; $n = 578$ White males), and the Black subsample ($n = 295$; $n = 153$ Black females; $n = 142$ Black males).

The negative binomial regression models will begin by regressing the Wave II Victimization scale on *DRD2*, on the socialization measures, and on the key control variables. Depending on the results of our models, we also conduct additional analyses to examine the robustness of our findings. Specifically, we introduce the Wave II Delinquency scale as a predictor variable. Past victimization research using the Add Health data has used a lagged measure of delinquency as a statistical control (Haynie & Piquero, 2006). However, including a contemporaneous delinquency scale provides a much more conservative estimate of the effects of the independent variables on victimization. Finally, we follow the lead of other researchers (Haynie & Piquero, 2006; Lauritsen et al., 1991) and control for prior victimization (measured at Wave I). When the measure of prior victimization is included as an independent variable in the models, no longer are we predicting victimization, but rather we are predicting relative change in victimization (Lauritsen et al., 1991). The Wave I Victimization scale strips away all of the variation in the Wave II Victimization scale that is attributable to stability in victimization. The variation that is left to explain is the result of change in victimization (from Wave I to Wave II), making it much more difficult to detect statistically significant effects.

Results

Table 1 presents the means of the variables/scales for the full sample, the White subsample, and the Black subsample. In addition, *t* statistics were calculated to determine

Table 1
Means and *t* Values for Add Health Sample Variables by Race

	Full Sample Mean	White Subsample Mean	Black Subsample Mean	<i>t</i> Value
Dependent variable				
Victimization	0.23	0.21	0.25	0.99
Genetic measure				
Dopamine D2 receptor gene (<i>DRD2</i>)	0.53	0.44	0.68	6.57*
Social factors				
Maternal attachment	9.40	9.38	9.54	2.45*
Maternal involvement	3.97	4.00	4.00	-0.04
Maternal disengagement	9.01	9.04	8.59	-2.51*
Parental permissiveness	5.09	5.16	4.86	-3.33*
Neighborhood disadvantage	4.65	4.57	5.20	6.50*
Delinquent peers	0.52	0.53	0.47	-2.21*
Control variables				
Age	16.06	16.02	15.87	1.62
Contemporaneous delinquency	2.55	2.54	2.46	0.44
Prior victimization	0.73	0.68	0.86	2.55*

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

whether Whites and Blacks differ significantly on any of the scales. As shown in the right-hand column of Table 1, Blacks have significantly more *DRD2* risk alleles, more victimization experiences (at Wave I), and more maternal attachment, and they live in more disadvantaged neighborhoods.⁵ However, Blacks also have significantly less maternal disengagement, less parental permissiveness, and fewer delinquent peers. Given that Blacks and Whites differ on a number of key variables, all of the statistical models will be calculated for the full sample and separately for Whites and for Blacks.

We next turn to the results of the negative binomial regression equations predicting victimization at Wave II. The left-hand column of Table 2 depicts the findings for the full sample broken down by delinquent peers status.⁶ For the low delinquent peers group, only *DRD2* and gender are significant. Of particular interest is the finding that respondents with more *DRD2* risk alleles are significantly more likely to be victimized during adolescence. However, when the same model is calculated for the high delinquent peers group, the *DRD2* variable drops from significance; gender and neighborhood disadvantage are significant predictors. The results garnered from the full sample reveal a gene X environment interaction between *DRD2* and delinquent peers in the prediction of victimization. Specifically, the effects of *DRD2* are visible only for adolescents who have relatively few delinquent peers. To make certain that the findings cannot be attributed to population stratification effects and to examine

Table 2
Negative Binomial Regression Equations Predicting Youth
Victimization by Delinquent Peer Status for the Full Sample,
the White Subsample, and the Black Subsample

	Full Sample		White Subsample		Black Subsample	
	# of Delinquent Peers		# of Delinquent Peers		# of Delinquent Peers	
	Low	High	Low	High	Low	High
Genetic measure						
Dopamine D2 receptor gene (<i>DRD2</i>)	.63*	.11	.79*	-.01	-.30	.50
	(.29)	(.16)	(.34)	(.20)	(.67)	(.28)
Social factors						
Maternal attachment	-.14	-.12	-.27	-.02	-.28	-.30
	(.22)	(.10)	(.30)	(.14)	(.37)	(.15)
Maternal involvement	-.17	.07	-.14	.07	-.11	.11
	(.10)	(.06)	(.11)	(.07)	(.21)	(.11)
Maternal disengagement	-.01	-.05	-.04	-.03	.10	-.06
	(.07)	(.04)	(.09)	(.05)	(.12)	(.07)
Parental permissiveness	-.17	.01	-.07	.04	.01	-.08
	(.13)	(.07)	(.15)	(.08)	(.25)	(.12)
Neighborhood disadvantage	.15	.14*	.18	.15	.09	.16
	(.10)	(.07)	(.13)	(.08)	(.19)	(.14)
Control variables						
Race	.47	.08				
	(.41)	(.26)				
Age	.07	-.06	-.19	-.10	.79*	.04
	(.11)	(.07)	(.14)	(.08)	(.25)	(.14)
Gender	1.90*	1.64*	2.31*	1.62*	1.67*	1.84*
	(.40)	(.24)	(.50)	(.27)	(.83)	(.54)
Pseudo R^2	.08	.05	.11	.05	.14	.09

Note: Unstandardized coefficients are presented; standard errors appear in parentheses.

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

whether the effects hold for Whites and Blacks, we also calculated the models separately by race.

As can be seen in the middle two columns of Table 2, the results generated from the White subsample are strikingly similar to those reported for the full sample. For the low delinquent peers group, *DRD2* and gender are significantly associated with victimization; however, for the high delinquent peers group, gender is the only variable related to victimization. The pattern of results for the Black subsample reveals a very different picture: The *DRD2* variable is not statistically significant for either the low delinquent peers group or the high delinquent peers group.

Table 3
Negative Binomial Regression Equations Predicting Youth Victimization
for White Males by Delinquent Peers Status

	# of Delinquent Peers		# of Delinquent Peers		# of Delinquent Peers	
	Low	High	Low	High	Low	High
Genetic measure						
Dopamine D2 receptor gene (<i>DRD2</i>)	1.14* (.40)	.09 (.23)	.99* (.39)	.26 (.21)	1.25* (.37)	.00 (.19)
Social factors						
Maternal attachment	-.52 (.49)	.10 (.18)	-.46 (.47)	.14 (.17)	-.19 (.44)	.06 (.16)
Maternal involvement	-.02 (.12)	.02 (.08)	-.02 (.12)	.08 (.07)	-.05 (.12)	.06 (.07)
Maternal disengagement	-.07 (.11)	-.06 (.06)	-.10 (.11)	-.09 (.05)	-.09 (.11)	-.05 (.05)
Parental permissiveness	.07 (.18)	.06 (.09)	.04 (.19)	.00 (.09)	-.11 (.17)	-.02 (.08)
Neighborhood disadvantage	.12 (.14)	.08 (.10)	.08 (.14)	.15 (.09)	-.01 (.14)	.05 (.08)
Control variables						
Age	-.19 (.16)	.04 (.09)	-.04 (.17)	.21* (.09)	-.12 (.16)	.15 (.08)
Contemporaneous delinquency			.20* (.09)	.18* (.03)	.16* (.08)	.11* (.02)
Prior victimization					.69* (.15)	.42* (.06)
Pseudo R ²	.04	.01	.07	.09	.16	.18

Note: Unstandardized coefficients are presented; standard errors appear in parentheses.
 **p* < .05, two-tailed.

The findings thus far suggest that there is a gene X environment interaction between *DRD2* and delinquent peers, but this effect is confined only to White respondents. To determine whether the effects are gender invariant, we recalculated all of the models separately for White females and White males. For females, the results of the negative binomial equations revealed that *DRD2* does not have a significant effect for the low delinquent peers group or for the high delinquent peers group (analyses not presented).

Table 3 depicts the findings of the negative binomial equations for White males by delinquent peers status. Similar to the results of the full sample and of the White subsample, *DRD2* is a statistically significant predictor of adolescent victimization for the low delinquent peers group but not for the high delinquent peers group—evidence of a gene X environment interaction in the creation of victimization. To examine the

robustness of this gene X environment interaction, we next introduce the Contemporaneous Delinquency scale (measured at Wave II). Even when controlling for delinquent involvement, the effect of *DRD2* remains significant (for the low delinquent peers group). Finally, we introduce the measure of prior victimization into the regression equations.⁷ The *DRD2* variable continues to be a significant predictor of victimization (for the low delinquent peers group), net of the effects of the family, of the neighborhood, of delinquent involvement, and of prior victimization. For the high delinquent peers group, the *DRD2* measure did not approach statistical significance. Analysis of the Add Health data thus provides strong empirical support for a gene X environment interaction between *DRD2* and delinquent peers in the creation of victimization for White males.

Conclusion

Studies of adolescent victimization have focused narrowly on the effects that environmental conditions have on the chances of being victimized. At the same time, the possibility that biogenetic factors may relate to personal victimization have been largely ignored. Recent research by Haynie and Piquero (2006), however, has revealed that biosocial factors may indeed play an important role in the etiology of victimization. Using their work as a springboard, we examined whether a single genetic polymorphism—*DRD2*—was predictive of youthful victimization. Analysis of the Add Health data revealed that the A1 allele of the *DRD2* gene significantly increased the chances that an adolescent would be the victim of a crime. Additional statistical models revealed that the effect of *DRD2* on victimization was confined to White males with a low number of delinquent peers. These findings remained even after controlling for the effects of prior victimization and for the effects of delinquent involvement. Given that *DRD2* was statistically significant for White males with few antisocial friends but was statistically nonsignificant for White males with a high number of delinquent peers, this study provides initial evidence of a gene X environment interaction. To our knowledge, this is the first study documenting that a gene X environment interaction is associated with victimization.

At first glance, the finding that *DRD2* had an effect only for White males in low-risk environments (i.e., few delinquent peers) may seem somewhat contradictory to what would have been expected based on the results of prior research (Caspi et al., 2002). For example, much of the extant genetic literature has found that genetic effects are visible only when paired with high-risk environments (Caspi et al., 2002; Foley et al., 2004; Rutter, 2006). Although admittedly post hoc theorizing, we offer two potential explanations for why *DRD2* interacted with a low-risk environment to predict victimization. First, highly criminogenic environments—such as being surrounded by delinquent peers—may be so powerful and so omnipotent that they are able to overshadow and blunt any genetic effects (J. R. Harris, 1998). Regardless of their genetic

makeup, adolescents who have a large number of antisocial friends are likely to be victimized simply because they are in constant contact with offenders. R. T. Wright and Decker's (1994, 1997) studies focusing on armed robbers and burglars revealed that offenders often victimize each other when presented with the opportunity. Even more revealing is that Wright and Decker found that burglars would victimize their friends and even devise schemes to steal from their peers. In this case, it is easy to see that genes may have very little effect on the odds of being victimized for respondents with a large number of delinquent friends.

Second, low-risk environments often afford an individual's genotype the maximum opportunity for genetic expression. For example, medical researchers have long recognized that susceptibilities to certain diseases and disorders are largely guided by genetic influences. Heart disease, in particular, has been found to be transmitted across generations, and much of this intergenerational transmission is accounted for by genetic factors (Marenberg, Risch, Berkman, Floderus, & de Faire, 1994). Take two individuals—one with a genetic vulnerability to heart disease and one without a genetic vulnerability to heart disease—and place them in environments that are associated with a low risk of heart disease. In these low-risk environments, the person with a genetic susceptibility is much more likely to develop heart disease than the person without the genetic vulnerability.

Now take these same two people and place them in a different environment—one in which they both smoke, eat fatty foods, do not exercise, and are overweight. For both people, the high-risk environments are so potent that the genetic predisposition to heart disease is not a distinguishing feature; both are at great risk for heart disease. A similar process may also begin to explain why *DRD2* was associated with victimization for White males with relatively few delinquent friends but not with White males with a relatively large number of antisocial peers.

Still, we are left to hypothesize why the effects of *DRD2* were observed only for White males but were statistically nonsignificant for White females, Black males, and Black females. Much of the research examining whether genetic polymorphisms relate to criminal behavior has used relatively homogenous samples of White males (e.g., Caspi et al., 2002), making it somewhat difficult to compare the results of our analyses with those reported in other studies. Even so, research has suggested that because allelic frequencies vary across different racial and ethnic groups and because certain genes may be expressed differentially between males and females (Alcock, 2001; Gelernter et al., 1998; Kang, Palmatier, & Kidd, 1999; Mountain & Risch, 2004; Sarich & Miele, 2004; Shields et al., 2005), the effects of the genes may also vary by race and gender subgroups. Results garnered from the Add Health data tend to support this view, but future research is needed to explore this possibility more fully.

Behavioral geneticists have recognized that most behaviors—including antisocial conduct—are due to the interactions between genes and the environment. Our research extends this body of literature by showing that gene X environment interactions may also be useful when studying the causes and correlates of youthful victimization. Of

course, we would be remiss if we did not point out the main limitations of our research. First, in line with previous research analyzing the Add Health data, we included a four-item measure of victimization. Unfortunately, this measure did not provide any type of information about the offender (e.g., stranger, acquaintance, significant other, or friend) or any type of information about the context within which the victimization experience occurred. It would be interesting to examine whether *DRD2* has effects that are ubiquitous for all types of victimizations (e.g., intimate partner aggression and bullying) and for all different types of perpetrators (e.g., friends and strangers). Relatedly, a number of scales used in the analyses had relatively low reliabilities. Although there is no reason to believe that fallibility in the scales would affect the substantive results, future research should address this concern.

The second main limitation of our research is that only a subsample of Add Health respondents was genotyped for the *DRD2* polymorphism. It is important to point out that prior research analyzing the Add Health data has shown that the distribution of alleles within this subsample is very similar to those reported in the population (Hopfer et al., 2005). Additionally, many genetically informative data sets are composed of convenience samples that include a relatively small number of participants, often less than 200. The current study used a sample size that contained 1,521 adolescents—a sample size that is virtually unheard of within genetic studies.

Finally, we note that only one gene, *DRD2*, was examined to determine whether it was related to adolescent victimization. There is reason to believe that other genes that are part of the dopaminergic system and genes that are part of the serotonergic system may also have effects on the odds of being victimized. In light of these limitations, and given that this is the first study to report a gene X environment interaction in the creation of victimization, follow-up studies are needed to corroborate the results before any policy implications should be recommended. As for now, our findings add to an emerging line of research using the Add Health data to examine the correlates of adolescent victimization (Haynie & Piquero, 2006; Schreck et al., 2004; Schreck & Fisher, 2004).

We close by underscoring the importance of examining crime, victimization, and other deviant behaviors from a biosocial perspective (Beaver & Wright, 2005; Raine, 2002; Walsh, 2002). To pretend that antisocial outcomes, including adolescent victimization, are created solely from environmental influences is to ignore a wealth of behavioral genetic and molecular genetic research that shows otherwise (Caspi et al., 2002; Foley et al., 2004; Niehoff, 1999; J. P. Wright & Beaver, 2005). The key to understanding and identifying the causes of offending behaviors and victimization is to examine the close interplay between nature and nurture (Moffitt, 2005; Rutter, 2006; Walsh, 2002).

Appendix A

Description of Add Health Measures and Scales

Dependent variable

Victimization Scale at Wave II: In the past 12 months, how often has someone

1. pulled a knife/gun on you?
2. shot you?
3. stabbed you?
4. jumped you?

Independent variables

Genetic measure

Dopamine D2 receptor gene (*DRD2*):

1. the number of A1 alleles the participant possesses

Socialization measures

Delinquent Peers at Wave I: Of your three best friends, how many

1. smoke at least one cigarette a day?
2. smoke pot more than once a month?
3. drink alcohol at least once a month?

Maternal Attachment at Wave I:

1. How close do you feel to your mother?
2. How much do you think your mother cares about you?

Maternal Involvement at Wave I: In the past 4 weeks, have you and your mother

1. gone shopping?
2. played a sport?
3. gone to a religious service or church-related event?
4. talked about someone you are dating or a party you went to?
5. gone to a movie, play, museum, concert, or sports event?
6. had a talk about a personal problem you were having?
7. talked about your schoolwork or grades?
8. worked on a project for school?
9. had a serious argument about your behavior?
10. talked about other things you are doing in school?

Maternal Disengagement at Wave I:

1. Most of the time, your mother is warm and loving toward you.
 2. Your mother encourages you to be independent.
 3. When you do something wrong that is important, your mother talks about it with you and helps you understand why it is wrong.
 4. You are satisfied with the way you and your mother communicate with each other.
 5. Overall, you are satisfied with your relationship with your mother.
-

Appendix A (continued)

Parental Permissiveness at Wave I: Do your parents let you make your own decisions about

1. the time you must come home on weekend nights?
2. the people you hang around with?
3. what you wear?
4. how much television you watch?
5. which television programs you watch?
6. what time you go to bed on weeknights?
7. what you eat?

Neighborhood Disadvantage at Wave I: Indicate whether each of the following statements is true:

1. You know most of the people in your neighborhood.
2. In the past month, you have stopped on the street to talk with someone from your neighborhood.
3. People in this neighborhood look out for each other.
4. You use a physical fitness or recreation center in your neighborhood.
5. You usually feel safe in your neighborhood.

Control variables

Delinquency scale at Wave II: In the past 12 months, how often did you

1. paint graffiti or signs on someone else's property or in a public place?
2. deliberately damage property that didn't belong to you?
3. lie to your parents or guardians about where you had been?
4. take something from a store without paying for it?
5. run away from home?
6. drive a car without its owner's permission?
7. steal something worth more than \$50?
8. go into a house or building to steal something?
9. use or threaten to use a weapon to get something from someone?
10. sell marijuana or other drugs?
11. steal something worth less than \$50?
12. act loud, rowdy, or unruly in public?
13. take part in a fight where a group of your friends was against another group?

Victimization scale at Wave I: In the past 12 months, how often has someone

1. pulled a knife/gun on you?
 2. shot you?
 3. stabbed you?
 4. jumped you?
 5. physically fought you?
-

Appendix B
Zero-Order Correlation Matrix for Add Health Sample Variables and Scales

	X1	X2	X3	X4	X5	X6	X7	X8	X9	X10	X11	X12	X13
Victimization (Wave II)	X1	1.00											
Dopamine Receptor Gene (DRD2) Risk Allele	X2	.05*	1.00										
Maternal Attachment	X3	.02	-.03	1.00									
Maternal Involvement	X4	-.02	-.02	.21*	1.00								
Maternal Disengagement	X5	-.03	.03	-.57*	-.23*	1.00							
Parental Permissiveness	X6	.01	-.04	-.05*	-.01	.03	1.00						
Neighborhood Disadvantage	X7	.04	.04	-.03	-.05*	.03	1.00						
Delinquent Peers	X8	.13*	.02	-.13*	-.02	.15*	.05*	1.00					
Race	X9	.02	.16*	.06*	-.00	-.06*	.17*	-.05*	1.00				
Age	X10	.04	.03	-.05*	-.02	.14*	-.02	.26*	-.04	1.00			
Gender	X11	.19*	-.00	.09*	-.15*	-.05*	-.01	.03	-.02	.02	1.00		
Delinquency (Wave II)	X12	.37*	-.02	-.09*	-.02	.12*	.02	.19*	-.01	-.10*	.08*	1.00	
Victimization (Wave I)	X13	.47*	.04	-.06*	-.05*	.04	-.01	.09*	.21*	.06*	.24*	.30*	1.00

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

Notes

1. Monozygotic twins (MZ) share 100% of their DNA. Analyzing both twins of an MZ twin dyad may result in biased parameter estimates because each twin is essentially being counted twice. As a result, we followed prior research analyzing the Add Health sample (Haberstick et al., 2005) and randomly removed one MZ twin from each MZ twin pair.

2. It is important to point out that Haynie and Piquero (2006) also include an additional item tapping whether the adolescent had taken part in a physical fight during the prior year. We opted to leave this item out of the Wave II Victimization scale because we also include a Delinquency scale in the analysis as a statistical control. The Delinquency scale includes questions that ask about the adolescent's involvement in physical fighting. Even so, we recalculated all of the models with a Victimization scale that includes the physical fighting item. The results of the supplementary analysis were virtually identical to those garnered with the more restricted Victimization scale. In addition, and similar to Haynie and Piquero, we also recoded all of the items into dichotomous variables indicating whether the respondent had experienced each of the four different types of victimization. Items were coded 0 = *no* and 1 = *yes*. We summed together the scores for these items and recalculated all of the models. Again, the pattern of results remained unchanged; thus, we report only the findings for the four-item Victimization scale.

3. We recognize that the reliabilities of the Maternal Attachment scale, the Maternal Involvement scale, the Parental Permissiveness scale, and the Neighborhood Disadvantage scale are relatively low, as indexed by Cronbach's α . However, we do not necessarily view the low reliabilities as problematic for four reasons. First, some of the scales, such as the Maternal Involvement scale, are composed of only a limited number of items. The α values are a function of the number of items in the scale and, as a result, scales with a small number of items will tend to have low α s (Carmines & Zeller, 1979). Second, two of the scales (Maternal Involvement and Parental Permissiveness) are count indexes created by summing together dichotomous variables, which attenuates Cronbach's α . Third, Cronbach's α is a conservative estimate of the scale's reliability (Carmines & Zeller, 1979). Fourth, there is no reason to suspect that fallibility in these scales would bias the dopamine D2 receptor gene (*DRD2*) coefficient.

4. All of the models were recalculated using Huber/White standard errors to correct for the clustering of data. The results were identical to those reported in the text.

5. In addition to the *t* test, a χ^2 test was also calculated to determine whether the number of *DRD2* risk alleles varied significantly between White and Black respondents. The results of the χ^2 test substantiated the results of the *t* test ($\chi^2 = 49.04, p < .05$).

6. Inspection of the correlation matrix in Appendix B reveals that collinearity does not appear to be a problem. However, to rule out the possibility that our analyses were hampered by multicollinearity, we calculated variance inflation factors and tolerance values. The values for both of these statistics revealed that multicollinearity was not problematic in any of our statistical models.

7. We also calculated the models by removing the Delinquency scale and replacing it with the Wave I Victimization scale. The results were the same as those reported when controlling simultaneously for prior victimization and delinquent involvement.

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