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Abstract

There is a burgeoning line of criminological research examining the genetic underpinnings to a wide array of antisocial phenotypes. From this perspective, genes are typically viewed as risk factors that increase the odds of various maladaptive behaviors. However, genes can also have protective effects that insulate against the deleterious effects of environmental pathogens. The authors use this logic as a springboard to examine whether four different genes protect against victimization in a sample of youths determined to be at risk for being victimized. Analysis of data drawn from the National Longitudinal Study of Adolescent Health (Add Health) revealed that certain genetic polymorphisms protected adolescents from victimization. The authors conclude by discussing the complex ways in which genes and the environment can promote resiliency to victimization.

Keywords

community violence, violence exposure, youth violence

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An impressive amount of research has revealed that genetic factors are implicated in the development of a wide range of antisocial behaviors. These findings have been garnered from behavioral genetic studies that employ samples of kinship pairs to estimate the relative influence of the environment and genes on virtually every human characteristic. The results of behavioral genetic studies examining antisocial outcomes have revealed that genetic factors account for approximately 50% of the variance in these measures (Ferguson, in press; Mason & Frick, 1994; Miles & Carey, 1997; Moffitt, 2005; Rhee & Waldman, 2002). More recently, there has been a wave of molecular genetic research that has begun to examine the precise genes that might be involved in the etiology of antisocial outcomes. Although these molecular studies are still in their infancy, they have produced some evidence linking genes involved in neurotransmission to the odds of displaying antisocial, criminal, and violent behaviors (Beaver, 2009; Morley & Hall, 2003). Against this backdrop, the perception among criminologists and other social scientists is that genes are risk factors that can only increase the odds of maladaptive outcomes, such as crime. However, it should be noted that the genes that are currently in existence in the human race have evolved because they have conferred some type of fitness advantage (Ferguson & Beaver, 2009). Seen from this perspective, then, it would be reasonable to turn the commonly held views about genes on their heads and examine the ways in which genetic factors may actually promote *prosocial* and *adaptive* outcomes.

One of the main problems with trying to identify prosocial or adaptive outcomes that might be genetically influenced is that what is prosocial and adaptive today might not have been prosocial and adaptive in our evolutionary past. For instance, today, many forms of violence and aggression are generally viewed as maladaptive. In the evolutionary past, it is easy to see that violent and aggressive behaviors (many of which would be criminalized today) would be beneficial in that the most violent persons, in comparison with the most passive persons, would tend to live longer, produce more children, and perhaps even have more of their children reach the reproductive years. The reason for this advantage is relatively straightforward: Those who are adept at using violence are better able to protect their kin, to protect themselves, and the use of violence may even be one characteristic of female mate choice (Miller, 2000; Potts & Hayden, 2008). In short, behaviors that are maladaptive today may have been highly advantageous in the evolutionary past.

One behavior, however, that does appear to have been adaptive both in our evolutionary past and today is *resiliency* to victimization. Being victimized is obviously associated with the risk of being seriously injured or perhaps even

killed. Moreover, being victimized is also associated with the loss of social status (Lamertz & Aquino, 2004; Schuster, 1999), which may repel potential mates, and females who are sexually victimized may also have their mate choice circumvented (Thornhill & Palmer, 2000). Taken together, these outcomes associated with victimization all reveal that victimization would be highly disadvantageous regardless of the time period being studied. Being resilient to victimization, especially in high-risk environments, would thus be highly advantageous. To make this point clear, consider that persons who are resilient to victimization, when compared to persons who are victimized, should live longer, have more mating opportunities, and produce more children—all of which are salient indicators of a fitness advantage. Although there is a paucity of research examining the protective factors to victimization, we use the preceding logic as a springboard to examine whether certain genes act as protective factors against victimization among a sample of high-risk youths.

Resiliency to Victimization

Adolescents are the most victimized age group in the United States, with somewhere between 30% and 50% of adolescents reporting being victimized each year (Christiansen & Evans, 2005; Esbensen & Huizinga, 1991; Menard, 2002; Snyder & Sickmund, 1999). These prevalence estimates of victimization for adolescents are approximately three times higher than those reported for adults (Hashima & Finkelhor, 1999). Even though victimization is widespread during adolescence, the outcomes associated with being victimized can be quite serious. Adolescent victimization can produce serious physical injuries and even death, but it can also result in long-term behavioral problems and emotional trauma as well. For example, adolescents who are victimized, compared with their nonvictimized counterparts, are more likely to become adult offenders, to develop drug abuse problems later in life, and to be diagnosed with a mental health problem in the future (Menard, 2002).

Given the adverse outcomes associated with adolescent victimization, it is not surprising that most victimization research has centered on identifying the factors that are related to being victimized (Beaver et al., 2007). Much of this research has examined the ways in which environmental, situational, and lifestyle factors affect the odds of being victimized (Anderson, 1999; Cohen & Felson, 1979; Felson, 2002; Miethe & Meier, 1994; Sherman, Gartin, & Buerger, 1989). This line of research has made significant contributions to the understanding of the potential causes and correlates of victimization, thereby providing some of the empirical scaffolding needed to develop programs that

are effective at preventing adolescent victimization. An equally important avenue of research—one that also could be used to prevent victimization—is identifying the various factors that foster resiliency to victimization among youths who are at high risk for being victimized. Stated differently, researchers need to uncover the unique characteristics that insulate certain adolescents from being victimized even when, statistically speaking, they should be victimized—That is, more research needs to examine the factors that promote resiliency to victimization. Although this is a critically important research question, only a handful of studies have examined factors that protect against adolescent victimization.

The results garnered from this line of research have pointed to a number of factors that appear to protect against adolescent victimization. For example, Lauritsen, Laub, and Sampson (1992) analyzed data from a sample of adolescents and found that youths who were bonded to their school and to their family were less likely to be the victim of an assault when compared to youths who lacked such bonds. Additional research has also uncovered other individual-level factors, such as friendliness, agreeableness, and assertiveness that have been shown to offer protection against victimization (Egan & Perry, 1998; Perry, Hodges, & Egan, 2001; Schwartz, Dodge, & Coie, 1993). It should be pointed out, however, that these studies have only examined protective factors that reduce the odds of victimization in the general population; they do not examine whether certain factors reduce the odds of victimization in populations that are at high risk for being victimized. The latter group—that is, the reduction in victimization among high-risk samples—deals with the factors that promote resiliency. Although the findings flowing from research examining protective factors in the general population are certainly applicable to the study of resiliency, there is not necessarily a one-to-one correspondence between protective factors in the general population and protective factors in high-risk samples.

To our knowledge, however, only one study has examined the effects that protective factors had on victimization among youths who were at risk for being victimized. Christiansen and Evans (2005) employed a sample of 992 eighth-grade students to determine whether a range of protective factors would buffer the effects of risk factors for victimization. The results of their analysis revealed that a composite protective factor index that included measures of social connectedness, parental monitoring, and neighborhood cohesion reduced the odds of being victimized. However, this protective factor index did not attenuate the effects that the risk factors had on victimization. Stated differently, the protective factor index worked to reduce the odds of being victimized for all students, but it did not reach statistical significance

for the high-risk students—In other words, the protective factor index was not involved in resiliency against victimization.

Taken together, the current literature has identified a handful of factors that, if present, decrease the odds of being victimized. These risk factors, however, tend to have ubiquitous effects across youths of varying risk categories. No empirical research, at least to our knowledge, has found protective factors that actually decrease the likelihood of being victimized in samples of adolescents who are a priori at high risk for being victimized—That is, factors that promote resiliency to victimization have yet to be discovered. One potential reason for why researchers have been unable to locate factors that promote resiliency from victimization is because only a narrow set of variables have been studied, meaning that a wealth of protective factors that might be tied to resiliency have yet to be studied empirically. Moreover, there is empirical and theoretical reason to suspect that genetic factors may be one group of variables that have been overlooked as potentially promoting resiliency to victimization.

Genetic Factors and Resiliency to Victimization

Recently, there has been growing interest in identifying the ways in which genetic factors may moderate the effects that certain environments have on behavioral outcomes (Rutter, 2006). The interest in this line of inquiry was spawned, in part, by the consistent finding that there is substantial heterogeneity in how people respond to the same environments. To illustrate, two people who are both exposed to the exact same environments often will react and respond to that environment in very different ways. This is particularly true for criminogenic risk factors, where the presence of a criminogenic risk factor, such as neighborhood disadvantage, increases the odds of subsequent antisocial behavior, but most people who are exposed to this criminogenic risk factor will not engage in delinquent conduct. Indeed, the overwhelming majority of people who are exposed to a criminogenic risk factor will refrain from serious criminal involvement. Purely sociological theories have a very difficult time reconciling why the same environments produce very divergent responses across people. By drawing attention to the close interplay between genetic and environmental factors, behavioral geneticists have been able to offer one potential explanation for why environments produce heterogeneous responses.

According to the logic behind behavioral genetic explanations, each person's unique genotype structures individual adaptations and responses to the same environments. When two (or more) people encounter a certain

environmental stimulus, their genotypes script, in part, how they will respond. One person—because of their genetic predispositions—may, for example, be highly susceptible to a criminogenic environment and thus engage in an array of criminal and delinquent behaviors. Another person, with a very different set of genetic predispositions, may be relatively resilient to a criminogenic environment and fail to display any antisocial behaviors. The environment, in short, may set the stage for a certain outcome to occur, but whether the outcome actually materializes may be wholly dependent on the presence of certain genetic factors. This is another way of drawing attention to the very real possibility that resiliency—that is, the absence of maladaptive outcomes in the presence of risky environments—may be wound up in the genotype.

A number of studies have emerged showing that certain genes are involved in resiliency to certain adverse outcomes. In a landmark study, Caspi et al. (2002) were interested in examining the potential reasons why childhood maltreatment leads to antisocial behaviors in some people and why others are resilient to the criminogenic effects of maltreatment. They hypothesized that a polymorphism¹ in the monoamine oxidase A (MAOA)² gene would be partially responsible for why some people who are maltreated develop into criminals while others do not. Analysis of data drawn from the Dunedin Multidisciplinary Health and Development Study provided empirical support for their hypothesis: Males with one version of the MAOA gene were relatively resistant to the criminogenic effects of childhood maltreatment, whereas males with a different version of the MAOA gene were highly susceptible to the criminogenic effects of childhood maltreatment. This study provided some of the strongest evidence indicating that genes can promote resiliency in the presence of adverse environments. Follow-up studies have substantiated Caspi et al.'s (2002) findings using different samples and slightly different measures of antisocial behaviors and maltreatment (Kim-Cohen et al., 2006). Importantly, genes have also been found to be involved in resilience against depression (Caspi et al., 2003), negative affect (Boardman, Blalock, & Button, 2008), and a host of diseases (Hill, 1999).

The results culled from this body of genetic research strongly suggest that genes may be protective factors against maladaptive outcomes (Hampton, 2006). Whether these findings would extend to adolescent victimization remains an open empirical question that has yet to be examined. Even so, there are at least three reasons to suspect that genetic factors may be involved in resiliency against victimization. First, behavioral genetic research has revealed that adolescent victimization is strongly influenced by genetic factors. For example, in a recent study, Beaver, Boutwell, Barnes, and Cooper (2009) analyzed twins drawn from the National Longitudinal Study of

Adolescent Health (Add Health) to estimate the proportion of variance in victimization that was attributable to genetic factors. The results of their analyses revealed that genetic factors explained about 40% to 45% of the variance in adolescent victimization and more than 60% of the variance in repeat victimization. However, just because genetic factors are associated with the etiology of victimization does not mean that genes would also be associated with resiliency to victimization; nonetheless, it does provide circumstantial evidence that genes could be involved in resiliency to victimization.

The second reason to believe that genetic factors might be related to resiliency against victimization comes from a molecular genetic study examining the interrelationships among the DRD2 gene, contact with delinquent peers, and adolescent victimization. In this study, Beaver et al. (2007) also analyzed data from the Add Health and found that the effect of DRD2 on victimization was evident but only for males with relatively few delinquent peers; the effect of DRD2 on adolescent victimization was not statistically significant for males with a high number of delinquent peers. This finding appears to be counterintuitive in that the genetic effect was a predisposing factor that increased the odds of being victimized only among adolescents who lacked exposure to the criminogenic environment (delinquent peers). For DRD2 to have fostered resiliency, the gene should have only had an effect among adolescents who were exposed to the criminogenic environment. It should be pointed out, however, that this study was only exploratory, and it only examined one criminogenic risk factor. Resiliency research draws attention to a host of risk factors and examines these risk factors simultaneously. As a result, it is possible that had a different modeling strategy been employed—one that examined a host of risk factors in combination—a genetic effect may have been detected in resiliency to victimization.

Third, an emerging line of research has revealed that individual-level factors, such as levels of self-control, physical size, and pubertal development, are associated with an increased likelihood of being victimized (Felson, 1996; Schreck, 1999; Schreck, Burek, Stewart, & Miller, 2007). In addition, research has also indicated that adolescents who engage in antisocial behaviors are likely to provoke violent responses from others that culminate in their own victimization (Anderson, 1999; Schreck et al., 2007). So youths who are at high risk for being victimized might exacerbate or blunt this risk of victimization based on their own unique suite of criminogenic risk factors (e.g., low self-control) and their own involvement in antisocial behaviors. This is particularly important because research has shown that individual-level factors, such as self-control, and antisocial behaviors are influenced by genetic factors

(Beaver, 2009; Ferguson & Beaver, 2009; Rhee & Waldman, 2002). As a result, it is quite possible that genetic influences on resiliency would be indirect and exert their influence by affecting criminogenic risk factors.

The Current Study

Although genetic effects have been detected on virtually every measurable human trait, and although genetic effects have been shown to moderate the effect of risk factors on behavioral outcomes, no study has ever examined whether genes may be involved in resiliency to victimization. The current study addresses this gap in the resiliency and genetic literatures by examining whether four genetic polymorphisms work to reduce the odds of being victimized among a sample of youths who were determined to be at high risk of victimization. To our knowledge, this is the first study to explore this issue.

Method

Data

Data analyzed in the current study were drawn from the Add Health. The Add Health is a three-wave, nationally representative sample of American youths enrolled in 7th through 12th grade during the 1994-1995 academic school year (Udry, 2003). The sample was generated by using a school-based sampling technique that netted a total of 80 high schools and 52 middle schools. During a specified school day, all students attending these schools were administered a self-report survey. More than 90,000 participated in the Wave 1 in-school component to the Add Health study. To gain more detailed information about some of the respondents, a subsample of youths was selected to be reinterviewed in their home, along with their primary caregiver (usually their mother). During the in-home interviews, questions were asked about the youth's social relationships, their family life, their involvement in risky behaviors, and a host of other questions germane to adolescents. A total of 20,745 adolescents and 17,700 of their primary caregivers took part in the Wave 1 in-home component of the Add Health study (Harris et al., 2003).

About 1.5 years after the Wave 1 in-home interviews were completed, the second wave of data was collected. Most of the respondents were still adolescents at Wave 2, and thus the questions asked at Wave 1 were still age appropriate. As a result, the survey instruments did not change much between Wave 1 and Wave 2. Respondents, for example, were still asked about their family

and peer relationships, their academic experiences, and their involvement in delinquent activity, among many other topics related to adolescence. A total of 14,738 respondents participated in the Wave 2 interviews. Then, between 2001 and 2002, the third wave of data was collected. Most of the respondents were young adults at Wave 3 and, as a consequence, the survey instruments were changed to include questions pertinent to early adulthood. For instance, participants were asked about their marital status, their employment history, their academic achievements, and their lifetime contact with the criminal justice system. Overall, 15,197 respondents were interviewed at Wave 3 (Harris et al., 2003).

One of the distinctive features of the Add Health data is that at Wave 3 a subsample of participants was asked to submit their buccal cells for genotyping. To be eligible for inclusion in the DNA subsample, respondents had to have a sibling or a cotwin who was also participating in the Add Health study. Respondents who agreed to participate were genotyped for a number of genes involved in neurotransmission. In total, 2,574 participants were genotyped, making the Add Health one of the largest data sets in the world to include genotypic, phenotypic, and environmental measures.

Measures

Genetic Measures

Before moving into a discussion of the genetic polymorphisms examined in this study, it is first important to provide some background information about genes. Most genes consist of two different copies: one that is inherited maternally and one that is inherited paternally. Most genes consist of the same two copies—That is, these genes do not vary from person to person. A small percentage of all genes do vary across people, and these genes are referred to as *genetic polymorphisms*. Alternative copies of a gene (i.e., variations in the genetic polymorphism) are referred to as *alleles*. Although there are a number of different ways that genetic polymorphisms can be coded for statistical analysis (e.g., dominant, recessive, etc.), we followed the lead of other researchers analyzing the Add Health data and coded all of the genes codominantly. With codominant coding, the value of the variable indicates the total number of risk alleles (i.e., alleles that increase the odds of antisocial phenotypes) the respondent possessed. Consequentially, the values for each genetic variable included 0 (indicating that the *respondent possessed no risk alleles*), 1 (indicating the *respondent possessed 1 risk allele*), and 2 (indicating the *respondent possessed 2 risk alleles*).

Dopamine D2 receptor gene. The dopamine D2 receptor (DRD2) gene has a polymorphism that is due to a TaqI restriction endonuclease. Two different alleles are available for this polymorphism: the A1 allele and the A2 allele. A line of research has indicated the A1 allele is a risk factor for developing a number of different antisocial phenotypes (Beaver, 2009). To take this into consideration, and following prior researchers analyzing the Add Health data, the DRD2 variable was coded such that the value indexed the number of A1 alleles that the respondent possessed (Beaver et al., 2007).

Dopamine D4 receptor gene. The dopamine D4 receptor (DRD4) gene has a polymorphism that results from a 48-base-pair variable number of tandem repeats that can be repeated between 2 and 11 times. This polymorphism has been studied extensively, and research has revealed that alleles that have repeat sequences greater than 7 are considered risk factors for antisocial phenotypes (Beaver, 2009). Following prior researchers analyzing the Add Health data, alleles for the DRD4 polymorphism were pooled into two groups: One group consisted of alleles that had repeat sequences less than 7, and the other group consisted of alleles that had repeat sequences greater than or equal to 7 (Hopfer et al., 2005). The value for the DRD4 variable thus indicated the total number of risk alleles (i.e., alleles with 7 or more repeats) that each respondent possessed.

Dopamine transporter gene. The dopamine transporter (DAT1) gene has a 40-base-pair variable number of tandem repeats in the 3' untranslated region of the gene (SLC6A3). The number of repeat sequences ranges between 3 and 13 copies. The 9-repeat allele and the 10-repeat alleles are the most common, and research has indicated that the 10-repeat allele is a risk factor for antisocial behaviors (Beaver, 2009). Most researchers examining the effect of DAT1 in the Add Health have excluded alleles that have repeat sequences other than 9 or 10 (Beaver, Wright, & DeLisi, 2008; Hopfer et al., 2005). We followed this procedure and coded the DAT1 variable such that the value indicated the number of 10-repeat alleles that each respondent possessed.

Serotonin transporter gene. The serotonin transporter gene has a polymorphism (5-HTTLPR) that arises from a 44-base-pair variable number of tandem repeats in the 5' regulatory region of the gene. In general, there are two main alleles at this polymorphism: a short allele (484 base pair allele) and a long allele (528 base pair allele). Although far from conclusive, research has revealed evidence indicating that the short allele is a risk factor for a range of antisocial outcomes (Beaver, 2009). As a result, the 5-HTTLPR polymorphism variable was coded codominantly, where the value on this variable indicated the total number of short alleles that each respondent possessed.

Resiliency Measures

Resiliency from victimization at Wave 1. To measure resiliency from victimization, we first created a Victimization Scale using the Wave 1 data. During Wave 1 interviews, adolescents were asked to indicate the frequency with which they were the victims of five different acts of violence. Specifically, respondents were asked how often someone had pulled a knife or a gun on them, shot them, cut or stabbed them, jumped them, and how often they had gotten into a physical fight. The response set for each of the questions was as follows: 0 = *never*, 1 = *once*, and 2 = *more than once*. Responses to each of the questions were then summed together to form a Wave 1 Violent Victimization Scale ($\alpha = .61$). Prior researchers analyzing the Add Health data have used similar scales (Beaver et al., 2007; Haynie & Piquero, 2006).

The Wave 1 Victimization Scale was then transformed into a dichotomous variable to measure resiliency from victimization at Wave 1. To do so, the values for the Victimization Scale were collapsed such that a score of 0 on the Victimization Scale was assigned a value of 1, whereas all other values were assigned a value of 0. With this coding scheme in place, a value of 0 = *at least one victimization experience at Wave 1* (i.e., the respondent was not resilient), whereas a value of 1 = *no victimization experiences at Wave 1* (i.e., the respondent was resilient).

Resiliency from victimization at Wave 2. Resiliency from victimization was measured at Wave 2 using the same process that was used to measure resiliency at Wave 1. First, a Wave 2 Victimization Scale was created. The Wave 2 Victimization Scale consisted of four items that asked the respondent to indicate the number of times in the past year someone had pulled a knife or gun on them, shot them, cut or stabbed them, and jumped them. Responses were coded as follows: 0 = *never*, 1 = *once*, and 2 = *more than once*. All four of the items were added together to create the Wave 2 Victimization Scale ($\alpha = .65$). Then, the Victimization Scale was recoded to create a variable that reflected resiliency from victimization at Wave 2. In particular, respondents who had received a value of 0 on the Wave 2 Victimization Scale were assigned a value of 1 on the resiliency variable, whereas all other values were assigned a value of 0. A value of 1 on the resiliency variable indicated someone who had *not been victimized and thus were resilient*, whereas a value of 0 on the resiliency variable indicated someone who *had been victimized at least once and thus were not resilient*.

Resiliency from victimization at Wave 3. Resiliency from victimization was also measured at Wave 3 using the same process that was used to create the two previous resiliency scales. To begin with, a Wave 3 Victimization Scale

was created. During Wave 3 interviews, respondents were asked to indicate whether someone had pulled a gun on them, pulled a knife on them, shot them, stabbed them, beaten them up and stolen something, and beaten them up but did not steal anything. Responses were coded dichotomously, such that 0 = *did not occur* and 1 = *did occur*. The six items were then summed together to create the Wave 3 Victimization Scale ($\alpha = .63$). To measure resiliency, the Wave 3 Victimization Scale was then dichotomized, such that respondents who received a value of 0 on the Victimization Scale were assigned a value of 1 on the resiliency variable (i.e., they were resilient), whereas respondents who received a value of 1 or greater on the Victimization Scale were assigned a value of 0 on the resiliency variable (i.e., they were not resilient).

Lifetime resiliency from victimization. In addition to creating a resiliency from Victimization Scale at each wave of data, we also created a lifetime resiliency from victimization measure. To create this measure, the three resiliency items from victimization variables were summed together. Scores on this measure ranged from 0 to 3, where a value of 0 reflected *no resiliency from victimization at each of the three waves* (i.e., the respondent was victimized at each wave), whereas a value of 3 indicated *resiliency from victimization at each of the three waves* (i.e., the respondent was not victimized at any wave). This variable was then dichotomized, where the value of 0 was assigned a value of 1, and all other values were assigned a value of 0. As a result, the scoring scheme for the lifetime resiliency from Victimization Scale was 1 = *respondent was resilient from victimization at all three waves* and 0 = *respondent was victimized during at least one wave*.

Total number of victimization experiences. The last way we measured resiliency from victimization was not through an either/or scale (i.e., either resilient or not resilient) as was used previously but rather with a continuous measure of victimization. This scale was created by summing together all three of the victimization scales (not the dichotomous resiliency scales but the full victimization scales). Values on this scale indicated the number of times that each respondent was victimized over the three waves of data collection.

Risk Factors for Victimization

Serious delinquency. One of the most consistent findings to emerge from the victimization research is that persons who are involved in crime and delinquency are much more likely to be victimized when compared with persons who abstain from such antisocial behaviors (Esbensen & Huizinga, 1991; Lauritsen, Sampson, & Laub, 1991). To examine the effect of delinquent

involvement on victimization, we created a Wave 1 Serious Delinquency Scale. During Wave 1 interviews, respondents were asked to indicate how often in the past 12 months they had engaged in 11 different acts of serious delinquency. For example, respondents were asked how frequently they had sold marijuana or other drugs, stolen something worth more than US\$50, and taken part in a group fight, among others. Responses to these questions were summed together to create the Wave 1 Serious Delinquency Scale, where higher values indicated greater involvement in serious delinquency ($\alpha = .77$).

Low self-control. An expanding body of empirical research has underscored the importance of self-control in the odds of being victimized (Schreck, 1999; Schreck, Wright, & Miller, 2002; Stewart, Elifson, & Sterk, 2004). As a result, we created a Wave 1 Low Self-Control Scale. Prior researchers analyzing the Add Health data have identified five items that can be employed to measure individual variation in levels of self-control (Beaver, 2008; Boutwell & Beaver, 2008; Perrone, Sullivan, Pratt, & Margaryan, 2004). For example, during Wave 1 interviews, respondents were asked to indicate how well they were able to keep their mind focused, how well they were able to concentrate, and how well they got along with their teachers. Responses to each of the items were summed together, and higher values on this scale reflected lower levels of self-control ($\alpha = .69$).

Delinquent peers. Exposure to delinquent peers is a strong risk factor for an array of antisocial outcomes (Warr, 2002), including victimization (Haynie & Piquero, 2006; Schreck & Fisher, 2004; Schreck, Fisher, & Miller, 2004). Persons who have extensive contact with delinquent peers are much more likely to be victimized when compared with persons who have limited or no contact with delinquent peers. To take this finding into account, a three-item Delinquent Peers Scale was created. During Wave 1 interviews, adolescents were asked to indicate how many of their three best friends smoked at least one cigarette each day, smoked marijuana more than once each month, and drank alcohol at least one time each month. Responses to each of the items were summed together, with higher values indicating more contact with delinquent peers ($\alpha = .76$). Importantly, this same scale has been used by previous researchers analyzing the Add Health data (Bellair, Roscigno, & McNulty, 2003).

Maternal disengagement. To examine whether an adolescent's family life can affect their odds of being victimized, we followed the lead of prior researchers and created a Maternal Disengagement Scale (Beaver et al., 2007). During Wave 1 interviews, respondents were asked five questions that related to their mother's level of disengagement. For example, respondents were asked how often they talked with their mother, the overall quality of the

relationship with their mother, and how much warmth and affection their mother showed them. Responses to these items were summed together, with higher values indicating more maternal disengagement ($\alpha = .82$).

Social support. There is reason to believe that adolescents who have relatively high levels of social support are less likely to be victimized when compared with youths who have relatively low levels of social support (Cullen, 1994). To examine this possibility, an eight-item Social Support Scale was created from questions asked to the adolescent during Wave 1 interviews. Specifically, adolescents were asked how much they felt that adults care about them, how much they felt that their teachers care about them, how much they felt that their parents care about them, and how much they felt that their friends care about them. Responses were coded as follows: 1 = *not at all*, 2 = *very little*, 3 = *somewhat*, 4 = *quite a bit*, and 5 = *very much*. The items were summed together to form the Social Support Scale ($\alpha = .77$).

Control Variables

Race. To control for the potentially confounding effect of race, and to help eliminate the possibility of population stratification, a race variable was included in all of the analyses. During Wave 1 interviews, respondents were asked to self-report their race. Only those respondents who indicated they were non-Hispanic White or Black were included in the analyses; all other minority groups were excluded from the final analytic sample. Race was coded as a dichotomous dummy variable, where 0 = *non-Hispanic White* and 1 = *Black*.

Gender. To take into account potential gender differences in victimization between males and females, a gender variable was included in all of the analyses (Haynie & Piquero, 2006). Specifically, gender was coded as a dichotomous dummy variable where 0 = *female* and 1 = *male*.

Plan of Analysis

The analysis for this study was conducted in a series of linked steps. First, as we are interested in examining the predictors of resiliency, we had to identify a sample that was at high risk for being victimized. To do so, we employed a dichotomous measure of whether the respondent had been victimized at Wave 1 (measured by using the Wave 1 Victimization Scale described above). If the respondent had been victimized at Wave 1, then they were assigned a value of 1; if they had not been victimized, then they were assigned a value of 0. Then a binary logistic regression model was estimated to predict victimization at Wave 1 with five covariates: serious delinquency, low self-control,

Table 1. Binary Logistic Regression Models Predicting Victimization at Wave 1

Covariates	<i>b</i>	SE	OR
Serious delinquency	.39*	.04	1.484
Low self-control	.08*	.02	1.081
Delinquent peers	.05*	.02	1.056
Maternal disengagement	-.06*	.02	0.945
Social support	-.03*	.01	0.969

Note: $N = 2,099$. Huber/White standard errors. OR = odds ratio.

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

delinquent peers, maternal disengagement, and social support. As can be seen in Table 1, all five of these covariates were statistically significant predictors of victimization at Wave 1. Using the results from this model, each respondent was assigned a predicted probability of being victimized. Respondents who had a predicted probability that was greater than .50 (i.e., 50%) were identified as being at high average risk for victimization; everyone else was characterized as being at low average risk for being victimized. All of the proceeding models that examined the correlates to resiliency were only conducted on the high-risk sample ($N = 287$ to 313).

Next, the three resiliency items from victimization variables were used as dependent variables in separate binary logistic regression models. Remember these variables were coded dichotomously, where 0 = *not resilient* and 1 = *resilient*. Essentially, these models examine the factors that promote resiliency from victimization during adolescence and young adulthood. Keep in mind that the high-risk sample was generated from Wave 1 data, so these models are cross-sectional and longitudinal, spanning nearly 7 years of human development. The last set of models examines the correlates to lifetime resiliency and to total number of victimization experiences. Recall that the lifetime resiliency variable is designed to measure respondents who were not victimized at any of the three waves. The total number of victimization experiences variable measures how many times the respondent was victimized across the three waves. This latter variable helps to examine whether some of the protective factors might also work in a continuous fashion, where they systematically decrease the number of victimization experiences. Negative binomial regression equations were estimated when using the total number of victimization experiences variable.

It should be noted that not all of the observations in the Add Health DNA sample were independent of each other. In some instances, more than one sibling from the same household was included in the sample. Nonindependence

Table 2. Binary Logistic Regression Models Predicting Resiliency From Victimization at Waves 1, 2, and 3 Among High-Risk Youths

	Resiliency at Wave 1			Resiliency at Wave 2			Resiliency at Wave 3		
	<i>b</i>	SE	OR	<i>b</i>	SE	OR	<i>b</i>	SE	OR
Genetic polymorphisms									
DRD2	-0.56*	.23	0.570	-0.47*	.21	0.625	-0.49*	.24	0.615
DRD4	-0.02	.23	0.979	-0.04	.23	0.959	-0.04	.27	0.975
DAT1	0.83*	.27	2.292	0.42	.25	1.523	0.24	.28	1.266
5-HTTLPR	0.39*	.19	1.475	0.13	.20	1.137	0.52*	.24	1.683
Control variables									
Race	-1.04*	.41	0.353	-0.39	.33	0.680	-0.60	.39	0.550
Gender	-0.83*	.28	0.436	-1.00*	.29	0.367	-1.14*	.38	0.318
<i>n</i>	313			289			311		

Note: Huber/White standard errors. OR = odds ratio.
 **p* < .05, two-tailed test.

results in downwardly biased standard errors, which, as a result, artificially increases the odds that a coefficient will be statistically significant. We corrected for nonindependence in two ways. First, and following prior researchers analyzing the Add Health data (Haberstick et al., 2005), one twin from each monozygotic twin pair was randomly selected and removed from the sample. Second, all of the models were estimated using Huber/White standard errors, which results in unbiased tests of statistical significance for the coefficients.

Results

The analysis began by estimating the correlates to resiliency from victimization at Waves 1, 2, and 3 among the sample of youths who were identified as being at high risk for victimization. The results of these models are presented in Table 2. As can be seen in the first model, three of the genetic measures—DRD2, DAT1, and 5-HTTLPR—were statistically significant predictors of resiliency at Wave 1. Specifically, DRD2 had a negative effect on resiliency, whereas DAT1 and 5-HTTLPR had positive effects on resiliency. In Model 2, where the Wave 2 resiliency measure was employed as the dependent variable, only one genetic measure—DRD2—had a statistically significant effect on resiliency. In line with the previous model, the effect of DRD2 was negative. Last, the Wave 3 resiliency measure was used as the dependent variable. Once again, DRD2 had a statistically significant and negative effect on

Table 3. Regression Models Predicting Lifetime Resiliency and Total Number of Victimization Experiences Among High-Risk Youths

	Lifetime Resiliency			Total Number of Victimization Experiences		
	<i>b</i>	SE	OR	<i>b</i>	SE	OR
Genetic polymorphisms						
DRD2	−0.43 ^a	.25	0.650	.26*	.09	1.299
DRD4	−0.08	.24	0.923	.06	.10	1.060
DAT1	0.83*	.31	2.297	−.25*	.11	0.775
5-HTTLPR	0.51*	.21	1.666	.03	.08	1.028
Control variables						
Race	−1.28*	.50	0.278	.19	.14	1.213
Gender	−1.00*	.30	0.367	.70*	.13	2.013
<i>n</i>		287			287	

Note: Huber/White standard errors. OR = odds ratio.

a. $p = .079$.

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

resiliency, whereas 5-HTTLPR had a positive effect on the odds of resiliency. DRD4 and DAT1 failed to have an effect on resiliency at Wave 3.

The results thus far have provided some evidence indicating that certain genetic polymorphisms are associated with resiliency from victimization. To examine these genetic effects in greater detail, two additional models were calculated with the sample of high-risk youths. First, and as Table 3 reveals, we estimated a logistic regression model using the lifetime resiliency measure as the dependent variable. As this table shows, DRD2, DAT1, and 5-HTTLPR all are associated with the odds of lifetime resiliency. DRD2 has a marginally significant and negative effect, whereas DAT1 and 5-HTTLPR have significant positive effects on lifetime resiliency. Second, the total number of victimization experiences measure was employed as the dependent variable in a negative binomial regression analysis. The results garnered from this model show that DRD2 has a positive effect on the total number of victimization experiences, whereas DAT1 has a negative effect on the total number of victimization experiences.

Discussion

Although a great deal of research has been published attempting to uncover the various risk factors that increase the odds of adolescent victimization

(Felson, 2002), relatively little empirical research has focused on the factors that promote resiliency to victimization. Moreover, the research that has been conducted has, in general, failed to identify factors that protect against victimization in samples of youth who are at risk for being victimized. The current study addressed this gap in the literature and examined whether four genetic polymorphisms—DRD2, DRD4, DAT1, and 5-HTTLPR—were involved in promoting resiliency to victimization in a sample of high-risk adolescents. The current work is the first molecular genetic association study to provide empirical evidence linking four dopaminergic and serotonergic polymorphisms to resiliency. Four findings are worthy of mentioning in greater detail.

First, DRD2 was found to decrease the odds of resiliency at Wave 1, Wave 2, and Wave 3. Not surprisingly, analysis of the Add Health data also revealed that DRD2 decreased the odds of lifetime resiliency, and it also was associated with an increase in the total number of victimization incidents across all three waves of data. Second, DRD4 was not associated with any of the measures of resiliency. Third, DAT1 increased the odds of resiliency at Wave 1 and increased the odds of lifetime resiliency, while also being associated with an increase in the total number of victimization experiences. Fourth, 5-HTTLPR increased the odds of resiliency at Wave 1 and Wave 2, and it also increased the odds of lifetime resiliency. In short, all of the genetic polymorphisms except DRD4 showed some associations with various measures of resiliency.

One of the looming questions generated from the analyses is why some of the genetic polymorphisms increased the odds of resiliency while others decreased the odds of resiliency? Because this is the first study exploring the genetic foundations to resiliency against victimization, we are unable to offer a definitive answer to this question. Although speculative, we can offer one plausible explanation. Recall that the genetic variables were coded, based on the findings of prior research, so that higher values indicated more risk for antisocial behaviors, such as crime and violence. As a result, it would seem somewhat reasonable that persons with higher genetic risk—that is, they had higher scores on each of the genetic measures—would be less likely to be resilient against victimization. After all, offenders are more likely to be victims than are nonoffenders, and if the genetic markers are proxies for delinquent involvement, we would expect there to be an inverse association between the genetic risk for antisocial behaviors and resiliency. This explanation was partially supported, in part, by the effects of DRD2 on resiliency.

The exact opposite pattern of results was detected for DAT1 and 5-HTTLPR, where both of these genetic measures actually increased resiliency. Stated differently, more genetic risk for DAT1 and 5-HTTLPR corresponded to

higher odds of being resilient. Although purely post hoc theorizing, we suggest that perhaps respondents with the risk alleles for DAT1 and 5-HTTLPR are extremely violent and thus have developed a reputation for being aggressive. As a result of being involved in crime and delinquency, other youths in their immediate social context know not to victimize them because they would likely retaliate and inflict a significant amount of injury on the perpetrator. Obviously, this explanation needs to be explored more closely in the future, but it does highlight the ways in which genetic predispositions could shape and mold social perceptions that define (sub)cultural theories of antisocial behavior (Anderson, 1999).

These genetic effects are also compatible with recent research carried out by Schreck, Stewart, and Osgood (2008). They examined the association between being an offender and being a victim and explored whether there were unique predictors of these two outcomes. The results of their analysis revealed that it was possible to differentiate between victims and offenders. Our analysis of the Add Health indicates that genes—especially the ones analyzed in the current study—could be one of many different factors that are able to differentiate between victims and offenders.

Although this study has provided perhaps the first findings linking certain factors to resiliency against adolescent victimization, it has a number of limitations that need to be addressed by future researchers. To begin with, the DNA subsample of the Add Health study is drawn from a nationally representative sample, but it is not necessarily nationally representative. Only a subsample of respondents was chosen to be genotyped, and so whether the findings reported here would be generalizable to all at-risk adolescents remains an empirical question. Future research needs to address this limitation by analyzing different samples. It should be noted that efforts are currently underway to genotype all Add Health respondents, which would circumvent issues related to generalizability in future analyses. In addition, the measure of risk, although empirically driven, likely included youths who were only moderately at risk for being victimized. Moreover, the most at-risk adolescents were probably not included in the sample. The main reason for this data limitation is because the Add Health sample was drawn from a school-based research design and the most at-risk and antisocial youths were likely excluded from the sample (Cernkovich, Giordano, & Pugh, 1985; DeLisi, 2005). Future researchers should employ samples that include a more liberal amount of at-risk youths to determine whether the findings of this study would be replicable. Moreover, we only examined the effects that four genetic polymorphisms had on resiliency to victimization. Given that resiliency and victimization are highly complex phenotypes, there is no doubt that hundreds or even thousands of other polymorphisms may be associated with resiliency. We hope that this

study spawns subsequent researchers to explore the effects that other genetic polymorphisms may have on resiliency to victimization.

Perhaps, the most serious limitation that needs to be addressed in the future is that we did not examine the interaction between environments and genes in the prediction of resiliency. Past research, for example, has revealed that genetic effects tend to be strongest when they are paired with certain environmental conditions (Caspi et al., 2002; Rutter, 2006). However, given that this study was exploratory, and given that no prior research had ever identified whether these genes were associated with resiliency, we opted to employ an analysis that was relatively straightforward. Of course, this does not mean that environments are unimportant; rather, our research represents an initial step in the attempt to identify the gene \times environmental effects on resiliency to victimization. We should note that we would anticipate that the genetic effects on resiliency would likely be amplified when paired with certain environmental factors. What those environmental factors might be still remains unknown at this point but represents a salient issue that needs to be examined in great detail.

There is increasing recognition that criminological research needs to move beyond simply delineating risk and protective factors and specify the dynamic nature–nurture interplay that produces antisocial and prosocial outcomes (Moffitt, 2005; Rutter, 2002). Although it may seem somewhat odd for criminologists to think of genes as having protective effects, researchers outside of criminology have already recognized this possibility. Still, much of this research focuses only on how genes may protect against antisocial behaviors, but as the results of our study indicate, genetic factors may also be integrally involved in resiliency to victimization as well.

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Notes

1. A polymorphism is a gene that has more than one allelic variant in existence. Stated differently, a genetic polymorphism is a gene that can vary from person to person.
2. MAOA is an enzyme that breaks down neurotransmitters, such as dopamine and serotonin, from the synapse. The MAOA gene is responsible for coding for the production of the MAOA enzyme.

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