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Desistance from delinquency: The marriage effect revisited and extended [☆]

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Abstract

Desistance from criminal offending has become the source of a considerable amount of research attention. Much of this literature has examined how environmental factors, such as marriage, employment, and delinquent peers contribute to the desistance process. A relatively unexplored possibility, however, is that desistance from criminal behavior is partially due to genetic factors. To test this possibility, data from the National Longitudinal Study of Adolescent Health (Add Health) were used to examine the effects that five different genetic polymorphisms (DAT1, DRD2, DRD4, 5HTT, and MAOA) have on desistance from delinquent involvement. Three broad findings emerged. First, marriage significantly increased desistance. Second, some of the genetic polymorphisms had significant independent effects on desistance. Third, for males, the genetic polymorphisms interacted with marital status to predict variation in desistance. The findings underscore the importance of using a biosocial perspective to examine factors related to criminal desistance.

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1. Introduction

Involvement in delinquency rises markedly in the beginning of adolescence, peaks around the ages of eighteen and nineteen, and begins to decline sharply thereafter (Hirschi and Gottfredson, 1983). By early adulthood most people have “aged out” of delinquent involvement and adult criminal activity is confined to a relatively small pool of chronic offenders (DeLisi, 2005). Although the age-crime curve is one of the most

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firmly established empirical regularities within criminology, the reasons that account for desistance remain the source of debate (Collins, 2004; Gottfredson and Hirschi, 1990; Sampson and Laub, 1993, 2005; Laub and Sampson, 2003; Moffitt, 1993; Warr, 1998). One of the more prominent explanations of desistance, and one that has achieved a considerable amount of empirical support, is Sampson and Laub's (1993) age-graded theory of informal social control. The theory posits that involvement in conventional social institutions, such as marriage, employment, and military, contributes to de-escalation in offending frequency among habitual offenders, even those with a seemingly high criminal propensity. One of the main findings to emerge out of their analysis—and one that paralleled the findings garnered in other studies—was that criminal behavior was relatively stable over long periods of time. Unlike most other extant theorists, Sampson and Laub also noted that some offenders—even those with lengthy criminal records—eventually desisted from engaging in unlawful acts. The question thus became: What factors account for criminal desistance?

To answer this question, Sampson and Laub extended the logic of Hirschi's (1969) social bonding theory into adulthood. Through extensive face-to-face interviews with participants of the Glueck sample and through quantitative analysis of the data, Sampson and Laub discovered that desistance from offending was related to three adult social bonds: employment, marriage, and military service. Offenders who had married, who had gained lawful employment, or who had a history of military service were much more likely to desist from crime than those offenders who lacked these bonds.

The relationship between adult social bonds and desistance from crime was relatively straightforward: once an individual begins to accumulate social capital, such as being married or obtaining a steady job, they have a stake in conformity. Any type of criminal action jeopardizes their social standing in conventional society. An arrest, for example, may cause a spouse to file for a marital dissolution. On the other hand, individuals who fail to develop adult social bonds will have much less to lose by engaging in crime and therefore they will be at-risk for persisting with their antisocial behavior throughout adulthood.

Although Sampson and Laub (1993) identified three different types of adult social bonds, research analyzing the predictors of criminal desistance has centered primarily on marriage. As a result, we follow the lead of other scholars and examine the effect that marriage has on desistance from delinquent involvement. We also explore the potential reasons why some married people desist from offending, whereas other married people persist with their antisocial conduct. Specifically, we examine whether five genetic polymorphisms (DAT1, DRD2, DRD4, 5HTT, and MAOA) condition the effect that marriage has on criminal desistance using data from the National Longitudinal Study of Adolescent Health (Add Health).

2. Literature review

2.1. Marriage and desistance

The most compelling evidence linking changes in marital status to desistance from crime comes from a series of studies by Sampson, Laub, and their colleagues utilizing data originally collected by Sheldon and Eleanor Glueck in the early 20th century (Laub and Sampson, 1993, 2001, 2003; Sampson and Laub, 1993, 2005; Sampson et al., 2006). In this work, a variety of sophisticated analytical procedures evaluated whether marriage has a causal effect on reducing criminal behavior. In one of the first studies investigating this possibility, Sampson and Laub (1993) found that marital attachment significantly increased the chances that an offender would desist from criminal involvement. Similar findings were reported in follow-up studies using both quantitative and qualitative data (Sampson and Laub, 1993). Most recently, Sampson et al. (2006) analyzed yearly data by using a counterfactual research design that employed inverse probability of treatment weighting. The findings generated from this statistical approach revealed convincing evidence linking marriage to desistance. The marriage-desistance association has also been corroborated by researchers using different samples, collected much more recently, and analyzed with different methodological strategies (Farrington and West, 1995; Horney et al., 1995). Taken together, the results of these publications have revealed that even after controlling for individual characteristics, such as latent antisocial personality traits, and even after controlling for prior childhood and adolescent risk factors, and even after taking into account the possibility of selection effects, marriage continues to exert an independent effect on desistance (Laub and Sampson, 2001; Sampson and Laub, 2005).

Sampson and Laub are careful to point out, however, that changes in marital status may not immediately lead to desistance from crime. Instead, they take the position that marriage is like an investment that takes time to grow and develop. For example, Laub et al. (1998) used a dynamic statistical modeling approach to examine whether marriage corresponded to changes in offending. The results of these models revealed that “desistance from crime is facilitated by the development of quality marital bonds, and that this influence is gradual and cumulative over time” (Laub et al., 1998, p. 225). Thus, for Sampson and Laub, marital status *per se* is not related to desistance, but rather the quality of marriage is the salient factor in determining who will and who will not desist from crime.

In spite of evidence suggesting that marriage causes desistance, Warr (1998) interpreted the results of these studies in a very different fashion. He did not dispute that changes in marital status were linked to changes in offending patterns; rather he contended that the association between marriage and desistance was accounted for by an unmeasured factor, delinquent peers. Warr (1998, 2002) began with the observation that associating with delinquent peers is one of the strongest and most consistent predictors of delinquent behavior. He then analyzed data from the National Youth Survey (NYS) to show that individuals who are married spend, on average, “half as much time with their friends each week as do unmarried persons.” Moreover, analysis of the NYS also revealed that “marriage alters the *kinds* of friends with whom individuals associate; it reduces exposure to deviant friends and increases exposure to conventional others” (Warr, 1998, p. 192/196).

Finally, Warr (1998) examined the relationship among marriage, time spent with delinquent peers, and desistance. Without including a measure indexing delinquent peers, marital status had a significant effect on desistance. However, when a measure of delinquent friends was entered into the statistical model it was a significant predictor of desistance, but the marital status variable was not. Thus, the models suggested that the effect of marriage on desistance was mediated by changes in the time spent with delinquent peers (also see Giordano et al., 2003). Similarly, Maume et al. (2005) analyzed waves 5 and 6 from the NYS data to provide more evidence bearing on the interrelationships among marriage, antisocial friends, and desistance. Overall, their models provided empirical support for perspectives advanced by Sampson and Laub (1993) and Warr (1998). Specifically, they found that not only did changes in time spent with delinquent peers facilitate the desistance process, but also that marital quality had an independent effect on desistance. From their study it appears as if both explanations are viable contributions to the desistance literature.

2.2. Desistance and population heterogeneity

That even formerly habitual offenders desisted from crime upon getting married conflicted with the idea that a small cadre of offenders have a high criminal propensity, are less likely to form conventional social bonds, and are more likely to engage in antisocial conduct throughout the life-course (DeLisi, 2005; Moffitt, 1993; Nagin and Paternoster, 2000; Robins, 2005). For instance, in their 30-year longitudinal study, Robins and O’Neal (1958, p. 170) concluded that “criminal activities are more frequently only one expression of a grossly disturbed life pattern of which transiency, violence, and unstable family relations, as well as crime, are typical.” In this sense, some individuals are so antisocial that they not only do not marry but also never form significant, meaningful primary relationships—except with other criminal offenders.

Recently, criminologists have begun to investigate the relationships between marriage, social bonding, and desistance among various types of delinquents. Wright et al. (2001) assessed whether the effects of social ties on crime varied by criminal propensity among respondents from the Dunedin Birth Cohort Study. They found that the desistance-inducing effects of education, employment, family ties, and partnerships were strongest among those with the lowest levels of self-control (measured at the 90th percentile of low self-control). However, the criminogenic effect of associating with delinquent peers achieved its greatest significance among the same group of persons with the lowest levels of self-control. In other words, protective factors and risk factors operated most strongly among those lowest in self-control. Wright et al. (2001, p. 343) speculated that severely antisocial individuals may form prosocial bonds differently from others and that future research should explore the specific characteristics that impel antisocial persons to form social bonds.

Cernkovich and Giordano (2001) examined stability and change in antisocial behavior using a household, community sample and an institutional sample of adjudicated delinquents. Although their work was not explicitly a test of the marriage effect, it did contain ten social bonding measures pertaining to family and peer

attachments. Their results indicated that informal social controls operate differently among individuals depending on their criminality. For instance, among the institutional sample, Cernkovich and Giordano found that only one social bonding variable was related to delinquency. Moreover, adolescent delinquency continued to exert an effect on adult crime even after controlling for demographic and bonding controls. This suggests that weak social bonding is a relative constant among these more chronic offenders (Cernkovich and Giordano, 2001, p. 401). Conversely, informal social controls did significantly reduce crime (i.e., contribute to desistance) among the household sample.

What explains these differential effects of marriage and other social bonds on desistance? A likely answer is criminality. Bushway and his colleagues (2001, pp. 496–497) argued that criminality as a concept is the systematic, causal component of crime and is driven by social, biological, and psychological factors. Similarly, desistance is a process that is theorized to be a function of the biological, psychological, and social changes that mark human development. Unfortunately, criminologists have until now ignored the biological underpinnings of various dimensions of the criminal careers, such as desistance. The following section examines the potential usefulness of behavioral genetics research to illustrate the ways that biological factors, in the current study genetic polymorphisms, interact with environmental conditions to affect delinquency and desistance from delinquency.

2.3. *Gene × environment interactions*

Even though marriage is one of the strongest predictors of desistance, not all married offenders—not even all of those in high quality marriages—eventually desist from criminal behavior. There is reason to believe, therefore, that some undiscovered or unmeasured factor *moderates* the effect that marriage has on desistance. According to this logic, if this “unmeasured factor” is absent, then desistance will not occur; if this “unmeasured factor” is present, then desistance will be much more likely. While we have been unable to locate any studies that examine this possibility, we draw from the behavioral genetic literature and suggest that certain genetic polymorphisms may interact with marriage to reduce criminal involvement.

A long line of behavioral genetic research has recognized the importance of examining the interplay between the environment and genetic factors in the creation of different phenotypes (Moffitt, 2005; Rutter, 2006). One of the more important findings to emerge out of this research is the recognition that genes often condition the effect of the environment and that the environment often conditions the effect of genes. This process is referred to as a gene × environment interaction (G × E). Gene × environment interactions are potentially important explanations to a wide range of phenomena because they can begin to explain why social influences do not always have blanket effects that impact all people in the same way.

Although behavioral geneticists have hypothesized about the importance of gene × environment interactions for the past 30 years or so, only recently, with the mapping of the human genome, have researchers been able to examine whether *measured* genes interact with the environment to create different phenotypes related to criminal involvement. While this type of research is still in its infancy, the results have proven to be very promising. Caspi and his colleagues (2002), for example, analyzed data from the Dunedin Longitudinal Study to examine the interrelationships among childhood maltreatment, the MAOA gene, and antisocial behavior. The results of their analyses revealed that the low-functioning version of the MAOA gene interacted with maltreatment to increase the likelihood of antisocial behaviors. Caspi et al. (2002, p. 851) concluded that their “findings may partly explain why not all victims of maltreatment grow up to victimize others, and they provide epidemiological evidence that genotypes can moderate children’s sensitivity to environmental insults.” The findings from the Caspi et al. (2002) study have been both substantiated (Foley et al., 2004) and negated (Haberstick et al., 2005; Young et al., 2006).

Also relevant to the current research is the recent study conducted by Dick and her associates (2006). In their work, Dick et al. examined the effects of GABRA2 and marital status on alcohol dependence. Analysis of data from the Collaborative Study of the Genetics of Alcoholism (COGA) indicated that GABRA2 and marital status interacted to predict alcohol dependence. The results of this study, along with those garnered in other studies, underscore the importance of examining whether genes modify the effect that social environments have on different life outcomes. Before describing the methodology used in the current study, and in

order to appreciate how the genetic polymorphisms were coded, we first provide a brief introduction to genetics.

2.4. Introduction to genetics

Most genes are made up of two different copies: one copy of the gene is inherited maternally and the other copy of the gene is inherited paternally. Alternative copies of a gene are referred to as alleles. Humans share, on average, 99.9% of their DNA with each other, meaning that most genes are identical across the entire human population. For these genes, only one allele is in existence and thus all people have the same allele. Since these types of genes do not vary from person-to-person, geneticists have not explored the possibility that these genes can contribute to phenotypic differences.¹ For a very small percentage of all genes, however, there are at least two different alleles that can be inherited. Genes that can be made up of more than one allele are referred to as genetic polymorphisms.

Molecular geneticists and behavioral geneticists have pinpointed genes related to the production, transportation, and degradation of neurotransmitters, such as dopamine and serotonin, as particularly promising candidate genes in the etiology of criminal behavior. For these polymorphisms, geneticists have identified certain alleles as “risk alleles” that are thought to increase the risk of antisocial phenotypes. It is important to note that no prior research has ever investigated the role of genetic polymorphisms on desistance from delinquent involvement. As a result, it is not possible to determine which allele should be hypothesized to increase or decrease the odds of desisting. We follow the lead of prior researchers analyzing the genetic determinants of criminal conduct and code alleles related to delinquency as “risk alleles” that should be associated with criminal persistence (i.e., non-desistance). However, it is possible that some alleles may confer an increased risk for one type of antisocial phenotype, but this same allele may actually confer a decreased risk of developing a different antisocial phenotype. To illustrate, cancer researchers have located a gene that suppresses tumor growth in one scenario, but actually triggers cancer growth in another scenario (Mazelin et al., 2004). A genetic “flip-flop effect” suggests that certain alleles may be “risk alleles” for one phenotype, but “protective alleles” for another phenotype.

3. Methods

3.1. Sample

Data for this study come from the National Longitudinal Study of Adolescent Health (Add Health), which is a prospective, nationally-representative sample of youths (Harris et al., 2003; Udry, 1998). The first wave of data was collected in 1994, when respondents were in seventh through twelfth grade. Multistage stratified sampling techniques were used to select 80 high schools and 52 middle and junior high schools. Students attending these schools were then administered a self-report questionnaire that asked questions pertaining to delinquent involvement, social relationships, family life, and other issues relevant to adolescents. Approximately 90,000 students were included in the wave 1 in-school component of the Add Health sample.

A sub-sample of respondents was also selected to take part in the wave 1 in-home component of the Add Health sample. At the in-home interview, the adolescent and the adolescent's primary caregiver (typically the mother) were each administered surveys. Questions on these instruments were designed to provide detailed information about the adolescent's involvement in risky behaviors, their use of drugs and alcohol, and their relationships with family members, among other topics germane to youths. In total, 20,745 adolescents and 17,700 of their primary caregivers were interviewed at wave 1.

¹ Mathematically speaking, constants cannot predict variation in a dependent variable. However, even though most genes may be constant across people, there is some evidence to suggest that because of complex splicing schemes, the proteins manufactured by the same gene may actually be different across people (Ridley, 2003). Also, there is some evidence to suggest that the same allele may be expressed differently depending on whether it was inherited maternally or paternally (Solter, 1988). If this is the case, then non-polymorphic genes, even though they are constants, may be able to account for phenotypic differences. Future research needs to address this possibility.

The second wave of data was collected approximately one to two years after wave 1. The wave 2 survey instrument contained many of the same questions that were used at wave 1. For example, items relating to delinquency, drug use, sexual contact, and social relationships were included on the wave 2 questionnaires. Of the original wave 1 in-home respondents, 14,738 were re-interviewed at wave 2. In 2001–2002, when the respondents were between the ages of 18 and 26 years old, the third wave of data was collected. Many of the questions used at wave 1 and wave 2 were no longer age-appropriate and thus the survey instrument was redesigned to include items that tapped issues relevant to young adults. Questions were added, for instance, that indexed marital history, involvement with the criminal justice system, and drug/alcohol use. Altogether, 15,197 respondents were followed from wave 1 through wave 3 (Harris et al., 2003).

Unlike most other nationally-representative samples, the Add Health data selected a sub-sample of respondents to submit samples of their DNA for genetic typing and analysis. Respondents were eligible to take part in the genetic sub-sample only if they had a sibling or co-twin who was also participating in the Add Health study. In total, 3787 participants were asked to submit buccal cells for genotyping and 2574 agreed to participate. After deletion of missing cases, and after removing one twin from each MZ twin pair, we were left with a final analytic sample size of $N = 1994$.

3.2. Measures

3.2.1. Desistance

One of the main obstacles faced by researchers interested in studying criminal cessation is how to measure desistance (Bushway et al., 2001; Laub and Sampson, 2001; Mulvey et al., 2004). Although different research designs and methodologies have been advanced, there still remains much variability in the way that desistance is operationalized. For instance, prior researchers have used qualitative assessments based on offenders' perceptions for why they desisted (Shover, 1983; Sommers et al., 1994), quantitative measures of criminal thinking styles (Walters, 2002), qualitative life histories (Steffensmeier and Ulmer, 2005), contingency tables with χ^2 statistics to measure desistance (Loeber et al., 1998), and hazards models to access time to failure (persistence/desistance) (Uggen and Kruttschnitt, 1998). Most studies first identify a pool of people who meet a minimum requirement to be considered delinquent or criminal. But, even here there is debate. Some social scientists (e.g., Maume et al., 2005; Warr, 1998), for example, focus on groups of people who have engaged in relatively minor offenses, such as using marijuana, whereas others examine offenders who have long life histories of criminal involvement (Laub and Sampson, 2003; Sampson and Laub, 1993). The time interval examined also varies drastically among desistance studies. Some studies examine offending over the life course, whereas others focus on desistance in adolescence, and still others center on just a few years or months of an offender's life (Horney et al., 1995; Maruna, 2001; Maume et al., 2005; Sampson and Laub, 1993; Shover, 1996; Warr, 1998). Given the heterogeneity in how researchers model desistance, it is apparent that there has yet to be a firmly-established "gold standard" for measuring desistance. However, one convention is to measure criminal behavior occurring at two or more time periods to explore how offending careers unfold (Paternoster, 1989; Shover and Thompson, 1992); the current study similarly follows this approach.

In order to measure desistance we followed a two-step process. First, we created a 15-item delinquency scale at wave 1 that encompassed fighting, stealing, cheating, vandalism, and other age-inappropriate behaviors. Delinquency measures that contain an assortment of offense types are viewed as preferable when examining desistance (Mulvey et al., 2004, p. 220). Given the high frequency with which adolescents experiment with drugs and alcohol, we removed any items measuring substance use. Respondents were asked to indicate the frequency with which they engaged in these different forms of delinquency. We recoded the items into dichotomous variables (0 = no, 1 = yes) and summed the responses together ($\alpha = .78$). Participants who obtained a score of 1 or higher were retained in our sample; those respondents who abstained from delinquency were removed from the final analytic data set.

Second, we created a 12-item criminal activity scale at wave 3. Respondents were presented with a list of items and asked if they taken part in any of the activities within the past 12 months. These items indexed stealing, weapon-use, physical fighting, and fraud, among others ($\alpha = .71$). If the participant responded affirmatively to any of the questions, they were assigned a score of "0"; if they had abstained from criminal

involvement at wave 3, they were assigned a value of “1.” Thus, desistance is scored as a binary variable, where a value of 0 = non-desistance and a value of 1 = desistance.²

3.2.2. Genetic polymorphisms

Participants of the genetic subsample were genotyped for a dopamine transporter gene (DAT1), two dopamine receptor genes (DRD2 and DRD4), a serotonin transporter gene (5HTT), and monoamine oxidase A (MAOA). The results from this genetic analysis provided specific information about each allele (a maternal allele and a paternal allele). Risk alleles were identified by consulting the findings generated from prior genetic studies. Each allele was either assigned a value of “0,” which indicated absence of the risk allele, or a value of “1,” which indicated the presence of the risk allele. We then transformed each polymorphism into a dichotomous variable, where 0 = no risk allele, and 1 = one or two risk alleles.³

3.2.2.1. Dopamine transporter gene (DAT1). The genetic literature has examined the effect that different alleles of the DAT1 gene on various antisocial behaviors. Most of this work has compared the difference between the 9-repeat allele (9R) and the 10-repeat allele (10R). The 10R allele has been identified as the risk allele, because possessing the 10R increases the likelihood of developing a number of maladaptive disorders (Gill et al., 1997; Rowe et al., 1998, 2001). Respondents who possessed either one or two 10R alleles were assigned a score of “1,” whereas participants with only 9R alleles were scored a value of “0”. Following prior researchers, participants who had alleles other than the 9R or the 10R were removed from the sample (Hopfer et al., 2005).

3.2.2.2. Dopamine receptor gene (DRD2). Two different alleles are available for the dopamine D2 receptor polymorphism: the A1 allele and the A2 allele. Most research has demonstrated that the A1 allele is considered the risk allele because it is positively related to a number of behavioral and psychiatric disorders (Arinami et al., 1993; Berman et al., 2002; Blum et al., 1997; Comings et al., 2001; Connor et al., 2002; Hopfer et al., 2005). The maternal and paternal allele variables were coded so the A2 allele corresponded to a score of “0” and the A1 allele corresponded to a score of “1.” If the respondent inherited either one or two risk alleles they were assigned a value of “1”; if they did not inherit a risk allele they were assigned a value of “0”.

3.2.2.3. Dopamine receptor gene (DRD4). The dopamine D4 receptor gene (DRD4) is one of the most examined polymorphisms in genetic research (Faraone et al., 1999, 2001). Findings garnered from quantitative genetic studies and the results of a recent meta-analysis indicate that the 7-repeat allele is the risk allele for a range of antisocial outcomes (Faraone et al., 1999, 2001). Following prior research using the Add Health data (Hopfer et al., 2005), alleles that had repeat sequences greater than or equal to 7 were coded as risk alleles and all other genetic variants were not. Participants who inherited either one or two risk alleles were assigned a value of “1”; participants who did not inherit a risk allele were assigned a value of “0.”

3.2.2.4. Serotonin transporter gene (5HTT). Human genetic research has identified two different alleles for the serotonin transporter gene (5HTT): a short allele (484 base pairs) and a long allele (512 base pairs). The

² The current authors calculated all of the models using alternative measures of desistance. For example, desistance was also measured by using a more rigorous score on the wave 1 delinquency. Instead of examining whether the adolescent had ever been involved in delinquency, we also examined those respondents who had a score of 6 or higher on the delinquency scale. However, this criterion truncated the sample size (especially for the gender-specific models) and reduced the power to detect significant findings. Nonetheless, most of the genetic polymorphisms that were significant in the findings reported in the text approached conventional $p < .05$ significance levels with this alternative measurement strategy.

³ Even though researchers often code genes, where 0 = 0 risk alleles and 1 = 1 or 2 risk alleles (Mill et al., 2006), we explored other coding schemes, such as a trivariate measure, where 0 = no risk allele, 1 = 1 risk allele, and 2 = 2 risk alleles. The general pattern of findings was the same, but the main effects of the dopamine genes were attenuated, with some of them, in some of the models, falling from statistical significance. The only interaction term, however, to drop from statistical significance was the DRD2 × marriage effect. All of the remaining interactions remained statistically significant with this different measurement technique. In addition, the serotonin transporter gene had a statistically significant effect on likelihood of marriage.

evidence is mixed on which of the two alleles should be considered the risk allele; some evidence suggests the short allele is the risk allele (Munafò et al., 2005; Türker et al., 1998), whereas other research suggests the long allele is the risk allele (Beitchman et al., 2003; Seeger et al., 2001). We opted to code the short allele as the risk allele, so participants who possessed either one or two short alleles were assigned a value of “1” and subjects who only inherited the long allele were assigned a value of “0.”

3.2.2.5. Monoamine oxidase A (MAOA). Two different alleles—a low-activity allele and a high-activity allele—are available for the MAOA polymorphism. The low-activity allele (i.e., the 2- and 3-repeat alleles), when compared with the high-activity allele (3.5-, 4-, and 5-repeat alleles) is usually identified as the risk allele (Haberstick et al., 2005).

The monoamine oxidase A gene (MAOA) is located on the X-chromosome. Bear in mind that males have one X-chromosome and one Y-chromosome, whereas females have two X-chromosomes. Thus, males only have one MAOA allele, whereas females have two MAOA alleles. Males were assigned a score of “1” if they inherited the low-activity allele; if they inherited the high-activity allele they were assigned a value “0.” Females were assigned a score of “1” if they inherited either one or two low-activity alleles; they were assigned a value of “0” if they had inherited two high-activity alleles. The MAOA variable is removed from the models that analyze both genders simultaneously, but it is included in the gender-specific models.

3.2.3. Life-course transition

3.2.3.1. Marital status. In order to assess the effect that marriage has on desistance, we included a one-item dichotomous variable that tapped the respondent’s marital status (0 = single, 1 = married). Unfortunately, respondents were not asked any questions about the quality of the relationship, about attachment to their spouse, or about their spouses’ involvement in antisocial behavior. Without having these types of questions available, we were unable to examine whether desistance was affected by the quality of the marriage. By using only a binary variable, we are likely underestimating the effect that marriage has on desistance and are also less likely to detect significant gene \times environment interactions between marriage and the genetic polymorphisms. We are essentially treating all marriages—even those that are highly dysfunctional or that entail daily physical abuse—as having equal effects on the odds of desisting. In all likelihood, if the Add Health data included measures about relationship quality, the effect of marriage on desistance would be even stronger because we would be able to differentiate low-quality marriages from high-quality marriages (Laub et al., 1998).

3.2.4. Control variables

3.2.4.1. Family risk. To capture the effects that early family environments have on desistance in adulthood, a global measure of family risk was created. To create this index, three different scales indexing the mother–offspring relationship were developed. At wave 1, adolescents were asked how close they felt to their mother and how much they thought their mother cared about them. Following prior researchers analyzing the Add Health data (Haynie, 2001; Schreck et al., 2004), responses to these two items were added together to form the maternal attachment scale ($\alpha = .64$). This scale was recoded so that higher scores reflected less maternal attachment.

At wave 1, adolescents were also asked whether their mother and them went shopping, played a sport, went to a movie, play, or sporting event, talked about a personal problem, and worked on a project for school in the past 4 weeks. Items that the adolescent endorsed were assigned a value of “1.” Similar to the measure developed by Crosnoe and Elder (2004) and by Sieving et al. (2001), responses to the items were then summed to form the maternal involvement scale ($\alpha = .55$). This scale was then recoded such that higher scores reflected less maternal involvement.

The last scale—the maternal disengagement scale—was comprised of five different questions asked to the adolescent at wave 1. This scale was developed to tap whether the mother was cold and detached from their adolescent. For instance, respondents reported whether they thought their mother communicated with them effectively. Higher scores on this scale indicate higher levels of maternal disengagement ($\alpha = .84$).

All three of these scales were then factor analyzed, and the results of the analysis and an inspection of the scree plot revealed that the maternal scales all loaded on the same factor. As a result, the regression factor scores were calculated to create a global measure of family risk.

3.2.4.2. *Criminal father.* We include a one-item measure of whether the respondent's biological father had ever been incarcerated (0 = no, 1 = yes). This measure captures not only socialization effects, but also genetic effects on propensity to offend (Moffitt, 2005).

3.2.4.3. *Low self-control.* Measures of low self-control are among the strongest predictors of delinquent involvement (Pratt and Cullen, 2000). To examine the effect that low self-control has on desistance, we developed a wave 1 low self-control scale. Prior researchers analyzing the Add Health data have advocated the use of a five-item low self-control scale that approximates Gottfredson and Hirschi (1990) conceptualization of self-control (Perrone et al., 2004). Adolescents, for example, were asked whether they had trouble keeping their mind focused, whether they had trouble paying attention, and whether they had difficulty finishing their homework. Responses to these items were then added together to form the wave 1 low self-control scale ($\alpha = .63$).

3.2.4.4. *Sociodemographics.* Finally, to control for potentially confounding effects, we included three different sociodemographic variables. Age was measured in years, and race (0 = white, 1 = nonwhite) and gender (0 = female, 1 = male) were coded as dichotomous dummy variables.

3.3. Analytical plan

We analyze the Add Health data in a series of interrelated steps. Recall that the measure of desistance is created by first identifying those adolescents who were engaged in delinquency during wave 1 interviews. Our first statistical model therefore is a logistic regression equation where 0 = non-delinquent and 1 = delinquent. Essentially these models will reveal the factors that are related to delinquent involvement. In order to determine whether the correlates to delinquency vary between males and females, the equations will also be estimated separately by gender. In addition, to include the MAOA variable (remember, MAOA is X-linked) as a covariate, we had to break the analyses out by gender.

Next, we employ the binary desistance measure (0 = persistence, 1 = desistance) as the dependent variable in a series of logistic regression equations. The first model will estimate the independent and additive effects that the genetic polymorphisms and marriage have on desistance. We also include three different interaction terms to determine whether the effect that marriage has on desistance is moderated by the genetic polymorphisms. All of the main effect terms were mean centered prior to constructing the multiplicative interaction terms to reduce the potential of collinearity (Jaccard et al., 1990). Variance inflation factors and tolerance limits were also calculated to check for multicollinearity. The results of these statistics revealed that the interaction between DAT1 and marriage was highly collinear. Including this term in the models would have provided unstable parameter estimates. As a result, we opted to remove the DAT1 \times marriage interaction term in all of the analyses. However, none of the other interaction terms were affected by multicollinearity and thus were retained in the analyses.⁴

Prior research has suggested that there may be gender differences in desistance (Uggen and Kruttschnitt, 1998). To take this possibility into account, we re-estimated all of the desistance models for males and for females. In these models, we were also able to introduce the MAOA polymorphism and examine whether MAOA interacted with marital status to predict desistance.

Finally, some behavioral genetic literature has revealed that the propensity to marry is highly heritable for both males and females (Johnson et al., 2004). Quantitative genetic analysis has also linked alleles in the GABRA2 gene to marital status (Dick et al., 2006). As a result, in our last model we use marital status (0 = single, 1 = married) as the dependent variable in a logistic regression model to determine whether the genetic polymorphisms are associated with marriage.

⁴ We chose to use a .05 and a .10 level of significance in the models. The main reason for doing so is because statistical interactions are inherently difficult to detect (McClelland and Judd, 1993). And as Rutter and Silberg (2002) note, gene \times environment interactions are extremely difficult to detect when using multiplicative interaction terms. To compensate, we chose to use both a .05 and .10 probability level.

4. Results

We begin our analysis by predicting involvement in delinquency at wave 1 (0 = non-delinquent, 1 = delinquent) for the full sample and separately by gender. As shown in the first column of Table 1, the serotonin transporter gene (5HTT) has a significant negative effect on delinquent involvement, whereas the other three genetic polymorphisms fail to reach statistical significance. In addition, and consistent with predictions, family risk and low self-control have significant positive effects on the wave 1 delinquency scale. The middle column of Table 1 contains the result for the male sample. Similar to the findings generated using the full sample, 5HTT maintains a significant inverse association with delinquency. The low self-control scale maintains a positive relationship with the delinquency scale. The last model in Table 1 depicts the results for females. In this equation, 5HTT has a negative effect on delinquency, while family risk, low self-control, and race all have positive effects on delinquent involvement.

Next, we examine the genetic and environmental effects on desistance. As Table 2 reveals, the dopamine D4 receptor gene (DRD4) has a direct and independent effect on desistance. The effect is negative, meaning that as the number of risk alleles increases, the odds of desistance increase as well. Remember, however, that no research has ever examined whether genetic polymorphisms are related to desistance, so the negative coefficient for this polymorphism is not unexpected. Consistent with prior research (Laub et al., 1998; Sampson and Laub, 1993), married persons are more likely to desist from delinquency. In addition, respondents with a criminal father and respondents with low self-control are at risk for persisting with their aberrant behavior into adulthood.

Models 2, 3, and 4 in Table 2 introduce the gene \times environment interactions. For all of these equations, DRD4 and marriage have significant independent effects on desistance, but none of the $G \times E$ interaction terms are significant for the full sample. In this case, the effect that marriage has on desistance is not conditioned by the genetic polymorphisms.

Table 3 presents the results of the logistic regression models predicting desistance for males. Model 1 includes all five of the genetic polymorphisms, marital status, and the control variables as covariates. For this model, the dopamine transporter gene (DAT1) has a statistically significant negative effect on desistance, while the dopamine D2 receptor gene (DRD2), DRD4, and monoamine oxidase A (MAOA) have statistically significant positive effects on desistance. Similar to the models estimated for the full sample, being married increased the odds of desisting.

Table 1
Logistic regression models predicting delinquent involvement at wave 1 ($N = 1994$)

	Full sample		Male sample		Female sample	
	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE
Genetic polymorphisms						
Dopamine transporter gene	.13	.25	.00	.37	.21	.36
Dopamine D2 receptor gene	.10	.12	.15	.18	.04	.17
Dopamine D4 receptor gene	.08	.12	.01	.18	.03	.17
Serotonin transporter gene	-.33 ^b	.13	-.32 ^a	.19	-.32 ^a	.18
Monoamine oxidase A gene			.22	.18	-.01	.17
Control variables						
Family risk	.19 ^b	.07	-.01	.10	.28 ^b	.10
Criminal father	-.08	.17	.07	.25	-.23	.23
Low self-control	.26 ^b	.02	.21 ^b	.03	.32 ^b	.04
Age	-.01	.04	.01	.05	-.03	.05
Race	.16	.13	-.20	.19	.45 ^b	.19
Gender	.03	.12				
Cox and Snell R^2	.09		.06		.13	

^a Significant at the .10 level, two-tailed.

^b Significant at the .05 level, two-tailed.

Table 2
Logistic regression models predicting desistance for the full sample ($N = 1555$)

	Model 1		Model 2		Model 3		Model 4	
	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE
Genetic polymorphisms								
Dopamine transporter gene	-.40	.29	-.40	.29	-.40	.29	-.40	.29
Dopamine D2 receptor gene	.10	.12	.05	.13	.10	.12	.10	.12
Dopamine D4 receptor gene	.22 ^a	.12	.22 ^a	.13	.22 ^a	.13	.22 ^a	.12
Serotonin transporter gene	-.16	.13	-.16	.13	-.16	.13	-.17	.13
Life-course transition								
Marriage	1.26 ^b	.22	1.03 ^b	.27	1.25 ^b	.27	1.11 ^b	.40
Control variables								
Family risk	-.01	.06	-.01	.13	-.01	.06	-.01	.06
Criminal father	-.28 ^a	.16	-.27 ^a	.16	-.28 ^a	.16	-.28 ^a	.16
Low self-control	-.08 ^b	.02	-.08 ^b	.02	-.08 ^b	.02	-.08 ^b	.02
Age	.16 ^b	.04	.16 ^b	.04	.16	.04	.16	.04
Race	-.14	.13	-.14	.13	-.14	.13	-.14	.13
G × E interactions								
DRD2 × marriage			.59	.45				
DRD4 × marriage					.04	.44		
5HTT × marriage							.21	.47
Cox and Snell R^2	.10		.10		.10		.10	

^a Significant at the .10 level, two-tailed.

^b Significant at the .05 level, two-tailed.

Table 3
Logistic regression models predicting desistance for males ($N = 745$)

	Model 1		Model 2		Model 3		Model 4		Model 5	
	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE
Genetic polymorphisms										
Dopamine transporter gene	-.75 ^b	.38	-.74 ^a	.38	-.77 ^b	.38	-.75 ^b	.38	-.75 ^b	.38
Dopamine D2 receptor gene	.27 ^a	.16	.18	.17	.26	.16	.27 ^a	.16	.27	.16
Dopamine D4 receptor gene	.32 ^a	.17	.30 ^a	.17	.24	.17	.32 ^a	.17	.32 ^a	.17
Serotonin transporter gene	.07	.17	.07	.17	.07	.17	.08	.17	.07	.17
Monoamine oxidase A	.28 ^a	.16	.28 ^a	.16	.28 ^a	.16	.28 ^a	.16	.18	.17
Life-course transition										
Marriage	1.58 ^b	.32	1.09 ^b	.37	1.23 ^b	.34	1.74 ^b	.64	1.16 ^b	.35
Control variables										
Family risk	-.04	.09	-.05	.10	-.04	.09	-.04	.09	-.05	.10
Criminal father	-.15	.22	-.14	.22	-.15	.22	-.15	.22	-.16	.22
Low self-control	-.07 ^b	.03	-.07 ^b	.03	-.07 ^b	.03	-.07 ^b	.03	-.08 ^b	.02
Age	.13 ^b	.05	.13 ^b	.05	.13 ^b	.05	.12 ^b	.05	.13 ^b	.05
Race	.04	.18	.03	.18	.04	.18	.04	.18	.05	.18
G × E interactions										
DRD2 × marriage			1.60 ^a	.82						
DRD4 × marriage					1.81 ^a	1.08				
5HTT × marriage							-.21	.73		
MAOA × marriage									2.03 ^a	1.08
Cox and Snell R^2	.08		.09		.09		.08		.09	

^a Significant at the .10 level, two-tailed.

^b Significant at the .05 level, two-tailed.

Models 2, 3, 4, and 5, introduce the gene \times environment interaction terms. In model 2, DRD2 interacts with marriage to predict desistance. Substantively, this means that respondents who possessed the A1 allele and who were married had a greater likelihood of desisting compared to respondents with just the A1 allele or with respondents who were only married. The inclusion of this $G \times E$ reduces the main effect of DRD2 to statistical insignificance; however, marriage continues to exert an independent effect on desistance. Model 3 shows that the interaction between DRD4 and marriage is statistically significant and that the independent effect of DRD4 falls from statistical significance. Marital status remains statistically significant. The interaction between DRD4 and marriage can be interpreted to mean that those respondents who possessed the 7R allele and who were married had at greater odds of desisting in comparison with respondents who were only married or who only possessed the 7R allele. As revealed in Model 4, the interaction between 5HTT and marriage is not significant. Finally, in Model 5, the interaction between MAOA and marriage is statistically significant and entering this $G \times E$ term into the equation reduces the main effect of MAOA to insignificance. As with all of the other equations, the independent effect of marriage on desistance remains statistically significant.

Table 4 presents the findings of the multivariate models for females. Across all five of the models in the table, three findings emerge. First, only one genetic polymorphism—5HTT—has a statistically significant and negative independent effect on desistance. Second, marriage increases the odds of desistance in models 1, 2, and 3; however, when interaction terms are introduced for 5HTT \times marriage and for MAOA \times marriage (models 4 and 5), the effect that marriage has on desistance falls from significance. Third, none of the $G \times E$ interaction terms are significant predictors of desistance.

Last, and as shown in Table 5, we examine whether the genetic polymorphisms have an effect on propensity to marriage. Once the effects of the control variables are held constant, none of the genetic polymorphisms are associated with marital status. These insignificant findings are observed for the full sample and for the gender-specific models.

Table 4
Logistic regression models predicting desistance for females ($N = 810$)

	Model 1		Model 2		Model 3		Model 4		Model 5	
	<i>B</i>	SE	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE
Genetic polymorphisms										
Dopamine transporter gene	.18	.43	.18	.43	.19	.43	.18	.43	.16	.43
Dopamine D2 receptor gene	-.13	.19	-.13	.20	-.13	.19	-.13	.19	-.14	.19
Dopamine D4 receptor gene	.09	.19	.09	.19	.17	.21	.09	.19	.09	.19
Serotonin transporter gene	-.45 ^b	.20	-.45 ^b	.20	-.45 ^b	.20	-.50 ^b	.22	-.46 ^b	.21
Monoamine oxidase A	-.11	.20	-.11	.20	-.11	.20	-.11	.20	-.22	.22
Life-course transition										
Marriage	.89 ^b	.30	.91 ^b	.41	1.22 ^b	.45	.52	.52	.39	.42
Control variables										
Family risk	.02	.09	.02	.09	.01	.09	.02	.09	.02	.09
Criminal father	-.40 ^a	.24	-.40 ^a	.24	-.38	.24	-.39 ^a	.24	-.42 ^a	.24
Low self-control	-.09 ^b	.03	-.09 ^b	.03	-.08 ^b	.03	-.09 ^b	.03	-.09 ^b	.03
Age	.22 ^b	.06	.22 ^b	.06	.21 ^b	.06	.22 ^b	.06	.22 ^b	.06
Race	-.42 ^b	.20	-.42 ^b	.20	-.41 ^b	.20	-.42 ^b	.20	-.41 ^b	.20
$G \times E$ interactions										
DRD2 \times marriage			-.05	.59						
DRD4 \times marriage					-.65	.60				
5HTT \times marriage							.53	.62		
MAOA \times marriage									.88	.59
Cox and Snell R^2	.06		.06		.06		.06		.06	

^a Significant at the .10 level, two-tailed.

^b Significant at the .05 level, two-tailed.

Table 5
Logistic regression models predicting marital status

	Full sample		Male sample		Female sample	
	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE
Genetic polymorphisms						
Dopamine transporter gene	.33	.36	-.05	.47	.75	.55
Dopamine D2 receptor gene	.01	.14	.23	.22	-.13	.19
Dopamine D4 receptor gene	.05	.14	-.15	.23	.18	.18
Serotonin transporter gene	.21	.15	.32	.24	.11	.20
Monoamine oxidase A gene			-.12	.23	-.19	.19
Control variables						
Family risk	-.06	.07	-.02	.13	-.09	.09
Criminal father	.43 ^a	.18	.55 ^a	.27	.32	.24
Low self-control	.05 ^a	.02	.03	.04	.06 ^a	.03
Age	.41 ^a	.05	.44 ^a	.07	.40 ^a	.06
Race	-.54 ^a	.16	-.40	.26	-.57 ^a	.21
Gender	-.62 ^a	.14				
Cox and Snell R^2	.07		.07		.07	

^a Significant at the .05 level, two-tailed.

5. Discussion

The current study examined the genetic and environmental correlates of desistance from delinquent involvement using data containing genetic markers, marriage, self-control and other important controls, and interaction terms modeled over a seven-year span of human development. The results of the multivariate models predicting desistance produced three broad findings. First, and consistent with prior research, married persons were significantly more likely to desist than were non-married persons. Second, the dopaminergic polymorphisms and MAOA had significant direct effects on desistance for males. For females, the serotonin transporter gene was associated with desistance. Third, and perhaps most importantly, analysis of the Add Health data revealed significant interactions between marriage and DRD2, DRD4, and MAOA in the prediction of desistance for males. *This is the first time that a gene × environment interaction has been detected for desistance from delinquency.*

The importance of the interaction between marriage and certain of the polymorphisms should not be casually overlooked. Researchers have long recognized that the same environmental conditions can have different effects on different people. The current research provides a genetic explanation of why marriage leads to desistance for one person, but may not lead to desistance for another person: the possession of certain alleles conditions the effect that marriage has on desistance. A married person with one type of allele may be very likely to desist, whereas another married person, with a different allele, will be at-risk for persisting with their antisocial behaviors. The current analyses of the Add Health data provide initial support of this view and provide empirical substantiation to studies that previously speculated that biological factors partially account for the differential effects of social bonding on antisocial behavior (Bushway et al., 2001; Cernkovich and Giordano, 2001; Collins, 2004; DeLisi, 2001; Moffitt, 1993, 2005; Wright et al., 2001).

Even though genes interacted with marriage in the prediction of desistance, some of the genetic effects were opposite of what has been reported in studies examining the genetic effects on antisocial behaviors. For example, the A1 allele of DRD2 has been linked to certain problem behaviors, such as compulsive gambling (Comings et al., 2001). In the current study, the A1 allele was related to an increased chance of desisting for males. Similar patterns were found in respect to DRD4 and MAOA. These findings were not wholly unexpected. Geneticists have long recognized that a certain allele may be a risk allele for one phenotype, but actually be a protective allele for another phenotype (see Rutter, 2006). Similarly, the causes of desistance are not necessarily the opposite of the causes of delinquent involvement. Given that no prior research has ever examined the genetic effects on desistance, there was not an *a priori* reason to hypothesize that alleles that increase antisocial behavior would decrease delinquent involvement. Taken

together, there is good reason to believe that some of the alleles found to increase maladaptive behaviors may also increase the odds of desistance.

Why would genes related to neurotransmitters be associated with desistance from offending behaviors? While only speculative, the current authors offer one reason. According to Raine (1993, p. 85) “serotonin plays an inhibitory role (in aggression) while norepinephrine and dopamine each play facilitative roles in aggression.” Heightened levels of dopamine and reduced levels of serotonin have been linked to an increased involvement in antisocial behavior. Most importantly, however, is that levels of neurotransmitters ebb and flow over the life course and these fluctuations in biochemical levels parallel closely the age-crime curve (Collins, 2004). During adolescence, for example, dopamine increases while serotonin is reduced. Near the end of adolescence and at the beginning of adulthood, dopamine levels recede while serotonin levels become elevated. It is at this point in the life course that delinquent involvement also plummets quite drastically (Collins, 2004). Bear in mind that certain genes—including the ones examined in our analysis—partially control neurotransmitter levels. Thus, the genes that are responsible for producing, transporting, and breaking-down neurotransmitters may be able to shed light on the potential causes of the age-crime curve, including the sharp decline in delinquent involvement in young adulthood.

There are limitations of our study. First, the Add Health data only contained information about marital status; questions assessing the quality of the marriage were not collected. Without being able to differentiate those individuals in high-quality marriages from those individuals in low-quality marriages provides a more conservative test of the effects that marriage has on desistance. At the same time, the ability to detect significant $G \times E$ s is also reduced. Even so, the models provided substantial evidence suggesting that marriage has significant direct effects and significant interactive effects on desistance from delinquent involvement. Future research needs to explore how genes interface with marital quality in the etiology of desistance. Second, the current authors were only able to examine the correlates of desistance for adolescents who were minimally involved in delinquency. A more elaborate approach, where only chronic delinquents were analyzed, would not be supported with the current sample size. Finally, only a sub-sample of Add Health respondents submitted samples of their DNA for genotyping, raising the possibility that the findings are not generalizable to all people. However, it is important to point out that Add Health sample is much larger and much more representative of the population than are those samples that are typically used in genetic analysis.

In their overview of the research on desistance, Mulvey and his colleagues (2004, p. 218) assessed:

It appears that the desistance process involves interactions among dynamic changes in offenders' psychological states, developmental capacities, and social contexts. Expanding the rich leads from qualitative work and the initial quantitative analyses of existing longitudinal data sets, therefore, will require a sustained and coordinated research agenda. It will require a series of systematic investigations, each illuminating another aspect of the larger desistance process.

The current study should be viewed as one aspect of a broader, interdisciplinary study of desistance. It is noteworthy that a growing number of social scientists have called for the integration of biological and genetic factors into current sociologically-oriented explanations of crime (Ellis, 1996; Walsh, 2002). As the current research reveals, genetic explanations are not incompatible with environmental explanations; in fact, they are complementary.

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