

Published in final edited form as:

*J Marriage Fam*. 2015 October ; 77(5): 1217–1233. doi:10.1111/jomf.12208.

# Does Marriage Moderate Genetic Effects on Delinquency and Violence?

**Yi Li**,

Department of Sociology, 155 Hamilton Hall, University of North Carolina, Chapel Hill, NC 27599

**Hexuan Liu**, and

Department of Sociology and Carolina Population Center, 206 W. Franklin St., University of North Carolina, Chapel Hill, NC 27599

**Guang Guo**

Department of Sociology, Carolina Population Center, and Carolina Center for Genome Sciences, 206 W. Franklin St., University of North Carolina, Chapel Hill, NC 27599

Yi Li: yili@live.unc.edu

## Abstract

Using data from the National Longitudinal Study of Adolescent to Adult Health (N = 1,254), the authors investigated whether marriage can foster desistance from delinquency and violence by moderating genetic effects. In contrast to existing gene–environment research that typically focuses on one or a few genetic polymorphisms, they extended a recently developed mixed linear model to consider the collective influence of 580 single nucleotide polymorphisms in 64 genes related to aggression and risky behavior. The mixed linear model estimates the proportion of variance in the phenotype that is explained by the single nucleotide polymorphisms. The authors found that the proportion of variance in delinquency/violence explained was smaller among married individuals than unmarried individuals. Because selection, confounding, and heterogeneity may bias the estimate of the Gene × Marriage interaction, they conducted a series of analyses to address these issues. The findings suggest that the Gene × Marriage interaction results were not seriously affected by these issues.

### Keywords

aggression; marriage; National Longitudinal Study of Adolescent to Adult Health (Add Health); youth/emergent adulthood

---

The impact of marriage on individuals' well-being has long been studied. Married individuals exhibit higher levels of healthy behaviors, survival probability, wages, and so forth, than unmarried individuals (Waite, 1995). Of particular interest has been the inhibiting effect of marriage on antisocial behavior such as delinquency and crime. Researchers have found that the transition to marriage is linked to a decline in antisocial behavior. This desistance effect of marriage has been noted in multiple cohorts (King, Massoglia, &

---

This article was edited by Robert Crosnoe.

Macmillan, 2007; Sampson & Laub, 1993) and in different countries (Blokland & Nieuwbeerta, 2005; Theobald & Farrington, 2009).

In recent years, researchers have increasingly incorporated genetic variables into their examinations of the effects of social environments on antisocial behavior (e.g., Caspi et al., 2002). The findings that social factors interact with genes to influence antisocial behavior underline the importance of *gene–environment interaction* (*G×E*, a term that refers to processes wherein genetic influences depend on environmental factors, or vice versa). But existing G×E research almost exclusively focuses on one or a few genetic variants. Unlike rare Mendelian traits that are determined by a single gene or allele, overall genetic influence on antisocial behavior comprises a large number of genetic effects (Anholt & Mackay, 2012). Therefore, it is essential to examine more than a few genetic variants in G×E research on antisocial behavior.

In this study, we extended previous G×E research by considering a large number of genetic variants. Drawing on data from the National Longitudinal Study of Adolescent to Adult Health (Add Health; see http://www.cpc.unc.edu/projects/addhealth), we examined whether marriage moderates the collective influence of 580 single nucleotide polymorphisms (SNPs) in 64 genes on delinquency and violence. To do so we used a recently developed mixed linear model implemented in the genome-wide complex traits analysis (GCTA) software (Yang, Lee, Goddard, & Visscher, 2011). This new method estimates a heritability parameter: the proportion of variance in the phenotype that is jointly explained by the SNPs. We examined the Gene × Marriage interaction by comparing the proportion of variance in antisocial behavior explained by 580 SNPs among married and unmarried individuals. The samples in the mixed linear models were drawn from the Add Health genetic subsample. Our approach might be implemented using larger data in the future. Selection, confounding, and heterogeneity can bias the estimate of the Gene × Marriage interaction. We conducted a series of analyses to address these issues.

## Background

### Marriage and Antisocial Behavior

Social scientists have long noticed that marriage is an important life course transition with seemingly far reaching impact. In general, married individuals consider marriage a long-term contract (Waite, 1995). To maintain the contract, they tend to do things that pay off in the long run, and they refrain from behaviors that bring instant gratification or the possibility of harmful consequences. This idea is supported by research findings indicating that marriage may deter criminal activity and deviant behavior (e.g., Blokland & Nieuwbeerta, 2005; Farrington & West, 1995; Horney, Osgood, & Marshall, 1995; Sampson & Laub, 1993; Warr, 1998).

The effect of marriage on antisocial behavior may be thought of as the result of three processes. First, marriage may strengthen connections within the family. Married couples are connected to each other in relationships for which there are strong social norms. Married people tend to fulfill the normative expectations implied by marriage. On the basis of social control theory (Hirschi, 1969), Sampson, Laub, and colleagues have focused on bonds and

ties created within marriage (Laub, Nagin, & Sampson, 1998; Laub & Sampson, 2003; Sampson & Laub, 1993; Sampson, Laub, & Wimer, 2006). This line of research proposes that marriage establishes strong bonds and ties that prevent individuals from committing crime over the life course. Interpersonal attachment to a partner serves as a control mechanism. Over time, individuals invest more and more social and financial resources in a marriage. Engaging in criminal activity is not a rational choice because it threatens that investment. Summarizing their position, Sampson and Laub (1993) stated that marriage creates "interdependent systems of obligation and constraints that impose significant costs for translating criminal propensities into action" (p. 141).

Second, marriage may weaken connections outside of the family that might lead to antisocial behavior. Peer influence can be a major source of variation in antisocial behavior (Osgood, Wilson, O'Malley, Bachman, & Johnston, 1996). The transition to marriage usually means that routine activities are primarily devoted to the spouse and family. Warr (1998) showed that marriage may weaken or disrupt connections with peers, including delinquent ones. After the transition to marriage, time spent with peers decreases dramatically. As a result, opportunities and motivations to engage in crime and delinquency are significantly limited. Warr found that these changes largely account for the association between marriage and antisocial behavior. In addition, the obligations that come with marriage tend to leave less time for leisure activities outside of the family (Osgood & Lee, 1993); therefore, unstructured socializing with delinquent peers may also be limited.

Third, marriage may lead to changes at the psychological level and, by extension, alter one's perception of antisocial behavior. Because marriage implies meaningful commitment, married persons may develop a sense of obligation to their partners that reduces the appeal of behaviors that might threaten the relationship. Cognitive and identity transformations are at work when individuals desist from antisocial behavior (Giordano, Cernkovich, & Rudolph, 2002). After getting married, individuals are open to making cognitive changes, and they usually treat the relationship seriously. For example, stealing and drug use, if once condoned, are no longer viewed as proper and viable. As a consequence, deviant behavior is less likely to occur. Emotional regulation is also important to the success of desistance (Giordano, Schroeder, & Cernkovich, 2007). Negative emotions associated with crime, coupled with an increased ability to manage one's emotions, may lead to a decline in criminal activity. An implication of these findings is that marriage might involve changes in emotional regulation that help individuals desist.

## G×E Interaction Research on Antisocial Behavior

G×E studies on antisocial behavior have focused on five genes (a) the monoamine oxidase A (*MAOA*) gene, (b) the dopamine D2 receptor (*DRD2*) gene, (c) the serotonin transporter gene (*5-HTT*), (d) the dopamine receptor gene (*DRD4*), and (e) the dopamine transporter gene (*DAT1*). Using data from the Dunedin Multidisciplinary Health and Development study, Caspi and colleagues (2002) reported that the effect of childhood maltreatment on antisocial behavior is weaker among individuals with high *MAOA* activity than those with low *MAOA* activity. Using data from Add Health, Guo and colleagues (Guo, Roettger, & Cai, 2008) found that the effects of the *DRD2* and *MAOA* genes on delinquency are

conditional on family processes, school processes, and social networks. More recently, Simons and colleagues (2011) found that the presence of both a short allele in the *5-HTT* gene and a long allele in the *DRD4* gene interacts with social environments to affect aggression.

Marriage is an important social institution that may also moderate genetic effects on antisocial behavior. To date, only one study has examined the effect of the Gene × Marriage interaction on delinquency (Beaver, Wright, DeLisi, & Vaughn, 2008). Using Add Health data, Beaver et al. (2008) tested the interactions between marriage and five genes: the *DAT1*, *DRD2*, *DRD4*, *5-HHT*, and *MAOA* genes. They found significant interactions (at the .10 level) only among men. The temporal order between marriage and delinquency was not considered.

## Genetic Effects on Antisocial Behavior

In the aforementioned G×E studies, genetic effects are represented by only a few genetic variants. Antisocial behavior, however, is influenced by a large number of genes (Craig & Halton, 2009). Researchers have identified numerous genes and biological mechanisms related to antisocial behavior in the human population. Genetic analyses have implicated the *MAOA* (Manucka, Flory, Ferrell, Mann, & Muldoon, 2000), *SLC6A4* (Murphy et al., 2008), *TPH1* (Hennig, Reuter, Netter, Burk, & Landt, 2005), 5-HT1B hetero-receptors (Soyka, Preuss, Koller, Zill, & Bondy, 2004); Dopamine-β-hydroxylase (DβH; Hess et al., 2009); and gamma-aminobutyric acid neurotransmitters (Miczek, Fish, De Bold, & De Almeida, 2002), among many others, in a predisposition toward aggression, delinquency, and violent behavior in human populations (for a review see Craig & Halton, 2009). Possible biological mechanisms include cortisol levels that monitor the hypothalamus–pituitary– adrenal axis (Shirtcliff, Granger, Booth, & Johnson, 2005); levels of the serotonin metabolite 5-hydroxy-indole acetic acid (5-HIAA) in cerebrospinal fluid (Coccaro et al., 1997); and potentially serotonin mechanisms, insulin levels, and glucose metabolism (Linnoila & Virkkunen, 1992).

Studying model organisms can help identify genes for antisocial behavior in humans. Humans and nonhuman animals share neurochemical and anatomical systems that are activated when aggressive behavior occurs (Nelson & Trainor, 2007). Rodents are among the ideal animals that can be studied to provide new knowledge about the genetics of aggression. Approximately 90% of genes in rats are orthologous to genes in humans (Rat Genome Sequencing Project Consortium, 2004). In addition, the phenotype of rodents can be measured more precisely, and the genetic background and environmental conditions can be controlled more easily. Anholt and Mackay (2012) reported that researchers can successfully identify genes and pathways that influence aggression by using quantitative trait locus mapping and analysis of single-gene mutations in mice. In our analysis, 39 genes are known to be related to aggression in mice (see Online Appendix Table A1 on the *Journal of Marriage and Family* website; http://onlinelibrary.wiley.com/journal/10.1111/(ISSN)1741-3737).

## Selection, Confounding, and Population Heterogeneity

Marriage is not a random event. Issues such as selection, confounding, and population heterogeneity may pose threats to the marriage–antisocial behavior association, thereby undermining the validity of the Gene × Marriage interaction results. Differential selection is one of the largest threats to claiming a causal effect of marriage on desistance (e.g., King et al., 2007; Sampson et al., 2006). Suppose that delinquent persons self-select out of marriage, either by remaining single or being more likely to divorce. If this were the case, then it is not marriage that makes individuals less antisocial, and the observation that genetic effects for delinquency depend on marital status possibly reflects only the difference in genetic effects between delinquent and nondelinquent persons.

Age may have a confounding effect on the inhibiting effect of marriage. Delinquency usually peaks during adolescence and young adulthood and declines dramatically thereafter (Hirschi & Gottfredson, 1983). In other words, along with a decline in antisocial behavior most people experience major changes in life circumstances, such as marriage. Thus, perhaps the reason married individuals are less likely to engage in antisocial behavior is that older individuals are more likely to get married and less likely to act antisocially. In this scenario, the interaction effect of marriage could merely represent the effect of age or maturity.

A third issue involved in the desistance process is that the effect of marriage may not be universal for every individual because of *population heterogeneity*, a term that refers to individuals' differing propensity to engage in deviant behavior (DeLisi, 2005; Nagin & Paternoster, 2000). Moffitt (1993) argued that one group of individuals repeatedly engages in deviant behavior over the life course (*persistent offenders*), whereas the remaining individuals act delinquently primarily during adolescence. Persistent offenders do not practice much prosocial behavior during early childhood. As a result, marriage may not have as much impact on persistent offenders as it does on others. The Gene × Marriage interaction therefore may vary in magnitude for persistent and nonpersistent offenders. In this study, we conducted analyses to examine whether the effect of marriage is threatened by the three issues just mentioned.

## Research Question and Hypothesis

As discussed above, marriage may foster desistance by strengthening bonds within the family, weakening antisocial ties outside of the family, and altering one's psychological perception of deviant behavior. Taking genetic influences into account, in this study we further explored the role of marriage in the desistance process. We examined whether marriage can inhibit delinquency and violence through a biological pathway: the modification of a large number of genetic effects for antisocial behavior. Given that the existing literature suggests that marriage has an inhibiting effect on antisocial behavior, we hypothesized that the collective influence of the genes on antisocial behavior is smaller among married individuals than that of unmarried individuals.

# Method

## Data

Our analysis used data from the genetic subsample of Add Health. Add Health is a nationally representative sample of U.S. adolescents in Grades 7–12 in 1994–1995 (Harris et al., 2009). The first wave of data collection took place in the 1994–1995 school year. A sample of about 20,000 adolescents was drawn. Respondents were surveyed through in-school questionnaires and in-home interviews. Three subsequent waves of data were collected at respondents' homes in 1996 (Wave II), 2001–2002 (Wave III), and 2008–2009 (Wave IV). A wide range of data, including information about social background and behaviors, was collected at each wave. In addition, the data have rich information on parents and romantic partners.

The genetic subsample consisted of 2,612 respondents identified as siblings or twins at Wave I. At Wave III saliva of the genetic subsample was collected and genotyped. DNA was isolated from buccal cells at the Institute of Behavior Genetics at the University of Colorado at Boulder. The average yield of DNA was 58 ± 1 μg. The genotype data were based on an Illumina GoldenGate assay. The GoldenGate array targeted 1,536 SNPs. A total of 1,140 SNPs in 130 genes were successfully genotyped. The number of respondents whose DNA was successfully genotyped was 2,281. The 2,281 respondents came from 1,428 families. Of the 1,428 families, 770 included two children both of whom had genotype data, 33 included three children all of whom had genotype data, and two families included four children all of whom had genotype data. There were 623 families in which only one child had genotype data, although this child had sibling(s) or a twin. We selected 580 SNPs in 64 autosomal genes for the current analysis. Of the 64 genes, 39 genes reviewed and summarized by Maxson (2009) are associated with aggression in transgenic or knock-out studies of mice, and 25 genes are related to risky behavior such as drinking and drug use in the human population (see Online Appendix Table A1 for information on the 64 genes).

## Measures

**Delinquency and violence—**A four-item nonviolence scale and an eight-item violence scale were used to measure delinquency and violence, respectively. *Nonviolent delinquency* included stealing amounts larger or smaller than $50, breaking and entering, and selling drugs within the past 12 months. *Violence* included serious physical fighting result in the need for medical treatment, use of weapons to get something from someone, physical fighting between groups, shooting or stabbing someone, deliberately damaging property, carrying a weapon (unavailable at Wave IV), and pulling a knife or gun on someone within the past 12 months (see the Online Supplementary Appendix for details about the coding of delinquency and violence). The sum of delinquency and violence was treated as the third dependent variable. The two scales are a variation of a scale that is widely used in research on delinquency and crime (Thornberry & Krohn, 2000).

**Desistance—***Desistance* can be defined either as a process or an end state (Laub & Sampson, 2001). Treating desistance as a process requires more frequent assessments of the behavior, whereas treating desistance as an end state requires a longer time frame (Mulvey

et al., 2004). Similar to Glueck and Glueck's (1950) study, in which participants were interviewed at an average age of 14, 25, and 32, Add Health collected information from participants at an average age of 15, 22, and 28. Given the relatively frequent assessments from adolescence to young adulthood among Add Health participants, we were able to study desistance as a process.

In accordance with the majority of research (e.g., Horney et al., 1995; Laub et al., 1998; Theobald & Farrington, 2009; Warr, 1998), we assumed that only individuals who were delinquent in the first place were eligible for desisting from delinquency and violence. Respondents who scored at least 1 on either the delinquency or violence scale at Waves I and II were included in our sample. The final sample consisted of 1,254 individuals.

Reports from the Bureau of Justice Statistics (2001–2009) show that the percentage of the U.S. adult population under age 35 who was or had ever been incarcerated in a state or federal prison or in a local jail ranged from about 1% to 4%, and this percentage was remarkably higher among the 20-and-older age group than the 18- to 19-year-old group. As a result, chronic offenders, especially those who were 20 and older, may have been more likely to drop out of the study. In Add Health, about 12 individuals from the genetic sample were not interviewed at Wave III because they were incarcerated at the time. Therefore, conclusions based on the sample may not necessarily apply to the correctional population.

**Marriage and Its Temporal Relation With Delinquency and Violence—**To isolate the effect of marriage it is crucial to sort out the temporal order between marriage and delinquency and violence. At Waves III and IV, respondents were asked to report the number of times they had been married and the start and end dates of each marriage, but we knew of delinquent and violent behaviors that had occurred only in the 12 months before the interview. No exact timing of the behaviors within this 12-month window was available. Figure 1 is an illustration of how marital status was defined. We divided marriages into two groups based on whether the marriage had ended before the 12-month window. The first group of marriages (Types A–D, represented by the white lines in Figure 1) overlapped the 12-month window. We assumed that these marriages could influence delinquency and violence that occurred during this time frame. The other group of marriages (Type E, represented by the black line in the figure) were those that ended at least 12 months prior to the interview. We assumed that these marriages could not influence delinquency and violence during this time frame.

Most of the studies that reported the inhibiting effect of marriage used data in which respondents married in the 1950s, 1960s, or 1980s (e.g., Farrington & West, 1995; King et al., 2007; Laub & Sampson, 2003). An advantage of the Add Health data is that we could test whether the marriage effect extended to a more recent cohort.

**Marriage and Cohabitation—**Mechanisms for antisocial behavior may be different between cohabitors and married persons (Horney et al., 1995); therefore, we first compared the levels of delinquent and violent behaviors in married, cohabitating, and single individuals. The results (not shown) suggested that cohabitors and single persons tended to report higher levels of antisocial behavior than married persons. Thus, married individuals

were coded as 1 and unmarried individuals—namely, cohabitating and single persons—were coded as 0.

**Control Variables—**Control variables included age, gender, race, education, employment, churchgoing frequency, household size, verbal IQ (Picture Vocabulary Test; Add Health used a computerized, abridged version of Peabody Picture Vocabulary Test—Revised (Dunn & Dunn, 1981) score, parental education, closeness to parents, and bio-ancestry scores. Parental education was a family-level variable, and the remaining controls were individual-level variables. Missing values in the control variables were imputed by the multiple-imputation technique (Rubin, 1987). The missing values were imputed five times to generate five complete data sets, and then the regression results using the five complete data sets were combined to produce inferential results. We did not impute missing values in the delinquency, violence, or marriage variables. The estimation of bio-ancestry scores (Pritchard, Stephens, & Donnelly, 2000) relied on 121 ancestral informative markers that were used to distinguish three major continental populations: (a) African, (b) East Asian, and (c) European. Each respondent was assigned three scores (i.e., African, East Asian, and European). The sum of the three scores was 1. Because using bio-ancestry scores to adjust for population stratification is a recommended method in genetic analysis (McCarthy et al., 2008), we controlled for bio-ancestry scores in the mixed linear models. Replacing bio-ancestry scores with self-reported race yielded similar results because bio-ancestry scores were highly correlated with self-reported race (e.g., the average European bio-ancestry score for White was .95). Descriptive statistics and brief descriptions of the variables are presented in Table 1.

## Analytical Strategy

**Assessing the effect of marriage on delinquency and violence—**First, we examined the effect of marriage on antisocial behavior in generalized estimating equation (GEE; Liang & Zeger, 1986). Equation 1 describes the structure of the model:

$$Y_{ijb}=\beta_0+\beta_1 X_{ijb}+\beta_2 Z_{ijb}+\beta_3 Z'_{ijb}+\beta_4 Z''_{ja}, \quad (1)$$

where $Y_{ijb}$ is the delinquent or violent behavior for individual *i* in family *j* at Wave *b*, that is, Wave III or IV; $X_{ijb}$ is the marital status; $Z_{ijb}$ represents the control variables including age, age squared, gender, race, education, employment status, churchgoing frequency, and household size; $Z'_{ija}$ represents Picture Vocabulary Test score and closeness to parents measured at Wave *a*, that is, Wave I; and $Z''_{ja}$ represents the family-level variable, parental education. An exchangeable working correlation structure was specified to address the within-person and within-family correlations.

**Modeling the Gene × Marriage interaction—**To model the interaction between 580 SNPs and marriage, we extended the mixed linear model implemented in the GCTA software (Yang et al., 2011). This model estimates the proportion of phenotypic variance that is accounted for by the linear, additive effects of the SNPs. Equation 2 describes the basic structure of the mixed linear model:

$$Y=X\boldsymbol{\beta}+W\mu+\varepsilon, \quad (2)$$

where $Y$ is delinquency or violence; $\boldsymbol{\beta}$ is a vector of fixed effects for the control variables; $\mu$ is a vector of SNP effects with $\mu_i \sim N(0, \boldsymbol{\sigma}^2_{\boldsymbol{\mu}})$ where $i = 1,\ldots, N$, with $N$ being the number of SNPs; $\varepsilon$ is a vector of residual effects with $\varepsilon_j \sim N(0, \boldsymbol{\sigma}^2_{\boldsymbol{\varepsilon}}$ where $j = 1,\ldots, n$, with $n$ being the number of individuals in the sample; and $W$ is a standardized genotype matrix with the $ij$ th element $w_{ij}=(s_{ij} - 2p_i/\sqrt{[2p_i(1-p_i]},$ where $s_{ij}$ is the number of copies of the reference allele for the $i$ th SNP of the $j$ th individual and $p_i$ is the frequency of the reference allele. SNPs were coded as minor allele dosage (0, 1, 2).

Next, by defining $g = W\mu$, $\mathbf{A} = WW'/N$ and $\sigma^2_g=N\sigma^2_\mu$, Equation 2 is mathematically equivalent to Equation 3, which can be estimated by the restricted maximum likelihood approach:

$$Y=X\beta+g+\varepsilon, \text{with} \ \ \text{Variance}=\mathbf{A}\sigma^2_g+I_\varepsilon\sigma^2_\varepsilon, \quad (3)$$

where $g$ is an $n \times 1$ vector of the total genetic effects of the individuals with $g \sim N(0, \mathbf{A}\,\sigma^2_g$ $\mathbf{A}$ is the genetic relationship matrix between individuals and $\boldsymbol{\sigma}^2_{\mathbf{g}}=N \times \boldsymbol{\sigma}^2_{\boldsymbol{\mu}}$ is the total genetic variance explained by the SNPs. Hence,), $\sigma^2_g$ can be estimated by the restricted maximum likelihood approach, depending on the genetic relationship matrix estimated from the SNPs. We provide a nontechnical and intuitive description of the mixed linear model in the Supplementary Online Appendix.

We assessed the Gene × Marriage interaction by comparing the proportion of variance explained—$(\sigma^2_g/(\sigma^2_g+\sigma^2_\varepsilon$ in Equation 3—between married and unmarried individuals. This form of G×E interaction is different from the traditional form of G×E in which a multiplicative interaction term is added in a regression. Conceptually, both of the two forms of G×E examine the processes by which the effects of genes are conditioned by environmental factors or vice versa. In the traditional form, when modeling the interaction between marriage and 580 SNPs, it is most likely that one needs to either put 580 two-way interactions into a regression or run 580 regressions with each regression containing one two-way interaction. In our approach, 580 SNPs are simultaneously considered as random effects.

The proportion of variance explained was estimated for antisocial behavior at Waves III and IV separately; specifically, we took the following steps to obtain the proportion of variance explained. First, the sample was divided into two groups: the married and the unmarried. Second, we performed subsample selection. Given that the sample consisted of siblings and twins, if related persons were included in the same mixed linear model the estimate of genetic effects would be biased by phenotypic correlations of, for example, siblings who shared common environments. Therefore, we randomly selected an individual from every family. We did this separately for the married and the unmarried groups. Next, we repeated

the subsample selection process 1,000 times to avoid the arbitrariness of which person in the family was selected. Finally, the mixed linear models were estimated for the married and unmarried groups separately, and results were averaged over results obtained from 1,000 analytical subsamples. We conducted Kolmogorov–Smirnov tests to compare the distribution of 1,000 proportions of variance explained between the married and the unmarried groups.

**Gene–environment correlations—***Gene–environment correlations* (rGEs) refer to situations in which genotypes are nonrandomly associated with environments. rGEs may bias estimates of G×E interactions (Wagner, Li, Liu, & Guo, 2013). Using the mixed linear model, we tested whether the 580 SNPs were associated with marital status. The association was not significantly different from 0 ($p = .96$). The evidence suggests that the rGE did not confound the G×E interaction results in this study.

## Results

### Marriage and Antisocial Behavior

Data on the effect of marriage on delinquency and violence at Waves III and IV estimated in GEE models (Equation 1) are listed in Table 2. Married individuals showed a significant decrease on the delinquency and violence scales of 0.17 and a decrease of 0.34 on the sum of the two scales. This suggests that getting married would decrease the likelihood of behaving antisocially.

### The Gene× Marriage Interaction

The results given in Table 3 were obtained from the mixed linear models implemented in the GCTA software. Presented first in the table is the percentage of variance in antisocial behavior explained by the 580 SNPs. Overall, the percentage of variance explained was significantly smaller in married individuals than in unmarried individuals, suggesting that marriage may suppress the collective influence of the genes. Our hypothesis was supported. At Wave III, the SNPs jointly accounted for about 1.09%, 3.56%, and 1.48% of the variance in delinquency, violence, and the sum of delinquency and violence, respectively, in unmarried individuals, whereas the SNPs explained virtually no variance in the married individuals. Similarly, at Wave IV the SNPs accounted for 0.26% and 0.14% of the variance in violence and the sum of delinquency and violence among the unmarried individuals and virtually none among the married individuals. Variance explained by the SNPs can be seen as an estimator for heritability. We did not report results for delinquency at Wave IV because its distribution was highly skewed to the right.

### Selection, the Confounding Effect of Age, and Population Heterogeneity

As mentioned previously, selection, age, and population heterogeneity may threaten the validity of the Gene × Marriage interaction findings. In regard to selection, we tested whether delinquent persons were less likely to get married. If earlier antisocial behavior at Waves I and II (1994–1995 and 1996) were not a significant predictor of marital status at Waves III and IV (2001–2002 and 2008–2009), this suggests that selection based on antisocial behavior may not pose a serious threat to the deterrent capacity of marriage. The

results are reported in Table 4. The dependent variable, marital status, was a dichotomous variable, with 1 indicating that a person is married and 0 otherwise. Logistic GEE models were used and the within-family correlations were addressed. None of the coefficients for delinquency, violence, and the sum of delinquency and violence at Wave I or II were statistically significant. In other words, the evidence suggests that the probability of getting married was not associated with the levels of earlier antisocial behavior.

To address the potential confounding effect of age, we first randomly excluded a subset of young unmarried individuals to generate a new sample so that the mean ages for married and unmarried individuals were the same. In our sample, the mean ages for married and unmarried individuals were 26 and 24, respectively. In the new sample the mean ages for the two groups were both 26. We called this new sample the *age-comparable sample*. A similar method has been used to equalize age in two groups in previous studies (e.g., Uggen, 2000). Next, using this age-comparable sample, we conducted analyses to examine whether marriage may suppress antisocial behavior in Equation 1 and whether marriage interacted with the genes in the mixed linear models. In the left panel of Table 5 are reported the results obtained from GEE models using the age-comparable sample. The marriage effect remained. Married individuals scored 0.23 less on delinquency, 0.20 less on violence, and 0.43 on the sum of the two than unmarried individuals of comparable age. We also reestimated the mixed linear models in Table 3 using the age-comparable sample. The reestimation yielded similar results (not shown) to those presented in Table 3. Therefore, we are more confident in saying that age did not confound the marriage–antisocial behavior association and the Gene × Marriage interaction results.

In the right panel of Table 5, we explore the possibility that the deterrent capacity of marriage differed between persistent and nonpersistent offenders due to population heterogeneity. In our data, 55 respondents scored 1 or more on the violent behavior scale at all four waves; these 55 respondents were considered persistent offenders. The remaining individuals were coded as nonpersistent offenders. We added a dummy variable for the persistent offenders and an interaction between marriage and the dummy variable in GEE models. As expected, persistent offenders exhibited higher levels of delinquency and violence. However, the interaction between marriage and persistent offender was not statistically significant. Therefore, no evidence supports the idea that marriage functioned differently for persistent and nonpersistent offenders. In addition, excluding the 55 persistent offenders did not change results in Tables 2 or 3.

## Discussion

In this study we investigated whether marriage moderates the effects of 580 SNPs in 64 genes that are related to aggression and risky behavior on antisocial behavior. The main findings showed that the SNPs explained much less variance in delinquency and violence among married individuals than unmarried individuals, implying that marriage may suppress the collective genetic influence. Past inquiries about the effect of marriage on antisocial behavior have primarily focused on the social, behavioral, and psychological aspects. The integration of genetics enriches the theoretical frameworks. We found that marriage could

work through a biological pathway—the modification of genetic effects—to deter delinquency and violence.

Moreover, evidence supports the inference that marriage caused declines in antisocial behavior and therefore supports the validity of the Gene × Marriage interaction results. Selection, age, and population heterogeneity do not seem to pose serious threats. Our results are consistent with those of previous research that addressed causality in the marriage–crime nexus. This research showed that marriage may causally inhibit crime and deviant behavior using policy changes as natural experiments (Cáceres-Delpiano & Giolito, 2008; Edlund, Yi, Li, & Zhang, 2013), using statistical techniques such as propensity score matching and inverse probability of treatment weighting (King et al., 2007; Sampson et al., 2006; Theobald & Farrington, 2009), and taking advantage of a co-twin control design (Burt et al., 2010). With respect to population heterogeneity, we found that marriage did not influence persistent and nonpersistent offenders differently. This pattern that emerged from a sample of the general U.S. population is consistent with Blokland and Nieuwbeerta's (2005) findings, which were based on a sample of the general Dutch population and indicated that the effect of marriage was the same for sporadic and low-rate offenders. It is interesting that, in the same study, using data from a sample of Dutch criminal offenders, Blokland and Nieuwbeerta found that the inhibiting effect of marriage existed only among low- and moderate-rate offenders, but not among high-rate offenders. Future work might examine why the marriage effect varies in different populations.

The Gene × Marriage interaction findings bear implications for researchers. High estimates of heritability for antisocial behavior from behavioral genetic studies (Rhee & Waldman, 2002) may make it look as though environmental influences are not as important as genetic influences. Our findings point to the opposite: The effect of genes was conditional on the environment. Individuals possess different forms of genes related to antisocial behavior. Some individuals are more genetically susceptible to delinquency and violence than others. Regardless of the genotype, the collective influence of the genes is subject to the presence of marriage, possibly because marriage can affect many aspects of an individual's life. Emotional attachment to one's spouse, time devoted to the family, and normalized activity after marriage might all play a role in curbing the manifestation of the genes. Future research might investigate what aspects of marriage interact with genetic factors to deter antisocial behavior. In addition, our results suggest that sources of variation in delinquency and violence comprise more than the 64 genes and marriage. Other genes, epistasis, epigenetics, and gene expression may be associated with committing deviant behavior. Also, life events such as employment are worth investigation because they are turning points for desistance across the life course (Laub & Sampson, 1993). More focused analyses of the roles of other biological pathways and life events would offer additional insights into the desistance process.

Several limitations should be acknowledged. We were unable to estimate the effects of genetic variants that were not covered by the SNP arrays. In addition, the 580 SNPs and causal alleles for delinquency and violence may not be in complete linkage disequilibrium. Therefore, the collective influence of the SNPs was likely underestimated. Also, this particular mixed linear model framework does not allow for analysis of genetically related

individuals, resulting in a reduction in sample size. Because of this, our ability to investigate the roles of other factors in the desistance process was limited. For example, prior research suggests that gender contingencies are relevant to the marriage effect, and men tend to benefit more from the inhibiting influence of marriage (Giordano et al., 2002; King et al., 2007; Sampson et al., 2006). Future G×E research might consider using a larger sample to examine the roles of gender, race, and other factors.

Social scientists interested in G×E interaction are faced with two challenging tasks. The first is to identify "truly exogenous, causal environmental effects" (Conley, 2009, p. 244). The second is to creatively use a variety of methods to detect G×E interactions (Shanahan & Boardman, 2009). This article is just one example of how researchers may undertake these two tasks. SNP data are now increasingly available in many large-scale social surveys. Rich data sets offer opportunities for future G×E research to use different study designs and methods to gain a more comprehensive understanding of complex traits and behaviors.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

## Acknowledgments

This research uses data from Add Health, a program project directed by Kathleen Mullan Harris and designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill, and funded by Grant P01-HD31921 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development, with cooperative funding from 23 other federal agencies and foundations. Special acknowledgment is due Ronald R. Rindfuss and Barbara Entwisle for assistance in the original design. Information on how to obtain the Add Health data files is available on the Add Health website (http://www.cpc.unc.edu/addhealth). No direct support was received from Grant P01-HD31921 for this analysis.

A National Science Foundation grant to Guang Guo supported the genotyping of the aggression-related genes in the genetic sample of Add Health (National Science Foundation's Human and Social Dynamics program BCS-0826913). A Challenge Grant (RC1 DA029425-01) to Guang Guo also provided support for the study. Special acknowledgment is due Kirk Wilhelmsen of the Genetics Department, Patricia Basta of the Bio-Specimen Process Center, Jason Luo of the Mammalian Genotyping Center, and the Odum Institute at the University of North Carolina at Chapel Hill. We received important assistance in single nucleotide polymorphism selection from David Goldman and his Neurogenetics laboratory at the National Institute on Alcohol Abuse and Alcoholism. We thank Ted Mouw, Francois Nielsen, Michael Shanahan, Yang Yang, and Ashton Verdery for their helpful comments on earlier drafts of this article.

## References

Anholt RRH, Mackay TFC. Genetics of aggression. Annual Review of Genetics. 2012; 46:145–164.10.1146/annurev-genet-110711-155514

Beaver KM, Wright JP, DeLisi M, Vaughn MG. Desistance from delinquency: The marriage effect revisited and extended. Social Science Research. 2008; 37:736–752.10.1016/j.ssresearch.2007.11.003 [PubMed: 19086113]

Blokland AAJ, Nieuwbeerta P. The effects of life circumstances on longitudinal trajectories of offending. Criminology. 2005; 43:1203–1240.10.1111/j.1745-9125.2005.00037.x

Bureau of Justice Statistics. Prison inmates at midyears 2001–09. 2001–2009. Retrieved from http://www.bjs.gov/pubseries.cfm

Burt SA, Donnellan MB, Mikhila N, Humbad M, Hicks BM, McGue M, Iacono WG. Does marriage inhibit antisocial behavior?: An examination of selection vs causation via a longitudinal twin design. Archives of General Psychiatry. 2010; 67:1309–1315.10.1001/archgenpsychiatry.2010.159 [PubMed: 21135331]

Cáceres-Delpiano, J.; Giolito, E. The impact of unilateral divorce on crime. IZA Discussion Paper No. 3380. Institute for the Study of Labor; Bonn, Germany: 2008.

Caspi A, McClay J, Moffitt TE, Mill J, Martin J, Craig IW, Poulton R. Role of genotype in the cycle of violence in maltreated children. Science. 2002 Aug 2.297:851–854.10.1126/science.1072290 [PubMed: 12161658]

Coccaro EF, Kavoussi RJ, Trestman RL, Gabriel SM, Cooper TB, Siever LJ. Serotonin function in human subjects: Intercorrelations among central 5-HT indices and aggressiveness. Psychiatry Research. 1997; 73:1–14.10.1016/S0165-1781(97)00108-X [PubMed: 9463834]

Conley D. The promise and challenges of incorporating genetic data into longitudinal social science surveys and research. Biodemography and Social Biology. 2009; 55:238–251.10.1080/19485560903415807 [PubMed: 20183907]

Craig IW, Halton KE. Genetics of human aggressive behaviour. Human Genetics. 2009; 126:101–113.10.1007/s00439-009-0695-9 [PubMed: 19506905]

DeLisi, M. Career criminals in society. Thousand Oaks, CA: Sage; 2005.

Dunn, LM.; Dunn, LM. Peabody Picture Vocabulary Test—Revised. Circle Pines, MN: American Guidance Service; 1981.

Edlund L, Yi J, Li H, Zhang J. Sex ratios and crime: Evidence from China. The Review of Economics and Statistics. 2013; 95:1520–1534.10.1162/REST_a_00356

Farrington, DP.; West, DJ. Effects of marriage, separation, and children on offending by adult males. In: Hagan, J., editor. Current perspectives on aging and the life cycle: Vol. 4. Delinquency and disrepute in the life course. Greenwich, CT: JAI Press; 1995. p. 249-281.

Giordano PC, Cernkovich SA, Rudolph JL. Gender, crime, and desistance: Toward a theory of cognitive transformation. American Journal of Sociology. 2002; 107:990–1064.10.1086/343191

Giordano PC, Schroeder RD, Cernkovich SA. Emotions and crime over the life course: A neo-Meadian perspective on criminal continuity and change. American Journal of Sociology. 2007; 112:1603–1661.10.1086/512710

Glueck, S.; Glueck, E. Unraveling juvenile delinquency. New York: Commonwealth Fund; 1950.

Guo G, Roettger ME, Cai T. The integration of genetic propensities into social-control models of delinquency and violence among male youths. American Sociological Review. 2008; 73:543–568.10.1177/000312240807300402

Harris, KM.; Halpern, CT.; Whitsel, E.; Hussey, J.; Tabor, J.; Entzel, P.; Udry, JR. The National Longitudinal Study of Adolescent to Adult Health: Research design. 2009. Retrieved from http://www.cpc.unc.edu/projects/addhealth/design

Hennig J, Reuter M, Netter P, Burk C, Landt O. Two types of aggression are differentially related to serotonergic activity and the *A779C TPH* polymorphism. Behavioral Neuroscience. 2005; 119:16–25.10.1037/0735-7044.119.1.16 [PubMed: 15727508]

Hess C, Reif A, Strobel A, Boreatti-Hümmer A, Heine M, Lesch KP, Jacob CP. A functional dopamine-β-hydroxylase gene promoter polymorphism is associated with impulsive personality styles, but not with affective disorders. Journal of Neural Transmission. 2009; 116:121–130.10.1007/s00702-008-0138-0 [PubMed: 18982239]

Hirschi, T. Causes of delinquency. Berkeley, CA: Free Press; 1969.

Hirschi T, Gottfredson M. Age and the explanation of crime. American Journal of Sociology. 1983; 89:552–584.

Horney J, Osgood DW, Marshall IH. Criminal careers in the short-term: Intra-individual variability in crime and its relation to local life circumstances. American Sociological Review. 1995; 60:655–673.

King RD, Massoglia M, Macmillan R. The context of marriage and crime: Gender, the propensity to marry, and offending in early adulthood. Criminology. 2007; 45:33–65.10.1111/j.1745-9125.2007.00071.x

Laub JH, Nagin DS, Sampson RJ. Trajectories of change in criminal offending: Good marriages and the desistance process. American Sociological Review. 1998; 63:225–238.

Laub JH, Sampson RJ. Turning points in the life course: Why change matters to the study of crime. Criminology. 1993; 31:301–325.10.1111/j.1745-9125.1993.tb01132.x

Laub, JH.; Sampson, RJ. Understanding desistance from crime. In: Tonry, M., editor. Crime and justice: A review of research. Vol. 28. Chicago: University of Chicago Press; 2001. p. 1-69.

Laub, JH.; Sampson, RJ. Shared beginnings, divergent lives: Delinquents boys to age 70. Cambridge, MA: Harvard University Press; 2003.

Liang KY, Zeger SL. Longitudinal data analysis using generalized linear models. Biometrika. 1986; 73:13–22.

Linnoila VM, Virkkunen M. Aggression, suicidality, and serotonin. Journal of Clinical Psychiatry. 1992; 53(Suppl 10):46–51. [PubMed: 1385390]

Manucka SB, Flory JD, Ferrell RE, Mann JJ, Muldoon MF. A regulatory polymorphism of the monoamine oxidase-A gene may be associated with variability in aggression, impulsivity, and central nervous system serotonergic responsivity. Psychiatry Research. 2000; 95:9–23.10.1016/S0165-1781(00)00162-1 [PubMed: 10904119]

Maxson, SC. The genetics of offensive aggression in mice. In: Kim, YK., editor. Handbook of behavior genetics. New York: Springer; 2009. p. 301-316.

McCarthy MI, Abecasis GR, Cardon LR, Goldstein DB, Little J, Ioannidis JPA, Hirschhorn JN. Genome-wide association studies for complex traits: Consensus, uncertainty and challenges. Nature Reviews Genetics. 2008; 9:356–369.10.1038/nrg2344

Miczek KA, Fish EW, De Bold JF, De Almeida RM. Social and neural determinants of aggressive behavior: Pharmacotherapeutic targets at serotonin, dopamine and γ-aminobutyric acid systems. Psychopharmacology. 2002; 163:434–458.10.1007/s00213-002-1139-6 [PubMed: 12373445]

Moffitt TE. Adolescence-limited and life-course–persistent antisocial behavior: A developmental taxonomy. Psychological Review. 1993; 100:674–701. [PubMed: 8255953]

Mulvey EP, Steinberg L, Fagan J, Cauffman E, Piquero AR, Chassin L, Losoya SH. Theory and research on desistance from antisocial activity among serious adolescent offenders. Youth Violence and Juvenile Justice. 2004; 2:213–236.10.1177/1541204004265864 [PubMed: 20119505]

Murphy DL, Fox MA, Timpano KR, Moya PR, Ren-Patterson R, Andrews AM, Wendland JR. How the serotonin story is being rewritten by new gene-based discoveries principally related to *SLC6A4*, the serotonin transporter gene, which functions to influence all cellular serotonin systems. Neuropharmacology. 2008; 55:932–960. [PubMed: 18824000]

Nagin D, Paternoster R. Population heterogeneity and state dependence: State of the evidence and directions for future research. Journal of Quantitative Criminology. 2000; 16:117–144.10.1023/A:1007502804941

Nelson RJ, Trainor BC. Neural mechanisms of aggression. Nature Reviews Neuroscience. 2007; 8:536–546.10.1038/nrn2174 [PubMed: 17585306]

Osgood DW, Lee H. Leisure activities, age, and adult roles across the lifespan. Society and Leisure. 1993; 16:181–208.

Osgood DW, Wilson JK, O'Malley PM, Bachman JG, Johnston LD. Routine activities and individual deviant behavior. American Sociological Review. 1996; 61:635–655.

Pritchard JK, Stephens M, Donnelly P. Inference of population structure using multilocus genotype data. Genetics. 2000; 155:945–959. [PubMed: 10835412]

Rat Genome Sequencing Project Consortium. Genome sequence of the Brown Norway rat yields insights into mammalian evolution. Nature. 2004 Apr 1.428:493–521.10.1038/nature02426 [PubMed: 15057822]

Rhee SH, Waldman ID. Genetic and environmental influences on antisocial behavior: A meta-analysis of twin and adoption studies. Psychological Bulletin. 2002; 128:490–529.10.1037/0033-2909.128.3.490 [PubMed: 12002699]

Rubin, DB. Multiple imputation for nonresponse in surveys. New York: Wiley; 1987.

Sampson, RJ.; Laub, JH. Crime in the making: Pathways and turning points through life. Cambridge, MA: Harvard University Press; 1993.

Sampson RJ, Laub JH, Wimer C. Does marriage reduce crime? A counterfactual approach to within-individual causal effects. Criminology. 2006; 44:465–508.10.1111/j.1745-9125.2006.00055.x

Shanahan, MJ.; Boardman, JD. Genetics and behavior in the life course: A promising frontier. In: Elder, GH.; Giele, JZ., editors. The craft of life course research. New York: Guilford Press; 2009. p. 215-235.

Shirtcliff EA, Granger DA, Booth A, Johnson D. Low salivary cortisol levels and externalizing behavior problems in youth. Development and Psychopathology. 2005; 1:167–184.10.1017/S0954579405050091 [PubMed: 15971765]

Simons RL, Lei MK, Beach SRH, Brody GH, Philibert RA, Gibbons FX. Social environment, genes, and aggression: Evidence supporting the differential susceptibility perspective. American Sociological Review. 2011; 76:883–912.10.1177/0003122411427580

Soyka M, Preuss UW, Koller G, Zill P, Bondy B. Association of *5-HT1B* receptor gene and antisocial behavior in alcoholism. Journal of Neural Transmission. 2004; 111:101–109.10.1007/s00702-003-0064-0 [PubMed: 14714219]

Theobald D, Farrington DP. Effects of getting married on offending: Results from a prospective longitudinal survey of males. European Journal of Criminology. 2009; 6:496–516.10.1177/1477370809341226

Thornberry, TP.; Krohn, MD. The self-report method for measuring delinquency and crime. In: Duffee, D.; Crutchfield, RD.; Mastrofski, S.; Mazerolle, L.; McDowall, D.; Ostrom, B., editors. Criminal justice 2000: Innovations in measurement and analysis. Washington DC: National Institute of Justice; 2000. p. 33-83.

Uggen C. Work as a turning point in the life course of criminals: A duration model of age, employment, and recidivism. American Sociological Review. 2000; 65:529–546.

Wagner B, Li J, Liu H, Guo G. Gene–environment correlation: Difficulties and a natural experiment–based strategy. American Journal of Public Health. 2013; 103(Suppl 1):S167–S173.10.2105/AJPH.2013.301415 [PubMed: 23927502]

Waite LJ. Does marriage matter? Demography. 1995; 32:483–507. [PubMed: 8925942]

Warr M. Life-course transitions and desistance from crime. Criminology. 1998; 36:183–216.

Yang J, Lee SH, Goddard ME, Visscher PM. GCTA: A Tool for genome-wide complex trait analysis. American Journal of Human Genetics. 2011; 88:76–82.10.1016/j.ajhg.2010.11.011 [PubMed: 21167468]

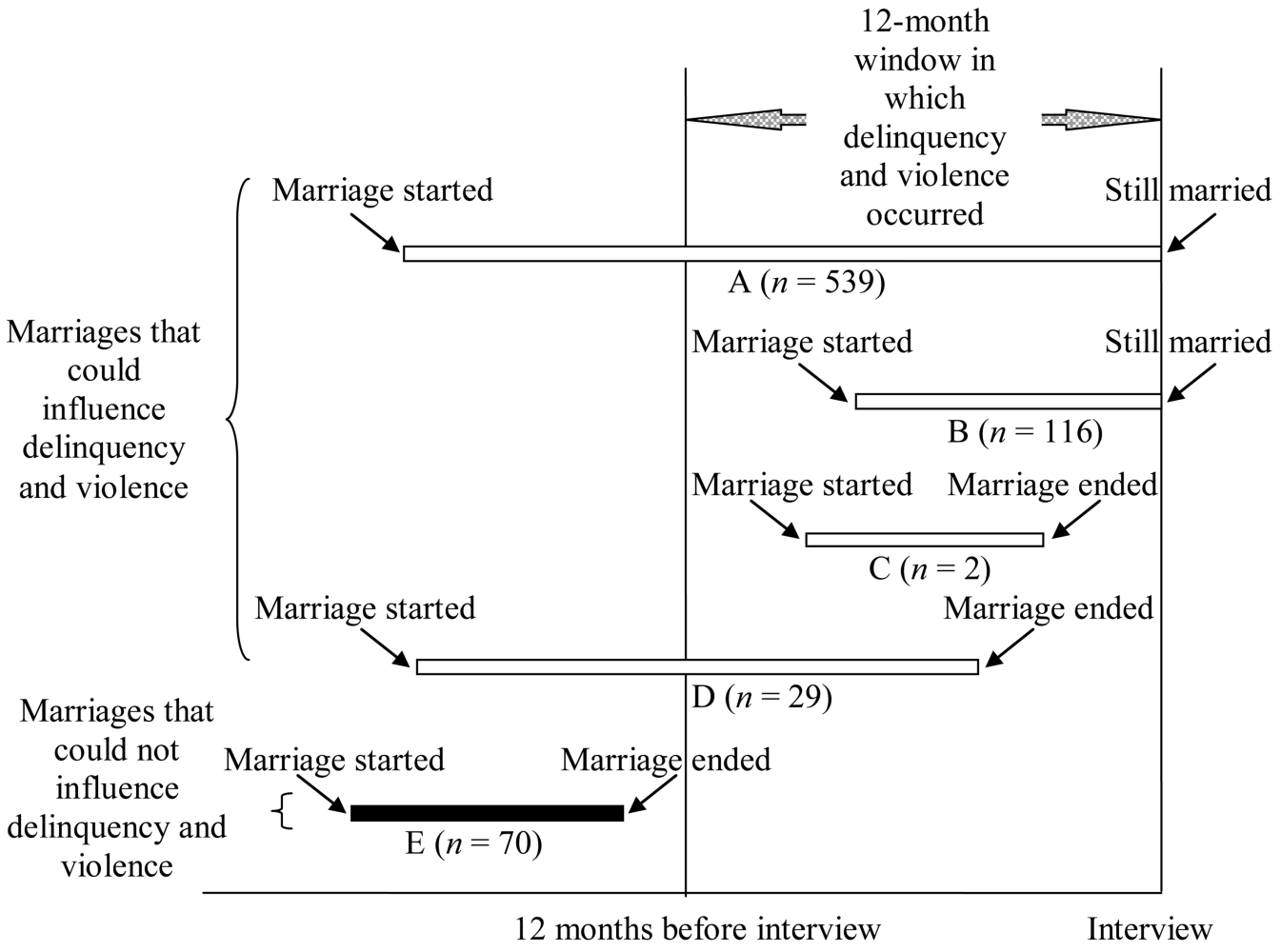


**Figure 1.**

Temporal Order Between Marriage and Delinquency/Violence: Distinguishing Marriages That Could Influence Delinquency/Violence and Marriages That Could Not.

*Note*. White bars represent marriages that overlapped the 12-month window and could influence delinquency and violence that occurred in the 12-month window (Types A–D). Black bars represent marriages that ended 12 months before the interview and could not influence delinquency and violence that occurred in the 12-month window (Type E). The ns in parentheses indicate the number of marriages for each type. Two marriages were intact when the couples were interviewed, but their start dates were missing. We considered these two marriages could influence delinquency and violence.

## Table 1
### Descriptive Statistics for All Variables: Means and Proportions

| Variable | Wave I 1994–1995 (*N* = 1,253–1,254) | Wave II 1996 (*N* = 1,196–1,254) | Wave III 2001–2002 (*N* = 1,249–1,254) | Wave IV 2008–2009 (*N* = 1,117–1,254) |
|---|---|---|---|---|
| | ***M* or proportion** | ***M* or proportion** | ***M* or proportion** | ***M* or proportion** |
| Delinquency | 1.05 | 0.80 | 0.44 | 0.21 |
| | (1.82) | (1.62) | (1.21) | (0.86) |
| Violence | 1.91 | 1.20 | 0.55 | 0.47 |
| | (2.65) | (2.05) | (1.31) | (1.08) |
| Marital status | | | | |
| Married | | | .16 | .43 |
| Unmarried | | | .84 | .57 |
| Age | 15.46 | 16.39 | 21.80 | 28.26 |
| | (1.60) | (1.62) | (1.64) | (1.68) |
| Gender[a] | | | | |
| Female | .42 | | | |
| Male | .58 | | | |
| Race[a] | | | | |
| American Indian | .03 | | | |
| Asian | .06 | | | |
| Black | .17 | | | |
| Multiracial | .05 | | | |
| Other | .01 | | | |
| White | .67 | | | |
| Bio-ancestry score[a] | | | | |
| African | .19 | | | |
| | (.35) | | | |
| East Asian | .12 | | | |
| | (.25) | | | |
| European | .70 | | | |
| | (.39) | | | |
| Education | | | | |
| No college | | | .51 | .34 |
| College | | | .48 | .55 |
| Missing | | | .01 | .10 |
| Employment | | | | |
| Unemployed | | | .30 | .17 |
| Employed | | | .70 | .72 |
| Missing | | | .00 | .11 |

| Variable | Wave I 1994–1995 (*N* = 1,253–1,254) | Wave II 1996 (*N* = 1,196–1,254) | Wave III 2001–2002 (*N* = 1,249–1,254) | Wave IV 2008–2009 (*N* = 1,117–1,254) |
|---|---|---|---|---|
| | ***M* or proportion** | ***M* or proportion** | ***M* or proportion** | ***M* or proportion** |
| Churchgoing frequency | | | | |
| Less than weekly | .61 | .61 | .83 | .76 |
| Weekly or more | .38 | .33 | .16 | .13 |
| Missing | .02 | .06 | .01 | .10 |
| Household size | | | | |
| < 3 | .01 | .08 | .29 | .30 |
| 3–6 | .67 | .66 | .58 | .51 |
| > 6 | .33 | .26 | .13 | .08 |
| Missing | .00 | .00 | .00 | .11 |
| Verbal IQ (PVT) score | | | | |
| < 90 | .23 | | | |
| 90–110 | .48 | | | |
| > 110 | .26 | | | |
| Missing | .03 | | | |
| Parental education | | | | |
| Below high school | .12 | | | |
| High school | .29 | | | |
| More than high school | .55 | | | |
| Missing | .04 | | | |
| Closeness to parents[b] | | | | |
| Not close | .39 | | | |
| Close | .60 | | | |
| Missing | .02 | | | |

*Note*. Numbers in parentheses are standard deviations. We imputed control variables to the maximum sample size: 1,254. We did not impute delinquency, violence, and marriage. PVT = Picture Vocabulary Test.

[a]The distributions of gender across four waves were almost identical; so were race and bio-ancestry score. Information at Wave I is presented for the three variables.

[b]*Not close* was defined as “somewhat,” “very little,” and “not at all” close to parents, and *close* was defined as “very much” and “quite a bit” close to parents.

## Table 2
### The Effect of Marriage on Delinquency and Violence: Generalized Estimating Equations

| Variable | Delinquency | Violence | Delinquency + violence |
|---|---|---|---|
| Married (ref.: unmarried) | −0.17*** | −0.17*** | −0.34*** |
| Age | −0.19* | −0.09 | −0.27 |
| Age$^2$ | 0.00 | 0.00 | 0.00 |
| Female (ref.: male) | −0.21*** | −0.43*** | −0.64*** |
| Race (ref.: White) | | | |
| American Indian | 0.20 | −0.04 | 0.16 |
| Asian | −0.24* | −0.11 | −0.24* |
| Black | 0.09 | 0.08 | 0.00 |
| Multiracial | −0.05 | 0.03 | −0.02 |
| Other | −0.25 | −0.32* | −0.57** |
| Education (ref.: no college) | | | |
| College or more | −0.01 | −0.15** | −0.15 |
| Employment (ref.: unemployed) | | | |
| Employed | −0.07 | −0.20** | −0.26** |
| Churchgoing frequency (ref.: less than weekly) | | | |
| Weekly or more | −0.06 | −0.10 | −0.17 |
| Household size (ref.: 3–6) | | | |
| < 3 | 0.03 | 0.04 | 0.07 |
| > 6 | −0.09 | 0.02 | −0.07 |
| PVT score (ref.: 90–110) | | | |
| < 90 | −0.05 | 0.02 | −0.03 |
| > 110 | 0.05 | −0.03 | 0.03 |
| Parental education (ref.: high school) | | | |
| Below high school | 0.05 | −0.06 | −0.01 |
| More than high school | 0.06 | 0.05 | 0.11 |
| Closeness to parents (ref.: close) | | | |
| Not close | 0.11 | 0.04 | 0.14 |
| Number of observations | 2,364 | 2,369 | 2,367 |

*Note*. *N* individuals = 1,254 for each dependent variable. The dependent variables are delinquency and violence measured at Waves III and IV. ref. = reference category; PVT = Picture Vocabulary Test.

* $p < .05$.

** $p < .01$.

*** $p < .001$ (two-tailed tests).

## Table 3

**Percentage of Variance in Delinquency and Violence Explained by 580 Single Nucleotide Polymorphisms (SNPs), Mixed Linear Models Estimated in Genome-Wide Complex Traits Analysis (GCTA) Software**

| Estimate | Wave III dependent variable | | | | | | Wave IV dependent variable | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Delinquency | | Violence | | Delinquency + violence | | Violence | | Delinquency + violence | |
| | Married | Unmarried | Married | Unmarried | Married | Unmarried | Married | Unmarried | Married | Unmarried |
| Percentage of variance explained by 580 SNPs | 0.00[a] | 1.09***[a] | 0.00[a] | 3.56***[a] | 0.00[a] | 1.48***[a] | 0.00[a] | 0.26***[a] | 0.09[a] | 0.14***[a] |
| Number of individuals | 191 | 835 | 193 | 837 | 191 | 837 | 428 | 546 | 428 | 546 |

*Note*. All models controlled for age, age squared, gender, race, education, employment, churchgoing frequency, household size, verbal IQ score, parental education, closeness to parents, and bio-ancestry scores. Because the distribution of delinquency at Wave IV was highly right skewed (over 90% of individuals scored 0 on the delinquency scale), estimates of the mixed linear models were not reliable. Therefore, we do not present results for delinquency at Wave IV.

[a] Kolmogorov–Smirnov test of whether the distribution of proportions of variance estimated in married individuals was smaller than in unmarried individuals.

*** $p < .001$.

**Table 4**

**Addressing Selection: Test of Whether More Antisocial Individuals Are Less Likely to Get Married by Using Delinquency and Violence at Waves I/II to Predict Marital Status at Waves III/IV: Generalized Estimating Equations**

| Variable | Married in 2001–2002, Wave III | | | | | | Married in 2008–2009, Wave IV | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 1[a] | 2[a] | 3[a] | 4[b] | 5[b] | 6[b] | 7[c] | 8[c] | 9[c] | 10[d] | 11[d] | 12[d] |
| Antisocial behavior in 1994–1995, Wave I | | | | | | | | | | | | |
| Delinquency | 0.02(0.62) | | | | | | −0.05(0.19) | | | | | |
| Violence | | 0.05(0.08) | | | | | | −0.02(0.38) | | | | |
| Delinquency + violence | | | 0.03(0.15) | | | | | | −0.02(0.23) | | | |
| Antisocial behavior in 1996, Wave II | | | | | | | | | | | | |
| Delinquency | | | | 0.04(0.50) | | | | | | −0.03(0.41) | | |
| Violence | | | | | 0.02(0.56) | | | | | | −0.02(0.46) | |
| Delinquency + violence | | | | | | 0.02(0.45) | | | | | | −0.02(0.36) |

*Note*. Numbers in parentheses are *p* values. All models controlled for age, gender, race, churchgoing frequency, household size, verbal IQ score, parental education, and closeness to parents. Results from (6 × 2 =) 12 regressions are presented as six measures of antisocial behavior at Waves I and II were used to predict marital status at Wave III or IV. Every entry is based on a separate regression.

[a] $N = 1,252$.

[b] $N = 1,195$.

[c] $N = 1,120$.

[d] $N = 1,066$.

**Table 5**

**The Confounding Effect of Age and Population Heterogeneity: Test of the Marriage Effect Using the Age-Comparable Sample (Left Panel) and Test of Whether the Marriage Effect Differs for Persistent Offenders (Right Panel): Generalized Estimating Equations**

| Variable | Age-comparable sample[a] | | | Original sample | | |
|---|---|---|---|---|---|---|
| | Delinquency | Violence | Delinquency + violence | Delinquency | Violence | Delinquency + violence |
| Married (ref.: unmarried) | −0.23*** | −0.20*** | −0.43*** | −0.15*** | −0.13** | −0.28*** |
| Persistent offender(ref.: nonpersistent offender) | | | | 0.67** | 1.72*** | 2.39*** |
| Persistent offender × married | | | | −0.16 | 0.37 | 0.18 |
| Number of individuals | 1,168 | 1,168 | 1,168 | 1,254 | 1,254 | 1,254 |
| Number of observations | 1,542 | 1,546 | 1,544 | 2,364 | 2,369 | 2,367 |

*Note*. The dependent variables are delinquency and violence measured at Waves III and IV. All models controlled for age, age squared, gender, race, education, employment, churchgoing frequency, household size, verbal IQ score, parental education, and closeness to parents. ref. = reference category.

[a]In the age-comparable sample, the mean ages were 26 for both married and unmarried individuals after we randomly excluded a subset of young unmarried individuals. In the original sample, the mean age was 26 for married individuals and 24 for unmarried individuals.

** $p < .01$.

*** $p < .001$.