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Does Marriage Inhibit Antisocial Behavior? An Examination of Selection versus Causation via a Longitudinal Twin Design

S. Alexandra Burt, PhD[1], **M. Brent Donnellan, PhD**[1], **Mikhila N. Humbad, MA**[1], **Brian M. Hicks, PhD**[2], **Matt McGue, PhD**[3], and **William G. Iacono, PhD**[3]

[1]Department of Psychology, Michigan State University, East Lansing, MI

[2]Department of Psychiatry, University of Michigan, Ann Arbor, MI

[3]Department of Psychology, University of Minnesota, Minneapolis, MN

Abstract

Context—Prior studies have indicated that marriage is negatively associated with male antisocial behavior. Although often interpreted as a causal association, marriage is not a random event. As such, the association may stem from selection processes, whereby men less inclined towards antisocial behavior are more likely to marry.

Objective—To evaluate selection versus causation explanations of the association between marriage and desistence from antisocial behavior.

Design—Co-twin control analyses in a prospective twin study provided an analog of the idealized counterfactual model of causation. The co-twin control design uses the unmarried co-twin of a married twin to estimate what the married twin would have looked like had he remained unmarried. Discordant monozygotic (MZ) twins are particularly informative as they share a common genotype and rearing environment.

Setting—General community

Participants—289 male-male twin pairs (65% MZ) from the Minnesota Twin Family Study assessed at ages 17, 20, 24, and 29 years. None of the participants were married at age 17, and 2.6% were married at age 20. By age 29, 58.8% of the participants were or had been married.

Interventions—None

Main Outcome Measure—A tally of Criterion C symptoms of DSM-III-R Antisocial Personality Disorder, as assessed via structured clinical interview.

Results—Mean differences in antisocial behavior across marital status were present even at ages 17 and 20, suggesting a selection process. However, the within-pair effect of marriage was significant for MZ twins, such that the married twin engaged in less antisocial behavior than his unmarried co-twin. Results were equivalent to those in dizygotic twins and persisted when controlling for prior antisocial behavior.

Conclusions—Results indicate an initial selection effect, whereby men with lower levels of antisocial behavior are more likely to marry. However, this tendency to refrain from antisocial behavior appears to be accentuated by the state of marriage.

Correspondence: Address correspondence to Alex Burt, Department of Psychology, Michigan State University, 107D Psychology Building, East Lansing, MI, 48824. Electronic mail may be sent to burts@msu.edu. Phone and fax numbers are 517-432-5602 and 517-432-2476, respectively.

Keywords

antisocial personality disorder; marriage; desistence; twin study; co-twin control analyses

A particularly provocative set of findings within the antisocial behavior literature concerns the role of marriage in inhibiting these behaviors in adult men. Indeed, there is now convincing evidence that the state of marriage is associated with lower crime rates[1–4]. For example, a recent study[2] examined within-individual associations between marriage and antisocial behavior in a sample of 475 high-risk boys followed from adolescence through adulthood and found that the average reduction in crime with marriage was approximately 35%, a rather remarkable decrease. Other research found that living with one's wife was negatively associated even with month-to-month variation in crime rates[4]. In short, the "marriage effect" on desistence from antisocial behavior appears to be a robust one.

Mechanisms thought to account for the association between marriage and desistence from antisocial behavior[2] typically center on social control or social bonding[3], decreased affiliation with deviant peers[5], and/or direct social control exerted by wives on their husbands[2]. In other words, extant research has generally interpreted the marriage effect as a causal one, whereby marriage inhibits subsequent antisocial behavior (either directly or indirectly). Should this be true, it could provide valuable leads for enhancing treatment development, as more concrete identification of specific mechanisms may provide a potentially powerful framework for improving interventions. Critically, however, marriage is not a random event, and thus the link between marriage and desistence from antisocial behavior could instead be attributable to selection effects (a possibility that was first discussed more than 20 years ago6). Under this scenario, men who are less inclined towards antisocial behavior (for whatever reason) are more likely to marry. As one example, there is a well-known association between antisocial behavior and low socioeconomic status7, the latter of which is also predicts marriage (in the form of economic potential)8. Selection is thus a key confound in studies suggesting that marriage causally contributes to desistence from antisocial behavior6.

This possibility of selection has not gone unnoticed by researchers, many of whom have attempted to address this issue using sophisticated within-person statistical techniques that draw on the counterfactual method2, 4. The counterfactual method9, [10] provides both a definition of a causal effect and an integrative framework for estimating these effects. The average causal effect would be the person's outcome when married as compared to the same person's outcome when not married. As simultaneous observations of these two outcomes are clearly impossible, however, the counterfactual method places the problem of causal inference within a general missing data framework. Specifically, researchers would estimate the missing non-married outcomes for those who were married, as well as the missing married outcomes for those who were not (yet) married, to infer causal effects.

Although these methods allow for reasonable inferences regarding the effects of marriage on antisocial behavior, we know of no study that has examined this question within a prospective, genetically-informative sample. Such samples are ideally suited for conducting counterfactual studies because monozygotic twins raised in the same family differ only to the extent that they have been exposed to different environmental factors[11]. Indeed, comparisons of twins discordant for marriage would offer particularly compelling support for or against an environmentally mediated effect of marriage on antisocial behavior, and in this way, would constitute the most powerful test of this association available to date. The current study sought to do just this, examining a sample of male-male twins pairs assessed at ages 17, 20, 24, and 29 years. Data were analyzed using an analog of the idealized

counterfactual model of causation, the co-twin control design[12], in which the unmarried co-twin of a married twin is used to estimate what the married twin would have looked like had he remained unmarried. The current study thus offers a unique and novel opportunity to evaluate the meaning and origins of desistence from antisocial behavior with marriage.

Methods

PARTICIPANTS

The sample was drawn from participants in the ongoing and longitudinal Minnesota Twin Family Study (MTFS). Detailed information regarding the design, recruitment procedures, and participation rates of the MTFS has been provided elsewhere[13]. The original intake sample of same-sex male twins consisted of 289 reared-together pairs ($n_{monozygotic}$ = 188, $n_{dizygotic}$ = 101). Participants were roughly 17 years-old at the time of their intake visit (which took place between 1990 and 1995). Twins were assessed again at approximately 20, 24, and 29 years of age. A total of 478 (83%), 495 (86%), and 532 (92%) twins completed the age 20, age 24, and age 29 assessments, respectively. Moreover, those who completed at least one follow-up assessment reported levels of antisocial behavior that were equivalent to those who participated only at intake (1.08 versus .92 symptoms of antisocial behavior, as defined below, p = .81).

ZYGOSITY DETERMINATION

Zygosity was determined by the agreement of several separate estimates: 1) Ponderal and cephalic indices and fingerprint ridge counts were measured; 2) MTFS staff evaluated visage, hair color, and face and ear shape for physical similarity; and 3) Parents completed a standard zygosity questionnaire at the intake assessment. When these estimates did not agree, a serological analysis was performed to determine zygosity. A previous validation study (n=50 pairs) found that when the three estimates agreed, zygosity was uniformly confirmed by the serological analysis[14]. This suggests that our method of zygosity determination is accurate.

MEASURES

Antisocial Behavior—Participants were assessed in-person for DSM-III-R mental disorders (the manual that was current at the onset of the study) by trained bachelor and masters-level interviewers at all four visits. The reporting period was “lifetime” (i.e., since age 15, per the DSM-III-R AAB criteria) at ages 17 and 20. The age 24 and age 29 interviews assessed symptoms present since the previous visit. Each twin within a pair was interviewed by a different interviewer. Adult antisocial behavior (AAB) was operationalized as a tally of endorsed and partially-endorsed Criterion C symptoms of DSM-III-R Antisocial Personality Disorder (ASPD; e.g., repeated illegal acts, irritability and aggressiveness, disregard for the truth, lack of remorse). Symptoms were assessed via the Structured Clinical Interview for personality disorders (SCID-II)[15]. Although AAB does not constitute a DSM diagnosis (ASPD diagnoses require at least three Criterion A symptoms of Conduct Disorder as well), if either 3 or 4 symptoms are used to define a “diagnosis” of AAB, the kappa inter-rater reliability exceeds .78. Roughly 4% of the sample (3.9%) met full criteria for lifetime ASPD. Another 4.3% were one symptom shy of a full ASPD diagnosis.

Following the interview, a clinical case conference was held in which the evidence for every symptom was discussed by at least two advanced clinical psychology doctoral students (neither of whom conducted the interview). As actual diagnoses were not used, duration rules were excluded. Computer algorithms were used to sum the number of symptoms. Symptoms judged to be definitely present (i.e., they were clinically significant in both severity and frequency) were counted as one full symptom. Symptoms judged to be

probably present (i.e., they were clinically significant in either severity or frequency, but not both) were counted as half of a symptom. Symptom counts, rather than diagnoses, were used primarily to increase statistical power, as diagnostic prevalence rates of ASPD in community-based samples are lower than in clinically-referred samples. To adjust for positive skew, all AAB symptom counts were log-transformed prior to analysis.

Marital History—Marital history was assessed at age 29 via a life events questionnaire16, which included questions on marital status and age at marriage. Nearly 60% of participants (58.8%) were or had been married at their age 29 assessment. Consistent with the demographics of marriage in the United States[17], none of the twins were married at their age 17 assessment, and only 15 were married at their age 20 assessment. By their age 24 assessment, 22.9% of participating twins had married. The remainder of the married participants did so between their age 24 and age 29 assessments. Of the 58.8% of participants who had ever married, 18 had divorced (4 of these men had married before their age 20 assessment and 9 had married by the time of their age 24 assessment). As we made use of current marital status for our analyses, these 18 men were coded as unmarried at age 29 (although several of them were coded as married at age 24). When analyses were repeated omitting these men, our conclusions remained entirely unchanged.

ANALYSES

Our analytic approach was predicated on the various sources of similarities and differences across reared-together twins. All twins shared their rearing environment at 100%. However, whereas monozygotic (MZ) twins share 100% of their genetic material, dizygotic (DZ) twins share an average of 50% of their segregating genetic material. Differences between MZ twins are thus due solely to person-specific or unique environmental influences (such as marriage), as well as measurement error. Differences between DZ twins, by contrast, are due to both these person-specific environmental influences as well as the 50% of segregating genes they do not share. More information on genetically-informative studies is provided elsewhere[11].

We conducted a series of interrelated analyses to examine the origins of the association between marriage and desistence from AAB. We first compared mean levels of AAB by age 29 marital status, both between- and within-persons. We next evaluated these associations within sibling pairs using an analog of the idealized counterfactual model of causation, the co-twin control design[12]. The co-twin control design uses the unmarried co-twin of a married twin to estimate what the married twin would have looked like had he remained unmarried. In more specific terms, let y_{ij} be the observed outcome for the j^{th} twin (j=1,2) in the i^{th} twin pair (i=1,2…, N) and let x_{ij} be the corresponding exposure index (in this case, marital status) for this individual. The overall, or individual-level, regression of the outcome on the exposure is given by the regression model

$$y_{ij}=\beta_0+\beta_1 x_{ij}+\varepsilon_{ij} \quad (1)$$

where β_1 is the individual-level effect of exposure (marriage) on outcome (AAB), β_0 is the intercept term, and ε_{ij} is the residual (correlated across the two members of a twin pair). The overall regression effect can be further represented in terms of a within-pair (β_w) and a between-pair (β_B) effect using the regression model

$$y_{ij}=\beta_0+\beta_w(x_{ij}-\bar{x}_{i.})+\beta_B\bar{x}_{i.}+\varepsilon_{ij} \quad (2)$$

where $\bar{x}_i$. is the mean exposure index for the i^{th} twin pair. The between-pair regression coefficient provides an approximation of the individual-level effect. The within-pair regression coefficient provides a direct estimate of the effect of exposure on the outcome (in this case, the effect of marriage on AAB) within discordant twin pairs.

This regression model can be further conceptualized within a genetically informed design[12, 19]. Individual-level associations reflect potential confounding of genetic effects (A), shared or familial environmental effects (C), and non-shared or person-specific environmental effects (E). Associations within DZ twin pairs discordant for exposure control for C effects and partially for A effects. Associations within MZ twin pairs discordant for exposure control for both C and A effects. Any remaining associations within discordant MZ pairs therefore directly index E effects. Accordingly, should marriage be environmentally or causally linked to reductions in antisocial behavior, we would expect to observe this association at the individual-level, within DZ twin pairs discordant for exposure, and within MZ twin pairs discordant for exposure (see Figure 1; scenario A). By contrast, the failure to observe an association within discordant MZ twin pairs would imply that the association of exposure with outcome is solely attributable to selection processes (i.e., the process is mediated by genetic and/or shared environmental effects, rather than non-shared environmental effects, given that there is no association between exposure and outcome in discordant MZ twins). In particular, if exposure was associated with outcome at the individual-level and in discordant DZ twins (scenario B), we would infer that the selection process was genetic in origin. If the exposure was associated with outcome only at the individual-level (scenario C), we would infer that the selection process was genetic and shared environmental in origin.

It is useful to note that prior work with MTFS twins[19] estimated the heritability of AAB at approximately 50%, with the remaining variance attributable to the non-shared environment. Moreover, changes in AAB over time (which would necessarily include desistence from AAB) were also found to be primarily non-shared environmental in origin. Such findings suggest that the co-twin control approach may be particularly useful for understanding environmental predictors of desistence from AAB.

The co-twin control analyses were conducted using multilevel modeling (MLM) in SPSS 17.0[18]. As MLM coefficients are unstandardized, we standardized our outcome variable for the MLM analyses (i.e., log-transformed AAB at age 29) to have a mean of zero and a standard deviation of 1.0 to facilitate interpretation of the magnitude of the fixed effect estimates.

Results

Between-persons analyses

Mean AAB symptom counts are presented in Table 1 separately by marital status. Analyses were conducted on the log-transformed AAB data, as they better approximate normality. However, the corresponding raw symptom counts are presented in Table 1 and in the text to promote ease of understanding. As seen there, mean levels of AAB varied significantly by age 29 marital status across all waves of data. The effect was particularly pronounced (and medium in magnitude20) for AAB at age 29, but was also relatively pronounced at age 24. At ages 17 and 20, the effect sizes were small in magnitude. Such findings circumstantially suggest that the impact of marriage on AAB may be more pronounced following the marriage. More importantly, however, the finding that levels of AAB differed well before marriage is noteworthy, as it suggests that adolescents who would later be married engaged in lower levels of AAB than did adolescents who remained unmarried at age 29.

Although we did know age at marriage, we were not able to unambiguously establish participants' ages when they met their future wives. It is thus possible (if unlikely) that most of our married participants were dating their future wives at age 17, and thus had already begun to desist. To circumvent this possibility, we took advantage of estimates indicating that the average length of courtship before a first marriage is between 2 and 3 years[21], and repeated our age 17 analyses omitting those men married before their age 24 assessment (i.e., we compared men married at age 25 or later to those that remained unmarried). Results were fully replicated. Married men again reported less AAB at age 17 (mean = 0.67, SD = 1.10) than did men who remained unmarried (mean = 1.02, SD = 1.33; $p < .01$; Cohen's *d* standardized effect size = .29). Expected mean differences were also present at ages 20 (*d* = .21), 24 (*d* = .29), and 29 (*d* = .41), all p<.05. In short, even when the time-lag between marriage and the intake assessment was 8 years or more, marriage was associated with lower levels of AAB in adolescence. Such results are most consistent with selection effects.

Within-persons analyses

Mean-level comparisons were also conducted within-persons via paired samples t-tests, so as to provide a preliminary examination of an additional impact of marriage on desistence from AAB. As seen in Table 1, AAB increased significantly from ages 17 to 20 regardless of later marital status (both $p < .001$). Such results are consistent with a normative increase in AAB during late adolescence[22, 23]. Following this increase, however, levels of AAB remained essentially constant for those who remained unmarried (means at ages 20 and 24, and at ages 24 and 29, were statistically equivalent; both $p > .16$). For married men, however, a different pattern emerged. Mean levels of AAB remained constant from ages 20 to 24 (p = .47), and then decreased significantly from ages 24 to 29 ($p = .001$). The timing of this decrease is noteworthy given that most men were married between their age 24 and age 29 assessments. Indeed, when we restricted our married analyses to those men who were married between their age 20 and 24 assessments, we found that mean levels of AAB decreased from ages 20 to 24 (age 20: mean (SD) = 1.21 (1.31); age 24: mean (SD) = 0.88 (0.87); $p < .01$). There thus appears to be a within-persons effect of marriage on desistence from antisocial behavior.

Co-twin control analyses

Results of the co-twin control analyses are presented in Table 2 and Figure 2. As seen there, both the between- and within-pair fixed effect estimates were negative, consistent with the notion that AAB decreases with the advent of marriage. The between-pair effect estimates were large and significant for both MZ and DZ twins, results that are in keeping with the between-persons comparisons presented above. The within-pair estimates were also statistically significant for both MZ and DZ twins. Moreover, the difference between the MZ and DZ within-pair estimates was not statistically significant ($p = .91$). Using the interpretative framework outlined in Figure 1, the latter results collectively suggest that involvement in marriage acts to inhibit AAB. That said, the MZ between-pair estimate (which can be used to approximate the individual-level effect) was significantly larger than the corresponding within-pair effect estimate in the first or no covariates model[24] (t = 1.79, p<.05, one-tailed, since we were explicitly testing whether between-pair estimate was larger than the within-pair estimate). Such findings are consistent with the notion that selection also plays a role in this association.

To more fully test the possibility of environmental causation, however, it is also necessary to control for pre-existing levels of AAB by making use of our longitudinal data. We thus re-ran the co-twin control analyses including reports of lifetime AAB prior to marriage (i.e., at ages 17 or 20) as covariates. Regardless of whether we controlled for AAB at age 17 or age 20, results were quite similar to those reported above. Specifically, the within-pair effect

was statistically significant for MZ twins (both $p < .05$), and moreover, was not significantly greater in DZ twins (both $p \geq .75$).

As a final check on our results, we re-ran analyses examining whether marital status at age 24 predicted AAB at age 24 even when controlling for AAB at ages 17 or 20. The within-MZ effect of marriage on AAB remained statistically significant (fixed effect estimate (SE) = −.28 (.18); $p < .05$) and was not statistically from that in the DZ pairs ($p = .69$), even when controlling for AAB at 17. Controlling for AAB at 20 yielded similar results: the within-MZ effect of marriage on AAB was statistically significant (fixed effect estimate (SE) = −.29 (.17); $p < .05$) and was not statistically from that in the DZ pairs ($p = .83$).

Supplemental analyses

To maintain consistency across assessments, we made use of the DSM-III-R criteria for AAB in the above analyses. However, because DSM-IV symptoms were also assessed at older ages, we repeated our no covariate co-twin control analyses using DSM-IV AAB at age 29 as our outcome variable. The between-pair effect estimates were large and significant (fixed effect estimate (SE) = −.56 (.15) for MZ twins and −.54 (.23) for DZ twins; $p < .01$). The within-pair estimates were also statistically significant (fixed effect estimate (SE) = −.26 (.15) for MZ twins and −.25 (.17) for DZ twins; $p < .05$). Moreover, the difference between the MZ and DZ within-pair estimates was not statistically significant ($p = .97$). Our results thus extend to DSM-IV AAB as well.

Discussion

The aim of the present study was to clarify the origins of the association between marriage and desistence from antisocial behavior using a population-based sample of male twins assessed up to four times between the ages of 17 and 29. Analyses offered support for both selection and causation explanations. Specifically, mean differences in antisocial behavior across married and unmarried men preceded the state of marriage by many years, and did so even when restricting the married sample to those who married at age 25 or later. Moreover, there was some evidence of selection in our co-twin control analyses, as the MZ between-pair and within-pair estimates could not be constrained to be equal. Such findings collectively point to an important role for selection processes, whereby men who eventually married were less prone to antisocial behavior during adolescence and emerging adulthood than were men who remain unmarried at age 29. However, visual inspection of the mean differences was also consistent with the possibility that entrance into the state of marriage may accentuate these pre-existing differences. These suspicions were borne out in our co-twin control analyses. At both ages 24 and 29, the within-pair effect of marriage on AAB was statistically significant for MZ twins. As MZ twins share all of their genes and early rearing environment, such results are indicative of a person-specific or non-shared environmentally mediated impact of marriage on desistence from AAB. That these results were equivalent to those in DZ twins, and moreover, persisted even when controlling for prior AAB, further bolstered our conclusion that marriage also serves to inhibit AAB. In short, the current results indicate that while men with lower levels of antisocial behavior are more likely to marry by age 29, entrance into the state of marriage accentuates their tendency to refrain from antisocial behavior.

Our findings are generally consistent with prior literature. Previous studies[1–4] within the field of criminology have pointed to a causal effect of marriage on desistence from antisocial behavior. Perhaps the strongest such study found that the average reduction in crime with entry into marriage was approximately 35%[2]. Our own results were very consistent with these findings. At age 29, the Cohen's *d* effect size for differences in AAB

by marital status was .48, which corresponds to slightly more than a 30% reduction in AAB with marriage.

That said, our results also implicated the presence of selection processes, such that men who married by age 29 were less prone to antisocial behavior as adolescents than were their unmarried peers. Other studies, by contrast, have found little evidence in support of selection2, 3. Although it is not clear what may account for this difference across studies, one possibility is clinical severity. Prior work has often examined high-risk/criminologic samples (such as delinquent boys who had been committed to reform schools during adolescence2, 3), whereas the current sample was population-based. It may be that selection processes are more important (or are simply easier to detect) in population-based samples. Cohort effects are yet another, potentially more important, difference between samples. The current sample was born between 1972 and 1978, whereas the aforementioned high-risk sample2, 3 was born between 1924 and 1932. These cohort differences may be particularly salient in the current study given changes in the frequency and psychological meaning of marriage since the 1960s and 1970s25. Indeed, the proportion of never married individuals has steadily increased since the 1970s as has the median age at first marriage26. As marriage thus seems to be increasingly linked to individual choice rather than societal expectations, selection processes could simply be more influential in more recent decades. By contrast, there may have been little room for selection to exert a detectable effect in prior decades.

There are several limitations to bear in mind when interpreting the results of this study. First, only men were examined in the current study, as the link between marriage and desistence from antisocial behavior among women has been less consistently supported2, 27. It thus remains unclear whether and how these findings might generalize to women. Building on this point, although we would expect assortative mating to operate in the choice of spouse (such that more antisocial men would marry more antisocial women28), this process was not examined here as we do not have this information on the twin spouses. Moreover, we did not account for the possibility of psychiatric comorbidity, which may well act as a hindrance to desistence from AAB. Future researchers should seek to understand the role of assortative mating and psychiatric comorbidity in desistence from antisocial behavior.

The current results apply only to early adulthood and not to later developmental periods. This point is particularly salient since it is likely that many of the men who were unmarried as of their age 29 assessment will eventually marry17. However, because antisocial behavior is more common in early adulthood than in later developmental periods29, 30, early adulthood is a critical time to investigate predictors of antisocial behavior. It is also unclear whether the effects identified here are specific to marriage or whether they extend to other committed romantic states (i.e., engagement or cohabitation). We would expect our findings to generalize beyond marriage, as the presumed mediators of these effects (e.g., social control) should generalize to other sorts of romantic bonds. That said, at least one study[4] found that the marriage effect did not extend to cohabitation. Future work should examine this possibility.

Finally, although extensive evidence now suggests that child- and adolescent-onset antisocial behavior differ etiologically[23], data regarding early-onset “caseness” was not available for the present study. One possible complication of this is that, if adolescent-onset cases were more numerous in the married group, they may be driving the change observed in response to marriage. That said, evidence indicating that the state of marriage inhibits antisocial behavior has also been found in high-risk/criminologic samples likely to contain a large(r) number of life-course persistent individuals[1–4]. In any case, future research should evaluate whether these findings vary by the age-of-onset of antisocial behavior.

In spite of these limitations, the current results provide an important constructive replication and extension of prior findings indicating that entrance into the state of marriage inhibits male antisocial behavior. Rather than resulting solely from misidentified selection processes, it appears that marriage represents a potent and at least partially environmentally mediated influence on desistence from antisocial behavior. As argued by prior scholars[2], however, it seems unlikely that the institution of marriage acts to inhibit men's antisocial behavior directly; rather, marriage is likely a marker for other more proximal and causal processes. For example, prior work has suggested that the quality of the marital bond may mediate this effect[3]. Future work should seek to more exhaustively identify the mechanisms mediating the impact of marriage on antisocial behavior.

Despite this evidence of an environmentally mediated effect of marriage on desistence from antisocial behavior, however, our results also implicate a clear role for selection processes, whereby men less prone to antisocial behavior as adolescents are more likely to marry (at least by age 29). There are many possible explanations for such findings[6]. It may be that less antisocial men simply make more attractive marital partners, and are thus more likely to be *selected for* marriage. Alternately, it may be that marriage is a less attractive option for men who engage in higher levels of antisocial behaviors, and they are thus less likely to *select into* marriage. The latter would be consistent with the theory of the active gene-environment correlation (in which individuals select into environments consistent with their genotype[31, 32]), a well-known theory thought to underlie mate selection in general[33]. Regardless, given that marriage also appears to facilitate desistence from antisocial behavior, future research should seek to distinguish between and better understand these selection processes.

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References

1. McGloin JM, Sullivan CJ, Piquero AR, Pratt TC. Local life circumstances and offending specialization/versatility. Journal of Research in Crime and Delinquency 2007;44:321–346.
2. Sampson RJ, Laub JH, Wimer C. Does marriage reduce crime? A counterfactual approach to within-individual causal effects. Criminology 2006;44:465–508.
3. Laub JH, Nagin DS, Sampson RJ. Trajectories of change in criminal offending: Good marriages and the desistence process. American Sociological Review 1998;63:225–238.
4. Horney JD, Osgood W, Marshall IH. Criminal careers in the short term: Intra-individual variability in crime and its relation to life circumstances. American Sociological Review 1995;60:655–673.
5. Warr M. Life-course transitions and desistence from crime. Criminology 1998;36:183–216.
6. Gottfredson, MR.; Hirschi, T. A General Theory of Crime. Stanford, CA: Stanford University Press; 1990.
7. Leventhal T, Brooks-Gunn J. The neighborhoods they live in: The effects of neighborhood residence on child and adolescent outcomes. Psychological Bulletin 2000;126:309–337. [PubMed: 10748645]
8. Xie Y, Raymo KG, Thornton A. Economic potential and entry into marriage and cohabitation. Demography 2003;40:351–367. [PubMed: 12846136]
9. Rubin DB. The design versus the analysis of observational studies for causal effects: Parallels with the design of randomized trials. Statistics in Medicine 2007;26:20–36. [PubMed: 17072897]
10. Rubin DB. For objective causal inference, design trumps analysis. Annals of Applied Statistics 2008;2:377–395.
11. Plomin, R.; DeFries, JC.; McClearn, GE.; McGruffin, P. Behavioral Genetics. 5th ed.. New York: Worth Publishers; 2008.

12. McGue M, Osler M, Christensen K. Causal inference and observational research: The utility of twins. Perspectives on Psychological Science. in press.
13. Iacono WG, Carlson SR, Taylor J, Elkins IJ, McGue M. Behavioral disinhibition and the development of substance-use disorders: Findings from the Minnesota Twin Family Study. Development & Psychopathology 1999;11:869–900. [PubMed: 10624730]
14. Han C, McGue M, Iacono WG. Lifetime tobacco, alcohol and other substance use in adolescent Minnesota twins: Univariate and multivariate behavioral genetic analyses. Addiction 1999;94:981–993. [PubMed: 10707437]
15. Spitzer, RL.; Williams, JBW.; Gibbon, M.; First, MB. Structured Clinical Interview for DSM-III-R Personality Disorders. New York: New York State Psychiatric Institute, Biometrics Research Division; 1987.
16. Billig JP, Hershberger SL, Iacono WG, McGue M. Life events and personality in late adolescence: Genetic and environmental relation. Behavior Genetics 1996;26:543–553. [PubMed: 8990533]
17. Goodwin, P.; McGill, B.; Chandra, A. NCHS data brief, # 19. Hyattsville, MD: National Center for Health Statistics; 2009. Who marries and when? Age at first marriage in the United States, 2002.
18. Norušis, MJ. SPSS 15.0 Advanced Statistical Procedures Companion. Upper Saddle River, NJ: Prentice Hall Inc.; 2007.
19. Burt SA, Carter LA, McGue M, Iacono WG. The different origins of stability and change in Antisocial Personality Disorder symptoms. Psychological Medicine 2007;37:27–38. [PubMed: 17049101]
20. Cohen, J. Statistical power analysis for the social sciences. 2nd ed.. Hillsdale, New Jersey: Lawrence Erlbuam; 1988.
21. Karney, BR.; Garvan, CW.; Thomas, MS. Family formation in Florida: 2003 baseline survey of attitudes, beliefs, and demographics relating to marriage and family formation. Gainesville, FL: University of Florida; 2003.
22. Moffitt TE. Adolescence-limited and life-course-persistent antisocial behavior: A developmental taxonomy. Psychological Reveiw 1993;100:674–701.
23. Moffitt, TE. Life-course persistent and adolescence-limited antisocial behavior: A research review and a research agenda. In: Lahey, B.; Moffitt, TE.; Caspi, A., editors. The causes of conduct disorder and serious juvenile delinquency. New York: Guilford; 2003.
24. Begg MD, Parides MK. Separation of individual-level and cluster-level covariate effects in regression analysis of correlated data. Statistics in Medicine 2003;22:2591–2602. [PubMed: 12898546]
25. Cherlin AJ. The deinstitutionalization of American marriage. Journal of Marriage and Family 2004;66:848–862.
26. Fields, J. Census Bureau. Washington, DC: 2003. America's Families and Living Arrangements: 2003. Current Population Reports, P20-553 U.S.
27. 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.
28. Humbad MN, Donnellan MB, Iacono WG, Burt SA. The association of marital quality with personality and psychopathology: Results from a very large sample of married couples. Journal of Abnormal Psychology 2010;119:151–162. [PubMed: 20141252]
29. Arnett JJ. Emerging adulthood: A theory of development from the late teens through the twenties. American Psychologist 2000;55(5):469–480. [PubMed: 10842426]
30. Arnett JJ. Emerging adulthood: What is it, and what is it good for? Child Development Perspectives 2007;1:68–73.
31. Plomin R, DeFries JC, Loehlin JC. Genotype-environment interaction and correlation in the analysis of human behavior. Psychological Bulletin 1977;84:309–322. [PubMed: 557211]
32. Scarr S, McCartney K. How people make their own environments: A theory of genotype-environment effects. Child Development 1983;54:424–435. [PubMed: 6683622]
33. Krueger RF, Moffitt TE, Caspi A, Bleske A, Silva PA. Assortative mating for antisocial behavior: Developmental and methodological implications. Behavior Genetics 1998;28:173–186. [PubMed: 9670593]

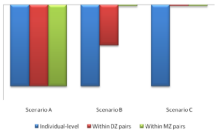

Figure 1. Interpretation of Co-Twin Control Results

Should marriage be environmentally or causally linked to reductions in antisocial behavior, we would expect to observe this association at the individual-level, within DZ twin pairs discordant for exposure, and within MZ twin pairs discordant for exposure (scenario A). By contrast, the failure to observe an association within discordant MZ twin pairs would imply that the association of exposure with outcome is solely attributable to selection processes. In particular, if exposure was associated with outcome at the individual-level and in discordant DZ twins (scenario B), we would infer that the selection process was genetic in origin. If the exposure was associated with outcome only at the individual-level (scenario C), we would infer that the selection process was genetic and shared environmental in origin.

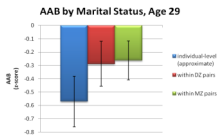

Figure 2. AAB by Marital Status, Age 29

Note. To facilitate interpretation of these unstandized fixed effect estimates (as also presented in Table 2), the Adult Antisocial Behavior (AAB) extended symptom count variable was standardized to have a mean of 0 and a standard deviation of 1 prior to analysis. Both marriage and AAB were assessed at age 29. Standard error bars are presented. The between-pair effect estimate, which approximates the individual-level effect, is also presented. Significant within-pair estimates for MZ twins are indicative of a non-shared environmentally-mediated relationship between marriage and AAB, and particularly so when the DZ within-pair estimate is equivalent to the MZ estimate.

Table 1

Mean Adult Antisocial Behavior (AAB) Extended Symptom Count by Age 29 Marital Status.

Outcome	Unmarried at 29 mean (SD)	Married at 29 mean (SD)	Cohen's *d* effect size
AAB at age 17	1.08 (1.38)	0.75 (1.17)	.26**
AAB at age 20	1.48 (1.45)	1.18 (1.30)	.22*
AAB at age 24	1.42 (1.19)	1.04 (1.03)	.34**
AAB at age 29	1.29 (1.06)	0.83 (0.83)	.48**

Note. ** and * indicate that mean difference in AAB across marital status is statistically significant at $p < .01$ and $p < .05$, respectively.

Table 2

Co-twin control analyses evaluating the association between Adult Antisocial Behavior (AAB) Extended Symptom Counts at age 29 and Marital Status at age 29.

Outcome	Fixed Effect Estimates (SE)					
	Between-pair		Within-pair		Prior AAB	
	MZ	DZ	MZ	DZ	Age 17	Age 20
AAB at 29						
1) *No covariate model*	−.57 (.15)***	−.57 (.23)***	−.26 (.14)**	−.29 (.17)**	---	---
2) *Controlling for AAB at age 20*	−.36 (.14)***	−.55 (.21)***	−.24 (.15)*	−.25 (.18)*	---	.68 (.07)***
3) *Controlling for AAB at age 17*	−.43 (.14)***	−.46 (.21)***	−.18 (.14)*	−.31 (.16)*	.32 (.04)***	---

Note. MZ and DZ represented monozygotic and dizygotic twin pairs, respectively. The former share 100% of their genetic material, whereas the latter share, on average, 50% of their segregating genetic material. Results from the first or no covariate model are also presented in Figure 2. To facilitate interpretation of these unstandized fixed effect estimates, the AAB extended symptom count variable was standardized to have a mean of 0 and a standard deviation of 1 prior to analysis. Between-pair effects can be used to approximate the individual-level effects. Significant within-pair estimates for MZ twins are indicative of a non-shared environmentally-mediated relationship between marriage and AAB, and particularly so when the DZ within-pair estimate is equivalent to the MZ estimate. Because AAB decreases with the advent of marriage, both the between- and within-pair effect estimates are negative. The prior AAB covariates, by contrast, are positive, since higher levels of AAB at ages 17 or 20 predict higher levels of AAB at age 29.

*** indicates that the fixed effect estimate is statistically significant at $p < .001$.

** and * indicate that the fixed effect estimate is statistically significant at $p < .01$ and $p < .05$, respectively.