ORIGINAL ARTICLE

Does Marriage Inhibit Antisocial Behavior?

An Examination of Selection vs Causation via a Longitudinal Twin Design

S. Alexandra Burt, PhD; M. Brent Donnellan, PhD; Mikhila N. Humbad, MA; Brian M. Hicks, PhD; Matt McGue, PhD; William G. Iacono, PhD

Context: Previous 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 toward antisocial behavior are more likely to marry.

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

Design: Co-twin control analyses in a prospective twin study provided an analogue 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 because they share a common genotype and rearing environment.

Setting: General community study.

Participants: Two hundred eighty-nine male-male twin pairs (65.1% MZ) from the Minnesota Twin Family Study underwent assessment at 17, 20, 24, and 29 years of age. None of the participants were married at 17 years of age, and 2.6% were married at 20 years of age. By 29 years of age, 58.8% of the participants were or had been married.

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 at age 29 years were present even at 17 and 20 years of age, 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 following marriage 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.

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Author Affiliations: Department of Psychology, Michigan State University, East Lansing (Drs Burt and Donnellan and Ms Humbad); Department of Psychiatry, University of Michigan, Ann Arbor (Dr Hicks); and Department of Psychology, University of Minnesota, Minneapolis (Drs McGue and Iacono).

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 observed 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 directly or indirectly. Should this be true, it could provide valuable leads for enhancing treatment development. In particular, more concrete iden-

tification 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 ago).[6] In this scenario, men who are less inclined toward antisocial behavior (for whatever reason) are more likely to marry. For example, there is a well-known association between antisocial behavior and low socioeconomic status,[7] the latter of which also predicts marriage (in the form of economic potential).[8] Selection is thus a key confounder in studies suggesting that marriage causally contributes to desistence from antisocial behavior.[6]

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 method.[2,4] The counterfactual method[9,10] 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 compared with the same person's outcome when not married. Because simultaneous observations of these 2 outcomes are impossible, however, the counterfactual method places the problem of causal inference within a general missing data framework. Specifically, researchers would estimate the missing nonmarried 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 (MZ) 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 present study sought to do just this, examining a sample of male-male twin pairs assessed at ages 17, 20, 24, and 29 years. Data were analyzed using an analogue 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 present 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 (188 MZ and 101 dizygotic [DZ]). Participants were roughly 17 years of age 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 (82.7%), 495 (85.6%), and 532 (92.0%) participants completed assessments at ages 20, 24, and 29 years, respectively. Moreover, those who completed at least 1 follow-up assessment reported levels of antisocial behavior that were equivalent to those who participated only at intake (1.08 vs 0.92 symptoms of antisocial behavior; as defined in the "Measures" section, $P = .81$).

ZYGOSITY DETERMINATION

Zygosity was determined by the agreement of the following 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 3 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 underwent in-person assessment for *DSM-III-R* mental disorders (the manual that was current at the onset of the study) by trained interviewers with bachelor's or master's degrees at all 4 visits. The reporting period was lifetime (ie, since age 15 years, per the *DSM-III-R* adult antisocial behavior [AAB] criteria) at ages 17 and 20 years. The interviews at ages 24 and 29 years assessed symptoms present since the previous visit. Each twin within a pair was interviewed by a different interviewer. Adult antisocial behavior was operationalized as a tally of endorsed and partially endorsed criterion C symptoms of *DSM-III-R* antisocial personality disorder (ASPD; eg, repeated illegal acts, irritability and aggressiveness, disregard for the truth, and lack of remorse). Symptoms were assessed via the Structured Clinical Interview for personality disorders.[15] Although AAB does not constitute a *DSM* diagnosis (ASPD diagnoses also require ≥3 criterion A symptoms of conduct disorder), if 3 or 4 symptoms are used to define a diagnosis of AAB, the κ interrater reliability exceeds 0.78. Roughly 4% of the sample (3.9%) met full criteria for lifetime ASPD. Another 4.3% were 1 symptom shy of a full ASPD diagnosis.

After the interview, a clinical case conference was held in which the evidence for every symptom was discussed by at least 2 advanced clinical psychology doctoral students (neither of whom conducted the interview). Because 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 (ie, they were clinically significant in severity and frequency) were counted as 1 full symptom. Symptoms judged to be probably present (ie, they were clinically significant in severity or frequency, but not both) were counted as half a symptom. Symptom counts, rather than diagnoses, were used primarily to increase statistical power because diagnostic prevalence rates of ASPD in community-based samples are lower than rates in clinically referred samples. To adjust for positive skew, all AAB symptom counts were log-transformed before analysis.

Marital History

Marital history was assessed at 29 years of age via a life events questionnaire,[16] 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 (2.6%) 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 by the time of their age 24 assessment). Because we made use of current marital status for our analyses, these 18 men were coded as unmarried at 29 years of age (although several of them were coded as married at 24 years of age). 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 MZ twins share 100% of their genetic material, 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 these person-specific environmental influences and 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 marital status at 29 years of age between and within persons. We next evaluated these associations within sibling pairs using an analogue 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 jth twin (j=1,2) in the ith 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

$$y_{ij} = \beta_0 + \beta_1 x_{ij} + \varepsilon_{ij},$$

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 2 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

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

where $\bar{x}_{i.}$ is the mean exposure index for the ith 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.

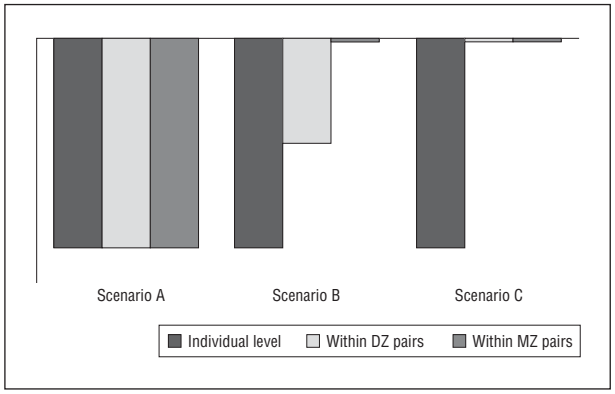

Figure 1. Interpretation of co-twin control results. Should marriage be environmentally or causally linked to reductions in antisocial behavior, we would expect to observe this association at the individual level, within dizygotic (DZ) twin pairs discordant for exposure and within monozygotic (MZ) twin 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.

This regression model can be further conceptualized within a genetically informed design.[12,18] Individual-level associations reflect potential confounding of genetic effects, shared or familial environmental effects, and nonshared or person-specific environmental effects. Associations within DZ twin pairs discordant for exposure control for shared environmental effects and partially for genetic effects. Associations within MZ twin pairs discordant for exposure control for shared environmental and genetic effects. Any remaining associations within discordant MZ pairs therefore directly index nonshared environmental effects. Accordingly, should marriage be environmentally or causally linked to reductions in AAB, 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 (**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 (ie, the process is mediated by genetic and/or shared environmental effects rather than nonshared 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[18] estimated the heritability of AAB at approximately 50%, with the remaining variance attributable to the nonshared environment. Moreover, changes in AAB over time (which would necessarily include desistence from AAB) were also found to be primarily nonshared 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 in SPSS software.[19] Because multilevel modeling coefficients are unstandardized, we standardized our outcome variable for the multilevel modeling analyses (ie,

Table 1. Mean AAB Extended Symptom Count by Marital Status at 29 Years of Age

AAB by Age, y	Status at Age 29 y, Mean (SD) No. of Symptoms		Cohen's d Effect Size
	Unmarried	Married	
17	1.08 (1.38)	0.75 (1.17)	0.26[a]
20	1.48 (1.45)	1.18 (1.30)	0.22[b]
24	1.42 (1.19)	1.04 (1.03)	0.34[a]
29	1.29 (1.06)	0.83 (0.83)	0.48[a]

Abbreviation: AAB, adult antisocial behavior.
[a] Mean difference in AAB across marital status is statistically significant at $P<.01$.
[b] Mean difference in AAB across marital status is statistically significant at $P<.05$.

log-transformed AAB at 29 years of age) to have a mean of zero and an SD 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 because 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 marital status at 29 years of age across all waves of data. The effect was particularly pronounced (and medium in magnitude[20]) for AAB at 29 years of age, but was also relatively pronounced at 24 years of age. At ages 17 and 20 years, the effect sizes were small. Such findings circumstantially suggest that the impact of marriage on AAB may be more pronounced following the marriage. More important, however, the finding that levels of AAB differed well before marriage is noteworthy because it suggests that adolescents who would later be married engaged in lower levels of AAB than did adolescents who remained unmarried at 29 years of age.

Although we knew 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 17 years of age 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 2 to 3 years[21] and repeated our analyses at 17 years of age, omitting those men married before their age 24 assessment (ie, we compared men married at ≥25 years of age with those who remained unmarried). Results were fully replicated. Married men again reported fewer AAB symptoms at 17 years of age (mean [SD] number of symptoms, 0.67 [1.10]) than did men who remained unmarried (1.02 [1.33]; $P=.004$; Cohen's d standardized effect size, 0.29). Expected mean differences were also present at ages 20 years (d=0.21), 24 years (d=0.29), and 29 years (d=0.41; all $P<.05$). In short, even when the time lag between marriage and the intake assessment was 8 years or longer, 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 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 years regardless of later marital status (both, $P<.001$). Such results are consistent with a normative increase in AAB during late adolescence.[22,23] After this increase, however, levels of AAB remained essentially constant for those who remained unmarried (means at ages 20 and 24 years and at ages 24 and 29 years 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 years ($P=.47$) and then decreased significantly from ages 24 to 29 years ($P=.001$). 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 analyses of married men to those who were married between their age 20 and 24 assessments, we found that mean levels of AAB symptoms decreased from ages 20 to 24 years (mean [SD], 1.21 [1.31] at 20 years vs 0.88 [0.87] at 24 years; $P=.008$). Thus, there appears to be a within-persons effect of marriage on desistence from AAB.

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 results of the between-persons analyses. 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 marriage inhibits AAB. Nevertheless, 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 (no-covariates) model[24] ($P=.038$ [1-tailed], 1 tailed, because we were explicitly testing whether the 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 preexisting levels of AAB by making use of our longitudinal data. We thus reran the co-twin control analyses, including reports of lifetime AAB before marriage (ie, at ages 17 or 20 years) as covariates. Regardless of whether we controlled for AAB at age 17 or 20 years, results were

Table 2. Co-twin Control Analyses Evaluating the Association Between AAB Extended Symptom Counts and Marital Status at 29 Years of Age[a]

	Fixed-Effect Estimate (SE)					
	Between-Pair		Within-Pair		Prior AAB by Age, y	
AAB at Age 29 y	MZ Twins	DZ Twins	MZ Twins	DZ Twins	17	20
No-covariate model	–0.57 (0.15)[b]	–0.57 (0.23)[b]	–0.26 (0.14)[c]	–0.29 (0.17)[c]		
Controlling for AAB at age 20 y	–0.36 (0.14)[b]	–0.55 (0.21)[b]	–0.24 (0.15)[d]	–0.25 (0.18)[d]		0.68 (0.07)[b]
Controlling for AAB at age 17 y	–0.43 (0.14)[b]	–0.46 (0.21)[b]	–0.18 (0.14)[d]	–0.31 (0.16)[d]	0.32 (0.04)[b]	

Abbreviations: AAB, adult antisocial behavior; DZ, dizygotic; MZ, monozygotic.

[a] The MZ twin pairs share 100% of their genetic material, whereas the DZ twin pairs share, on average, 50% of their segregating genetic material. Results from the no-covariate model are also presented in Figure 2. To facilitate interpretation of these unstandardized fixed-effect estimates, the AAB extended symptom-count variable was standardized to have a mean of 0 and an SD of 1.0 before analysis. Between-pair effects can be used to approximate the individual-level effects. Significant within-pair estimates for MZ twins are indicative of a nonshared 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, the between- and within-pair effect estimates are negative. The previous AAB covariates, by contrast, are positive because higher levels of AAB at age 17 or 20 years predict higher levels of AAB at age 29 years.

[b] The fixed-effect estimate is statistically significant at $P < .001$.

[c] The fixed-effect estimate is statistically significant at $P < .01$.

[d] The fixed-effect estimate is statistically significant at $P < .05$.

quite similar to those in the previous paragraph. 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 reran analyses examining whether marital status at 24 years of age predicted AAB at the same age, even when controlling for AAB at ages 17 or 20 years. The within-MZ effect of marriage on AAB remained statistically significant (fixed effect estimate [SE], –0.28 [0.18]; $P = .027$) and was not statistically different from that in the DZ pairs ($P = .69$), even when controlling for AAB at 17 years of age. Controlling for AAB at 20 years of age yielded similar results; the within-MZ effect of marriage on AAB was statistically significant (fixed effect estimate [SE], –0.29 [0.17]; $P = .037$) and was not statistically different from that in the DZ pairs ($P = .83$).

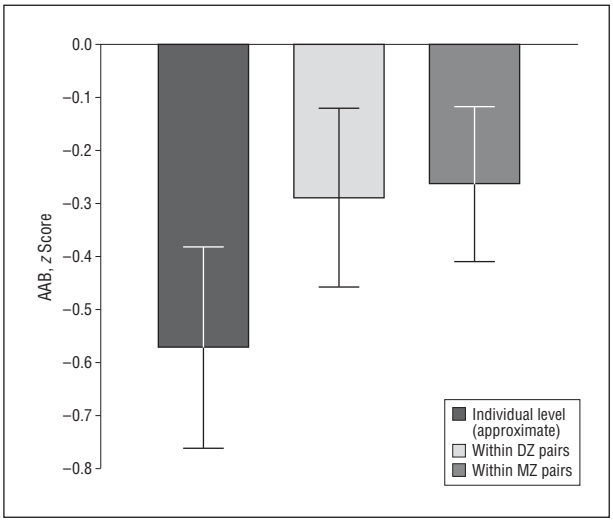

Figure 2. Adult antisocial behavior (AAB) by marital status at 29 years of age. To facilitate interpretation of these unstandardized fixed-effect estimates (as also presented in Table 2), the AAB extended symptom-count variable was standardized to have a mean of 0 and an SD of 1.0 before analysis. Marriage and AAB were assessed at age 29 years. Standard error bars are presented. The between-pair effect estimate, which approximates the individual-level effect, is also presented. Significant within-pair estimates for monozygotic (MZ) twins are indicative of a nonshared environmentally mediated relationship between marriage and AAB and particularly so when the dizygotic (DZ) within-pair estimate is equivalent to the MZ estimate.

SUPPLEMENTAL ANALYSES

To maintain consistency across assessments, we used the *DSM-III-R* criteria for AAB in these 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 29 years of age as our outcome variable. The between-pair effect estimates were large and significant (fixed effect estimate [SE], –0.56 [0.15] for MZ twins and –0.54 [0.23] for DZ twins; $P < .01$). The within-pair estimates were also statistically significant (fixed-effect estimate [SE], –0.26 [0.15] for MZ twins and –0.25 [0.17] 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 also extend to *DSM-IV* AAB.

COMMENT

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 undergoing assessment up to 4 times between the ages of 17 and 29 years. Analyses offered support for both selection and causation explanations. Specifically, mean differences in AAB across married and unmarried men preceded the state of marriage by many years and did so even when we restricted the married sample to those who married at 25 years or older. Moreover, there was some evidence of selection in our co-twin control analyses because 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 remained unmarried at 29 years of age. However, visual inspection of the mean differences was also consistent with the possibility that entrance into the state of marriage may accentuate these preexisting differences. These suspicions were borne out in our co-twin control analyses. At ages 24 and 29 years, the within-pair effect of marriage on AAB was statistically significant for MZ twins. Because MZ twins share all their genes and early rearing environment, such results are indicative of a person-specific or nonshared 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, these results indicate that, although men with lower levels of AAB are more likely to marry by 29 years of age, entrance into the state of marriage accentuates their tendency to refrain from AAB.

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 AAB. 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 29 years of age, the Cohen's d effect size for differences in AAB by marital status was 0.48, which corresponds to slightly more than a 30% reduction in AAB with marriage.

Nevertheless, our results also implicated the presence of selection processes such that men who married by 29 years of age were less prone to antisocial behavior as adolescents than were their unmarried peers. Other studies, by contrast, have found little evidence in support of selection.[2,3] Although it is not clear what may account for this difference across studies, one possibility is clinical severity. Previous work has often examined high-risk/criminologic samples (eg, delinquent boys who had been committed to reform schools during adolescence[2,3]), whereas the present 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 present sample was born between 1972 and 1978, whereas the aforementioned high-risk sample[2,3] was born between 1924 and 1932. These cohort differences may be particularly salient in the present study given changes in the frequency and psychological meaning of marriage since the 1960s and 1970s.[25] Indeed, the proportion of never-married individuals has steadily increased since the 1970s, as has the median age at first marriage.[26] Because 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 previous decades.

There are several limitations to bear in mind when interpreting the results of this study. First, only men were examined because the link between marriage and desistence from antisocial behavior among women has been less consistently supported.[2,27] 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 (eg, more antisocial men would marry more antisocial women[28]), this process was not examined herein because 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.

These results apply only to early adulthood and not to later developmental periods. This point is particularly salient because it is likely that many of the men who were unmarried as of their age 29 assessment will eventually marry.[17] However, because antisocial behavior is more common in early adulthood than in later developmental periods,[29,30] early adulthood is a critical time to investigate predictors of antisocial behavior. It is also unclear whether the effects identified herein are specific to marriage or whether they extend to other committed romantic states (ie, engagement or cohabitation). We would expect our findings to generalize beyond marriage because the presumed mediators of these effects (eg, social control) should generalize to other sorts of romantic bonds. That said, at least 1 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. Even so, evidence indicating that the state of marriage inhibits antisocial behavior has also been found in high-risk/criminologic samples likely to contain a larger 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.

Despite these limitations, the current results provide an important constructive replication and extension of previous 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 other scholars,[2] however, it seems unlikely that the institution of marriage inhibits men's antisocial behavior directly; rather, marriage is likely a marker for other more proximal and causal processes. For example, previous 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 29 years of age). There are many possible explanations for such findings.[6] Perhaps 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 possibility 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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Correspondence: S. Alexandra Burt, PhD, Department of Psychology, Michigan State University, 107D Psychology Bldg, East Lansing, MI 48824 (burts@msu.edu).
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