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## Accounting for the Physical and Mental Health Benefits of Entry Into Marriage: A Genetically Informed Study of Selection and Causation

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### Abstract

Married adults show better psychological adjustment and physical health than their separated/divorced or never-married counterparts. However, this apparent “marriage benefit” may be due to social selection, social causation, or both processes. Genetically informed research designs offer critical advantages for helping to disentangle selection from causation by controlling for measured and *unmeasured* genetic and shared environmental selection. Using young-adult twin and sibling pairs from the *National Longitudinal Study of Adolescent Health* (Harris, 2009), we conducted genetically informed analyses of the association between entry into marriage, cohabitation, or singlehood and multiple indices of psychological and physical health. The relation between physical health and marriage was completely explained by nonrandom selection. For internalizing behaviors, selection did not fully explain the benefits of marriage or cohabitation relative to being single, whereas for externalizing symptoms, marriage predicted benefits over cohabitation. The genetically informed approach provides perhaps the strongest nonexperimental evidence that these observed effects are causal.

### Keywords

marriage; cohabitation; selection; internalizing; externalizing

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Empirical research consistently demonstrates that married individuals are better adjusted psychologically (e.g., Wade & Pevalin, 2004), healthier physically (e.g., Hughes & Waite, 2009; Dupre & Meadows, 2007), and less prone to engaging in risky behaviors (e.g., Horwitz & White, 1991; Power, Rodgers, & Hope, 1999). However, skeptics question how this correlation should be interpreted. A recent essay, “What if marriage is bad for us?” represents the unconvinced: “To say marriage creates wealth is to confuse correlation with causation. If there is more wealth in Manhattan than in Brooklyn, that does not mean that moving to Manhattan will make you wealthier” (Essig & Owens, 2009, p. 1).

### Social Selection Versus Social Causation

There is little doubt that married individuals enjoy more psychological and physical health benefits than their unmarried counterparts, but it is uncertain whether this “marriage benefit” is the result of social selection or social causation (Carr & Springer, 2010). The *social selection* hypothesis posits that better-adjusted, healthier individuals become and remain married, and that this selection effect accounts for observed group differences between

married and unmarried individuals. Some empirical evidence supports this hypothesis. For example, psychological well-being strongly predicts the probability of later marriage (Mastekaasa, 1992; Stutzer & Frey, 2006), as do lower psychological distress (Hope, Rodgers, & Powers, 1999), fewer alcohol problems (Horwitz & White, 1991), and lower levels of antisocial behavior (Burt et al., 2010; Barnes & Beaver, 2012). On the other hand, the *social causation* hypothesis suggests that something about marriage causes positive changes and/or protects against negative changes in mental or physical health. However, the social causation hypothesis is a challenge to support unequivocally, because it is practically and ethically impossible to use experimental methods to study the effects of marriage (i.e., we cannot randomly assign couples to marry or divorce). In nonexperimental designs, it always remains conceptually possible that some unknown or uncontrolled selection factor accounts for group differences.

### Selection and Causation Revisited

Causal effects of marriage are often assumed after statistically adjusting for factors known to be associated with selection into marriage. However, traditional social science studies are limited to controlling for measured covariates, typically only environmental ones. Genetically informed family designs, in contrast, control for all genetic selection effects, as well as many *unmeasured* environmental selection effects.

Genetically informed research designs, and twin and sibling studies in particular, offer traditional correlational studies an additional layer of control for parsing selection from causation. By using sibling pairs of varying degrees of genetic relatedness who have been reared together, it is possible to examine a phenotypic—or observed—relationship *after* taking into account genetic and shared environmental confounds (Turkheimer & Harden, in press; Turkheimer & Waldron, 2000). For example, any observed difference between monozygotic (MZ; identical) twins discordant for a life experience (e.g., marital status) cannot be attributable to genetic or shared environmental selection (D’Onofrio et al., 2005; Kendler et al., 1993), and therefore must be the result of environmental factors not shared by the twins (i.e., that life experience for which they are discordant—marital status).

To illustrate, consider an identical twin pair and a fraternal (dizygotic; DZ) twin pair, each discordant for marital status. Suppose these twins inherited genetic characteristics that increase both their risk for experiencing depressive symptoms and their choice to not marry. Despite observing a relationship between marital status and depression at the population level, we would find no relationship when comparing the MZ twins (i.e., the married member of the pair would not be less depressed than her unmarried cotwin, because the relation is genetically mediated).

Suppose now that growing up, these twin pairs were exposed to environmental factors—such as socioeconomic status, parental divorce, or neighborhood characteristics—that are related to both depression and propensity to marry. A population-level relationship would again be observed, but in this instance it would be caused by familial experiences shared between twins. In this case, we would find no differences in depression associated with marital status when comparing either the MZ or the DZ twins, because the relation is mediated by the environment shared between members of sibling pairs.

Finally, suppose that marital status *is* causally related to depression. In this case, nonshared experience (either marriage or something correlated with marriage within sibling pairs) accounts for differences in outcomes, even after controlling for genetic and environmental confounds shared by members of the same family. We would find that the married MZ and DZ co-twins were better adjusted than their unmarried counterparts (although differences

may be larger for the DZ than the MZ pair, if genetic selection partially accounted for the effect).

In pointing to the benefits of genetically informed designs, we note that observing a significant phenotypic association after controlling for genetic and shared environmental confounds is consistent with a causal relationship. It is not, however, probative, because it is possible that other nonshared environmental factors are responsible for the observed effect (e.g., one twin may have pursued more postsecondary education, which tends to be related to both greater marital stability and less depression). Although such possibilities exist, by controlling for all possible genetic and shared environmental confounds—measured or *unmeasured*—twin studies provide a rigorous test of whether an observed effect is due to selection. Thus, we use the term *quasi-causal* to describe a phenotypic association that remains significant after these confounds have been controlled.

### Genetically Informed Research on the Benefits of Marriage

Among the few genetically informed studies of marital status and its correlates completed to date, a few support the selection hypothesis. In a study of middle-aged Danish twins, Osler, McGue, Lund, and Christensen (2008) observed genetic and shared environmental selection into both marriage and divorce across several physical and psychological health outcomes, including depression and self-rated health. Similarly, Johnson, McGue, Krueger, and Bouchard (2004) demonstrated that personality factors and propensity to marry share common genetic influences. On the other hand, some genetically informed studies demonstrate evidence for causation. Barnes and Beaver (2012) and Burt et al. (2010) demonstrated, in separate samples, that marriage predicts desistance from delinquency after taking into account between-family confounds. Prescott and Kendler (2001) observed reduced alcohol consumption following marriage in a sample of female twins. Likewise, in a matched case-sibling control study, Agerbo, Qin, and Mortensen (2006) found risk ratios for completed suicide to be comparable between siblings and controls discordant for marital status. Heath, Eaves, and Martin (1998) demonstrated that being in a marriage-like relationship (marriage or cohabitation) mitigates the impact of genetic liability to depression.

### Entry into Marriage or Cohabitation and the Present Study

Benefits associated with marriage are generally more pronounced for relationship dissolution than for entry into marriage (Hope, Rodgers, & Powers, 1999), and research often focuses on exits from marriage while neglecting union formation. As a result, it is difficult to know whether getting married is beneficial, or whether getting divorced is detrimental (or both). For example, substantial negative differences in mental health have been demonstrated repeatedly when comparing married and divorced adults (e.g., Booth & Amato, 1991; Hope et al., 1999; Wade & Pevalin, 2004), but analogous research on union formation is sparse. Furthermore, selection effects often account for the observed benefits of entry into marriage. Still, after accounting for propensity to marry, entry into marriage is associated with less depression (Lamb, Lee, & DeMaris, 2003; Simon, 2002), psychological distress (Strohschein, McDonough, Monette, & Shao, 2005), alcohol abuse (Power et al., 1999; Simon, 2002), and antisocial behavior (King, Massoglia, & Macmillan, 2007; Burt et al., 2010; Barnes & Beaver, 2012).

Another question to consider is whether legal marriage confers benefits over cohabitation, a step toward or alternative to marriage that has grown common in the United States and throughout much of the industrialized world. It follows, therefore, that a rigorous test of the marriage benefit is the comparison of never-married, cohabiting, and married young adults (excluding married individuals who have divorced). In the present study, we make a series

of comparisons between these groups to inform whether marriage confers psychological and physical health benefits. We build upon and extend existing research of the marriage benefit, reporting one of the first behavior genetic studies of marriage in early adulthood, and the first genetically informed study to explicitly examine the effect of cohabitation on benefits typically associated with marriage.

Using a nationally representative sample of young adults in the United States, we use biometric quasi-causal modeling techniques to examine levels of internalizing, externalizing, and physical-health behaviors commonly associated with marital status. First, we compare coupled young adults (combining married and cohabiting individuals—what we call a “marriage-like relationship”) with single young adults. We predicted that a benefit of a marriage-like relationship would exist even after adjusting for self-selection (due to prior health levels, as is practiced in traditional correlational studies) and selection effects due to genetic or shared environmental factors. Second, to create a more robust test of the marriage benefit, we compared married young adults with cohabiting young adults to test the hypothesis that marriage confers greater benefits than cohabiting relationships after adjusting for selection factors. Consistent with previous research contrasting the correlates of cohabitation with those of marriage (Brown, 2000; Marcussen, 2005; Wu & Hart, 2002), we predicted that the benefits of marriage-like relationships would be greater for married individuals.

## Method

### Sample

Data were obtained from the National Longitudinal Study of Adolescent Health (Add Health; Harris, 2009), a nationally representative sample of young adults in the United States. This ongoing longitudinal study includes four complete waves of data collected between 1994 and 2009; details of data collection and survey procedures are described elsewhere (Harris et al., 2009). The present study uses data collected from 3,226 (1,681 females) members of the Add Health genetic subsample (described below), interviewed during Wave III (between August, 2001 and April, 2002) and Wave IV (between January, 2008 and February, 2009). This subsample is diverse, with 54.4% identifying as non-Hispanic Caucasian, 22.1% as African American, 4.8% as Asian American, and 18.7% as bi- or multiracial or other ethnicity. All respondents at this time had reached early adulthood (mean age = 28.88 years;  $SD = 1.74$  years, range = 24–34), making it suitable for studying physical and psychological costs and benefits associated with entry into cohabitation and marriage. Because marital dissolution is reliably associated with increased distress (e.g., Booth & Amato, 1991; Wade & Pevalin, 2004), respondents who reported being married at Wave III but not at Wave IV (2.9% of the genetic subsample) were excluded from analyses to eliminate the possibility that combining divorced and single individuals could inflate negative effects associated with being single. We also excluded respondents who described themselves as homosexual (1.9% of the genetic subsample). Romantic relationships of same-sex and heterosexual couples do not differ in terms of well-being (e.g., Kurdek, 2005), yet it is unclear how these individuals should be grouped regarding marital status. The legal recognition of same-sex marriage in the United States is varied, and for many lesbian or gay couples, cohabitation may be more a necessity than a choice; for many of these respondents, marriage may not be an option at all.

The Add Health genetic sample is composed of participants identified as monozygotic twin pairs (MZ), dizygotic twin pairs (DZ), full biological siblings (FS), half siblings (HS), cousins (CO), and genetically unrelated siblings (NR;  $n = 3,139$  pairs). Twins' zygosity was determined primarily on the basis of four self-report items concerning similarity of physical features and frequency with which one twin is confused for the other. The questions are

standard nonserological determinates of zygosity and demonstrate high validity (greater than 90% accuracy) when compared with DNA-determined zygosity (Loehlin & Nichols, 1976; Spitz et al., 1996). Pairs of undetermined zygosity were excluded from analysis except in cases where DNA was used to identify twins as MZ or DZ (Harris et al., 2009). To ensure no families were overrepresented, one sibling pair from each family was randomly selected for inclusion in statistical analyses. Overall there were 171 MZ pairs, 264 DZ pairs, 712 FS pairs, 213 HS pairs, 93 CO pairs, and 160 NR pairs included in the analyses, for a total of 1,613 sibling pairs.

## Measures

**Marital status**—Marital status was determined from a household roster completed by respondents. Similar procedures for determining family structure in the Add Health sample have been used elsewhere (Peris & Emery, 2004). Respondents identified individuals with whom they lived as (a) husband/wife; (b) partner/boyfriend/girlfriend; or (c) one of 17 other types of family member (e.g., parent, sibling, grandparent, etc.). We made two orthogonal contrasts in the current analyses. For the first contrast, respondents indicating residence with a spouse or romantic partner were coded as *coupled* (1); remaining respondents were coded as *single* (0). For the second contrast, those reporting living with a spouse were coded as *married* (1); respondents reporting living with a romantic partner were coded as *cohabiting* (0).

Approximately 40.9% of the sample was single, 17.4% cohabiting, and 41.7% married. There were no significant age differences between coupled ( $M = 28.85$ ,  $SD = 1.74$ ) and single ( $M = 28.92$ ,  $SD = 1.74$ ) individuals ( $t = -.997$ ,  $df = 3220$ ,  $p = .319$ ), or between married ( $M = 28.85$ ,  $SD = 1.76$ ) and cohabiting ( $M = 28.85$ ,  $SD = 1.69$ ) individuals ( $t = -.003$ ,  $df = 1903$ ,  $p = .998$ ). No gender differences existed in the coupled–single classification ( $\chi^2 = .778$ ,  $df = 1$ ,  $p = .378$ ), but women were more likely to be married (54.6% vs. 45.3% of males) in the married–cohabiting marital-status classification ( $\chi^2 = 5.894$ ,  $df = 1$ ,  $p = .015$ ). Marital status did not differ by ethnicity (European American vs. ethnic minority) for either the coupled–single classification ( $\chi^2 = 1.749$ ,  $df = 1$ ,  $p = .186$ ) or the married–cohabiting distinction ( $\chi^2 = .001$ ,  $df = 1$ ,  $p = .976$ ).

**Internalizing behavior**—We used two measures to operationalize internalizing behavior: depressive symptom count and endorsement of suicidal ideation. Depressive symptom count was assessed using nine items from the Center for Epidemiological Studies Depression Scale (CES-D; Radloff, 1977). The original 20-item CES-D Scale is a reliable measure of the frequency of depressive symptoms in young-adult samples (Radloff, 1991). Respondents indicated the extent to which they experienced a cluster of depressive symptoms (bothered by things, could not shake off blues, felt just as good as others, trouble keeping mind on things, felt depressed, too tired to do things, enjoyed life, felt sad, and people disliked me) during the past seven days: *never or rarely* (0), *sometimes* (1), *a lot of the time* (2), *most of the time or all of the time* (3). Cronbach's  $\alpha$  and McDonald's  $\omega$  (McDonald, 1999) for these items (after reverse coding where appropriate) demonstrated adequate internal consistency ( $\alpha_{\text{Wave3}} = .81$ ,  $\omega_{\text{Wave3}} = .74$ ;  $\alpha_{\text{Wave4}} = .82$ ,  $\omega_{\text{Wave4}} = .75$ ). We created latent scores composed of these items, and maintained structural invariance across waves. Respondents also indicated whether they had seriously thought about committing suicide in the 12 months prior to data collection (*no* = 0; *yes* = 1). Approximately 6.5% of the sample reported having such thoughts during the past year at Wave III and 7.1% at Wave IV.

**Externalizing behavior**—We used two measures to operationalize externalizing behavior: alcohol use and antisocial behavior. A latent variable comprised of four drinking items was created to examine alcohol use, maintaining structural invariance across waves.

Respondents indicated their frequency of drinking, heavy drinking (defined as five drinks or more in a row for males, four drinks for females), and drunkenness during the 12 months prior to data collection: *never* (1), *once or twice* (2), *once a month or less* (3), *2 or 3 days a month* (4), *1 or 2 days a week* (5), *3 to 5 days a week* (6), *every day or almost every day* (7). Respondents also indicated how many drinks they typically consumed on each occasion. Cronbach's  $\alpha$  and McDonald's  $\omega$  for these items indicated adequate reliability ( $\alpha_{\text{Wave3}} = .89$ ,  $\omega_{\text{Wave3}} = .88$ ;  $\alpha_{\text{Wave4}} = .88$ ,  $\omega_{\text{Wave4}} = .87$ ).

To assess antisocial behavior, a dichotomous variable was created indicating *none* (0) or *any* (1) participation in any of 10 criminal acts during the 12 months prior to data collection: damage property, petty theft, burglary, threaten another with a weapon, sell drugs, grand theft, gang fighting, buy/sell stolen goods, and write a bad check. Approximately 25.8% of the sample reported committing illegal acts during the past year at Wave III and 14.8% at Wave IV.

**Health behavior**—We used two measures to operationalize health behavior: subjective physical health and cigarette use. Respondents rated their general health as *excellent* (5), *very good* (4), *good* (3), *fair* (2), or *poor* (1;  $M_{\text{Wave3}} = 3.99$ ,  $SD_{\text{Wave3}} = .87$ ;  $M_{\text{Wave4}} = 3.63$ ,  $SD_{\text{Wave4}} = .93$ ). This single-item measure of health is considered a “gold standard” of overall health and has been widely used in psychological research (Idler & Benyamini, 1997). It is established as a valid measure of mortality, and tends to be superior even to objective physician ratings in predicting mortality (Ferraro & Farmer, 1999; Idler & Benyamini, 1997). Respondents also reported the number of days in the past month they smoked and the number of cigarettes smoked on each occasion. From this, we created a measure of cigarettes smoked per day ( $M_{\text{Wave3}} = 3.46$ ,  $SD_{\text{Wave3}} = 7.23$ ;  $M_{\text{Wave4}} = 3.48$ ,  $SD_{\text{Wave4}} = 7.37$ ).

## Statistical Analyses

For all outcomes in each orthogonal contrast, we fit two structural equation models to the data using the robust weighted least-squares (WLSMV) estimation option in the structural equation modeling software Mplus v. 6.0 (Muthén & Muthén, 2010b). WLSMV is a pairwise present estimation method, and assumes data to be missing completely at random (Muthén & Muthén, 2010a). Bias-corrected bootstrapped 95% confidence intervals around parameter estimates were computed using 1,000 independent resamplings from the data. These confidence intervals are corrected for nonnormality of parameter-estimate distributions, and as such, may not be symmetric around parameter estimates (Muthén & Muthén, 2010a). Goodness of fit was assessed using the root mean squared error of approximation (RMSEA; Browne & Cudeck, 1993). Values below .05 indicate close fit, and values up to .08 represent reasonable errors of approximation (Steiger, 1990). We also report values for the comparative fit index (CFI; Bentler, 1990) and the Tucker-Lewis index (TLI; Bentler, 1990). Values .95–1.00 indicate good fit (Hu & Bentler, 1999).

In biometric models with twin and sibling pairs, the variance of a variable is partitioned into three latent constructs: additive genetic (A), shared environmental (C), and nonshared environmental (E). Variance decomposition is achieved by constraining the extent to which twins covary on these components. The additive genetic components (A) correlate at ratios appropriate for the proportion of segregating genes shared by sibling dyads ( $r = 1.0$  for MZ twins, 0.5 for DZ twins and full siblings, 0.25 for half siblings, and so on). The shared environmental components (C) correlate at unity for all pairs, since this represents environmental experiences shared by members of a sibling pair. The nonshared environmental components (E) comprise experiences and environments unique to an individual, and thus are not permitted to correlate between siblings. To provide sufficient

degrees of freedom to estimate between- and within-pair variance parameters, we fixed thresholds of marital status and binary outcomes to empirically estimated values (see Prescott, 2004). Means and variances of continuous measures were estimated in the same model.

Just as individual indicators (single variables) have their own ACE components, correlated indicators (e.g., the relation between marital status and mental or physical health) may share variance attributable to genes or shared environments, and this covariance may spuriously inflate their phenotypic relationship. The greater the extent to which variables covary through A or C, the more their true relationship is inflated. The effect of marital status on mental or physical health may operate through any ACE variance components, thus A and C must be controlled for to get an unbiased estimate of the marriage benefit.

Controlling for the between-family variables A and C involves a simple mediation analysis (Baron & Kenny, 1986). First, an outcome is regressed onto marital status to get an estimate of its total effect (parameter  $b_{phen}$  in Figure 1a). The outcome is then simultaneously regressed onto both marital status and its between-family (A and C) variance components (see Figure 1b). The direct effect of marital status on the outcome (parameter  $b'_{phen}$  in Figure 1b) is examined while accounting for its indirect effects (parameters  $b_A$  and  $b_C$  in Figure 1b). A quasi-causal effect of marital status on the outcome is indicated when its direct effect (parameter  $b'_{phen}$ ) is significantly different from zero, holding constant its indirect effects (parameters  $b_A$  and  $b_C$ ; Turkheimer & Harden, in press). In other words, when genes and the shared environment do not fully mediate the relation between marital status and the phenotype, the remaining association cannot be due to genetic or environmental selection, and the social causation explanation is supported. On the other hand, nonrandom selection into marriage is supported if the direct effect of marital status on the phenotype ( $b'_{phen}$ ) is no longer statistically different from zero when the mediators (A and C) have been included in the model.<sup>1</sup> If genes and environments shared between siblings fully mediate the relation between marital status and the phenotype, the relation is explained by familial selection effects rather than unique experience (E), and causation is ruled out.

As is often done in traditional correlational studies, in all analyses we included gender (0 = *female*, 1 = *male*), ethnicity (*European American* = 0, *ethnic minority* = 1), mean-centered age, and Wave III scores as covariates. The first model for each outcome was a simple linear (probit where appropriate) regression of phenotype onto marital status (as in Figure 1a). We then fit a mediation model to each outcome demonstrating a statistically significant phenotypic effect.

## Results

### Coupled Versus Single

**Phenotypic models**—To estimate the total effect of marital status on a given phenotype, each phenotype was first regressed onto the coupled–single marital status classification, controlling for gender, ethnicity, age, and Wave III score. These estimates and their corresponding bootstrapped standard errors are presented in Table 1. Compared with singlehood, marriage or cohabitation was significantly associated with less self-reported depression ( $b_{phen} = -.050$ , 95% CI =  $-.096$  to  $-.005$ ), less alcohol use ( $b_{phen} = -.577$ , 95% CI =  $-.792$  to  $-.422$ ), fewer cigarettes smoked per day ( $b_{phen} = -1.011$ , 95% CI =  $-1.547$  to  $-.324$ ), lower risk for suicidal ideation ( $b_{phen} = -.495$ , 95% CI =  $-.743$  to  $-.296$ ), and lower

<sup>1</sup>These processes are not necessarily mutually exclusive—nonrandom selection and quasi-causation may co-occur.

risk for engaging in antisocial behavior ( $b_{phen} = -.099$ , 95% CI =  $-.216$  to  $-.012$ ). No physical health differences existed between coupled and single individuals ( $b_{phen} = -.008$ , 95% CI =  $-.102$  to  $.073$ ) after adjusting for covariates.

**Biometric models**—To estimate the direct and indirect effects of marital status on phenotype, each outcome demonstrating a statistically significant phenotypic association with marital status was regressed simultaneously onto marital status and its between-family (A and C) components. These estimates are also presented in Table 1. Compared with being single, being coupled remained statistically significantly associated with fewer depressive symptoms ( $b'_{phen} = -.113$ , 95% CI =  $-.300$  to  $-.007$ ), lower risk of suicidal ideation ( $b'_{phen} = -.926$ , 95% CI =  $-1.938$  to  $-.295$ ), and less alcohol use ( $b'_{phen} = -.688$ , 95% CI =  $-1.080$  to  $-.223$ ) after taking into account genetic and shared environmental selection effects. The significant direct effect of marital status on these outcomes is consistent with a quasi-causal relation. Direct effects of marital status on risk for antisocial behavior ( $b'_{phen} = -.158$ , 95% CI =  $-.693$  to  $.097$ ) and cigarettes smoked per day ( $b'_{phen} = -1.357$ , 95% CI =  $-4.132$  to  $.376$ ) were no longer significant, suggesting that the relationship is mediated by genetic and/or shared environmental confounds. The nonsignificant direct effect of marital status on these outcomes is evidence for nonrandom selection into marriage (or marriage-like relationships) due to genetic or shared environmental factors.

### Married Versus Cohabiting

**Phenotypic models**—Table 2 presents the results for the phenotypic regressions of each outcome onto marital status as defined by married versus cohabiting, controlling for age, gender, ethnicity, and Wave III score. Compared with cohabitation, marriage was associated with significantly lower risk for suicidal ideation ( $b_{phen} = -.590$ , 95% CI =  $-1.072$  to  $-.305$ ) and antisocial behavior ( $b_{phen} = -.332$ , 95% CI =  $-.509$  to  $-.224$ ). No phenotypic relations were observed for depressive symptom count ( $b_{phen} = .003$ , 95% CI =  $-.057$  to  $.064$ ), alcohol use ( $b_{phen} = .092$ , 95% CI =  $-.124$  to  $.643$ ), cigarette use ( $b_{phen} = -.245$ , 95% CI =  $-1.073$  to  $.812$ ), or subjective physical health ( $b_{phen} = .024$ , 95% CI =  $-.063$  to  $.113$ ), after controlling for the covariates.

**Biometric models**—Once again, we found evidence for non-random selection into marriage due to genetic and shared-environmental factors: Risk of suicidal ideation ( $b'_{phen} = .117$ , 95% CI =  $-.293$  to  $.863$ ) was no longer significantly associated with marital status after accounting for genetic and shared-environmental confounds. Consistent with the social causation hypothesis, however, marital status was quasi-causally related to antisocial behavior ( $b'_{phen} = -.326$ , 95% CI =  $-.754$  to  $-.067$ ), such that married individuals fared better than cohabiting individuals.

### Discussion

We used a genetically informed research design to provide a powerful test of whether social selection or social causation accounted for various mental and physical health benefits associated with entry into marriage and/or cohabitation. In this first genetically informed study of cohabitation, we predicted that, relative to being single, marriage or cohabitation would be associated with better psychological and physical health, and that these associations would hold after accounting for genetic and shared environmental selection. We also predicted that, after controlling for selection, marriage would confer greater mental and physical health benefits than cohabitation.



Our hypotheses were partially supported. While physical health benefits associated with marriage appeared to be due entirely to nonrandom selection into marriage, a different pattern emerged for internalizing and externalizing behaviors. Holding constant the influence of genetic and shared environmental selection into marriage, we found a quasi-causal protective effect of being coupled on self-reported depression, risk of suicidal ideation, and alcohol use. For example, married or cohabiting MZ twins had mean depression scores that were .13 *SD* lower than their single co-twins, and were just one fourth as likely to report suicidal ideation. We also observed a quasi-causal protective effect of marriage (compared with cohabitation) on risk for engaging in antisocial behavior. As a concrete example of this overall pattern, cohabiting MZ twins were 67% more likely than their married co-twins to have engaged in antisocial behavior. These latter findings extend upon previous genetically informed studies of the benefits of marriage for externalizing symptoms (Barnes & Beaver, 2012; Burt et al., 2010; Prescott & Kendler, 2001): Like marriage, it appears that cohabitation is protective against alcohol use, but is *not* protective against antisocial behavior.

The differences between internalizing and externalizing behavior are particularly noteworthy. Marriage and cohabitation both offer positive supports such as a shared emotional life, companionship, and practical assistance, all of which may lead to greater happiness and lower levels of depression. Similarly, marriage and cohabitation both protect against negative experiences such as loneliness and social isolation, factors that may increase the risk for anxiety and depression.

On the other hand, it is of considerable interest that marriage offers benefits over cohabitation in relation to externalizing problems. Young adults who marry rather than enter or maintain a cohabiting relationship exhibit a greater commitment to one another and perhaps adopt longer time horizons in relation to both their intimate partnerships and other aspects of their lives (Emery, Horn, & Beam, in press; Stanley & Markman, 1992). Delay of gratification is a well-known protective factor against externalizing behavior among children. It may well be that young people who marry similarly look further into the future in relation to their intimate partnerships, their antisocial behavior, and, we would predict, other long-term investments, ranging from buying a home to having children to managing expected life difficulties. It is also possible that husbands and wives have or assume “permission” to monitor their partner’s behavior more closely, fostering greater engagement in prosocial activities and less engagement in antisocial ones.

Although we found some important (and apparently causal) benefits associated with relationship and marital status, we expect that considerable variation exists in when and whether cohabitation or marriage benefits individual well-being. Emerging research suggests that cohabitation differs, for example, when couples cohabit with the intention of marrying versus when they do not (Rhoades, Stanley, & Markman, 2009b). Furthermore, considerable research suggests that relationship quality is a critical link between marriage and positive mental health (Brown, 2000; Gove, Hughes, & Style, 1983; Marcussen, 2005). Relationship quality may be an important moderator of the observed similarity between marriage and cohabitation, an important topic for future research, but one that is beyond the scope of the present study. Similarly, relationship duration and stability (Marcussen, 2005) may impact the relation between marriage or cohabitation and individual well-being, and perhaps account for some differences between the two statuses. Finally, the presence of children in the home can be either a positive or a negative for married or cohabiting couples, another topic for future research (Beam et al., 2011). In short, marriages and cohabiting relationships are both heterogeneous, and some forms of cohabitations apparently resemble marriage more than others. Parsing the nature of cohabiting relationships is an important

consideration for future research (see Rhoades, Stanley, & Markman, 2009a; Rhoades et al., 2009b), including in genetically informed research designs.

Given clear evidence of the long-term benefits of marriage—for example, regarding longevity (Brockmann & Klein, 2004; Sbarra, Law, & Portley, 2011)—we were somewhat surprised to find that selection effects fully accounted for the physical health “benefit” of entry into marriage. On the one hand, this finding underscores our general concern with selection effects and the benefits of using genetically informed family designs to detect and control for them. On the other hand, we recognize that the possible physical health benefits of marriage may accrue over the life course (Brockmann & Klein, 2004). Young adults generally are in good physical health in comparison with adults in later life, making any differences more difficult to detect. Moreover, we observed that being in a coupled relationship (married or cohabiting) was associated with at least one behavior that is often linked to better, long-term physical health—less alcohol use. If single individuals engage in more unhealthy behaviors—such as heavier alcohol use—we may well observe a causal effect of marital status on physical health in future waves of this sample or in other, older adult samples.

### Limitations

Genetically informed designs offer many strong methodological benefits, but we note several limitations of the present study. We did not explicitly compare marriage with singlehood (in order to conduct orthogonal contrasts), thus limiting our ability to detect direct differences between these two groups. At the same time, the inclusion of a cohabiting group allowed us to examine the experience of a substantial portion of our sample (approximately 20%) and of the United States population. Moreover, the inclusion of the cohabiting group provided a rigorous test of the causal impact of marriage per se, and our use of orthogonal contrasts kept our comparisons statistically independent. Future research, in addition to replicating the present findings, may consider alternative contrasts among these groups, for example married versus unmarried and cohabiting versus single.

We also excluded two groups from our analyses: individuals who had experienced marital dissolution and respondents endorsing homosexuality. Our rationale for excluding divorced individuals lies in our primary interest for conducting the present analyses—mental and physical health benefits associated with entry into marriage. Many of the consequences of divorce for adults are better studied later in life when many more people will have divorced, although early divorce and contemporaneous effects of divorce are both of interest to marriage benefit researchers. We did not address either topic in this report, and thus highlight that our findings are relevant for young adults in a marriage (or marriage-like relationship) who have not experienced divorce. Regarding gay and lesbian individuals, research shows that same-sex and heterosexual couples do not differ in terms of well-being (e.g., Kurdek, 2005), and investigating whether the marriage benefit extends to same-sex marriage is an important topic as same-sex marriage becomes more widely recognized. Add Health sample sizes do not allow for such analyses, however, and we believe same-sex relationships are best studied separately until they are more completely accepted and institutionalized.

Finally, we controlled for gender in our analyses rather than explicitly testing for gender-typed expressions of the marriage benefit (e.g., less depression in women vs. less alcohol use in men; Simon, 2002). Of note, in analyses we do not present here, we did not find evidence of gender differences for any of the outcomes presented in this report. However, it is possible that we may be lacking statistical power to detect such differences in this sample. Gender-specific marriage benefits should be addressed by future research on this topic.

Despite these limitations, we believe that the present study contributes to the marriage benefit literature in important ways. This is the first genetically informed study to include cohabitation as a marital status, and it is also the only genetically informed comprehensive study of the marriage benefit in young adults. The strengths of the genetically informed design allow us to provide strong evidence that (a) some putative effects of marriage are attributable to nonrandom selection, (b) others are due to cohabitation either inside or outside of marriage, and (c) still other observed benefits indeed do arise from marriage above and beyond cohabitation.

## Implications

Our findings have implications for primary and clinical interventions. The past decade has witnessed legislation supporting marriage promotion initiatives such as the Marriage and Fatherhood Provisions of the Deficit Reduction Act of 2005. Our findings that marriage-like relationships apparently are (despite age, gender, ethnicity, genes, and rearing environment) causally protective against internalizing and externalizing problems are critical to the rationale behind such efforts that assume causation, not just correlation. With respect to more direct clinical interventions, meta-analyses demonstrate that social support and marital status are positively related to medical treatment adherence (DiMatteo, 2004), and social support is related to psychotherapy treatment success (Roehrl & Strouse, 2008). Patients with spouses or partners may have a built-in social support network that can be recruited in the treatment of externalizing and internalizing problems to increase treatment effectiveness.

## Genetically Informed Research in Family Psychology

As a final note, we wish to emphasize the broader utility of genetically informed research in distinguishing correlation from causation in family psychology. The present study focuses on marital status, a phenotype for which twins and siblings may be discordant. Other behavior genetic studies of family processes have taken this approach as well, examining such topics as the relationships between marital support and depressive symptoms (e.g., Beam et al., 2011; South & Krueger, 2008; Spotts et al., 2004), and child psychological adjustment and parent-child conflict (e.g., Klahr, McGue, Iacono, & Burt, 2011; Spanos, Klump, Burt, McGue, & Iacono, 2010). Methods have also been developed to examine experiences *shared* by siblings. Samples of children of twins and children of siblings have been used to study the relationship between children's psychological adjustment and parental marital instability (e.g., D'Onofrio et al., 2005), marital conflict (Harden et al., 2007), psychopathology (e.g., Singh et al., 2011; Slutske et al., 2008), family functioning (Schermerhorn et al., 2011), and family structure (e.g., Mendle et al., 2009). These are but a few examples of contributions behavior genetic research has made to the field of family psychology, and we encourage researchers to turn their attention to genetically informed research as a quasi-experimental tool in the correlational study of families.

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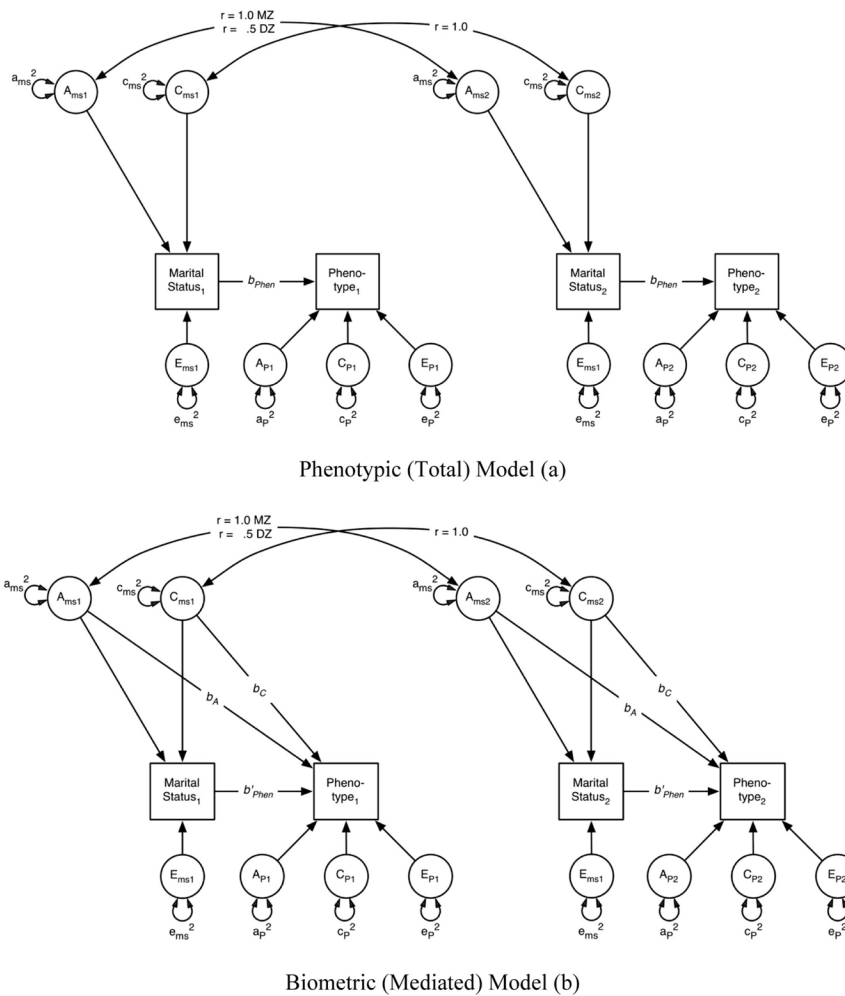
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**Figure 1.**

Phenotypic (a) and biometric (b) models fit to the data.  $a_{ms}^2$ ,  $c_{ms}^2$ , and  $e_{ms}^2$  are the additive genetic, shared environmental, and nonshared environmental (ACE) variance components of marital status;  $a_p^2$ ,  $c_p^2$ , and  $e_p^2$  are the ACE components of the phenotype;  $b_{phen}$  is the total effect of marital status on the phenotype;  $b'_{phen}$  is the direct effect of marital status on the phenotype controlling for indirect effects ( $b_A$  and  $b_C$ ). Although not shown for clarity, ACE components of the phenotypes were constrained to correlate exactly as is shown for the ACE components of marital status. Covariates were partialled from marital status and the phenotype in both models.



**Table 1**  
 Unstandardized Parameter Estimates for Phenotypic and Biometric Models Comparing Coupled (1) and Single (0) Individuals

Estimate [95% CI]	Internalizing			Externalizing			Health		
	Depression	Suicidal ideation	Alcohol use	Antisocial behavior	Smoking	Physical health			
Variance components of marital status									
$a^2$	.048 [.000, .113]	.044 [.000, .101]	.068 [.000, .135]	.037 [.000, .089]	.047 [.000, .158]	.063 [.000, .168]			
$c^2$	.031 [.000, .079]	<b>.031 [.001, .067]</b>	.021 [.000, .066]	.026 [.000, .062]	.035 [.000, .080]	.030 [.000, .061]			
$e^2$	<b>.139 [.089, .229]</b>	<b>.136 [.087, .215]</b>	<b>.133 [.083, .209]</b>	<b>.112 [.074, .187]</b>	<b>.151 [.083, .158]</b>	<b>.140 [.122, .27.077]</b>			
Regression coefficients									
Total model									
$b_{phen}$	<b>-.050 [-.096, -.005]</b>	<b>-.495<sup>†</sup> [-.743, -.296]</b>	<b>-.577 [-.792, -.422]</b>	<b>-.099<sup>†</sup> [-.216, -.012]</b>	<b>-1.001 [-1.547, -.324]</b>	<b>-.008 [-.102, .073]</b>			
Mediated model									
$b'_{phen}$	<b>-.113 [-.300, -.007]</b>	<b>-.926<sup>†</sup> [-1.938, -.295]</b>	<b>-.688 [-1.080, -.223]</b>	<b>-.158<sup>†</sup> [-.693, .097]</b>	<b>-1.357 [-4.132, .376]</b>	—			
$b_A$	.159 [-.080, .561]	1.108 <sup>†</sup> [-.575, 2.834]	.275 [-.927, 1.006]	.151 <sup>†</sup> [-.542, 1.099]	.896 [-3.876, 5.248]	—			
$b_C$	.159 [-.080, .561]	1.108 <sup>†</sup> [-.575, 2.834]	.275 [-.927, 1.006]	.151 <sup>†</sup> [-.542, 1.099]	.896 [-3.876, 5.248]	—			
Covariates									
Effect on marital status									
Wave III score <sup>†</sup>	<b>-.243 [-.336, -.160]</b>	-.123 [-.237, .011]	.008 [-.012, .030]	<b>-.110 [-.157, -.059]</b>	.002 [-.004, .007]	.003 [-.009, .016]			
Gender <sup>†</sup>	-.036 [-.075, .014]	-.035 [-.072, .013]	-.038 [-.077, .012]	-.029 [-.066, .011]	-.036 [.081, .012]	-.042 [-.092, .003]			
Ethnicity <sup>†</sup>	<b>-.199 [-.238, -.161]</b>	<b>-.192 [-.232, -.156]</b>	<b>-.199 [-.236, -.162]</b>	<b>-.176 [-.210, -.142]</b>	<b>-2.06 [-2.248, -.165]</b>	<b>-.204 [-.259, -.158]</b>			
Age <sup>†</sup>	<b>.051 [.035, .072]</b>	<b>.049 [.032, .067]</b>	<b>.052 [.033, .070]</b>	<b>.045 [.030, .061]</b>	<b>.051 [.033, .070]</b>	<b>.051 [.036, .072]</b>			
Effect on phenotype									
Wave III score	<b>.467 [.387, .535]</b>	<b>.962<sup>†</sup> [.656, 1.443]</b>	<b>.570 [.513, .619]</b>	<b>.488<sup>†</sup> [.388, .597]</b>	<b>.766 [.288, .940]</b>	<b>.459 [.416, .501]</b>			
Gender	<b>-.104 [-.143, -.069]</b>	<b>-.288<sup>†</sup> [-.459, -.069]</b>	<b>.681 [.565, .797]</b>	<b>.313<sup>†</sup> [.263, .368]</b>	<b>1.231 [.509, 1.867]</b>	<b>.091 [.012, .160]</b>			
Ethnicity	.037 [-.009, .078]	<b>-.361<sup>†</sup> [-.625, -.147]</b>	<b>-.593 [-.743, -.450]</b>	<b>.113<sup>†</sup> [.002, .206]</b>	<b>-2.615 [-3.628, -1.889]</b>	<b>-.147 [-.218, -.071]</b>			
Age	-.001 [-.015, .011]	.040 <sup>†</sup> [-.025, .110]	-.043 [-.083, .007]	-.024 <sup>†</sup> [-.051, .008]	.070 [-.113, .316]	<b>-.022 [-.044, -.001]</b>			
Goodness of fit									
RMSEA (CFI/TLI)	.027 (.934/.938)	.058 (.979/.982)	.051 (.921/.928)	.072 (.971/.975)	.094 (.948/.956)	.065 (.975/.979)			

*Note.* Bolded values are significant at  $p < .05$ ;  $a^2$ ,  $c^2$ , and  $e^2$  are the additive genetic (A), shared environmental (C), and non-shared environmental (E) variance components of marital status;  $b_{Phen}$  and  $b'_{Phen}$  are the total and direct effects, respectively, of marital status on the phenotype;  $b_A$  and  $b_C$  are the indirect effects of marital status on the phenotype due to the A and C components of marital status. In each model,  $b_A$  and  $b_C$  were constrained to be equal.

<sup>†</sup> Probit regression weights.

**Table 2**  
 Unstandardized Parameter Estimates for Phenotypic and Biometric Models Comparing Married (1) and Cohabiting (0) Individuals

	Internalizing			Externalizing			Health	
	Depression	Suicidal ideation	Alcohol use	Antisocial behavior	Smoking	Physical health		
Variance components of marital status								
$a^2$	.166 [.000, .502]	.221 [.076, .409]	.142 [.000, .408]	.150 [.042, .319]	.160 [.000, .368]	.184 [.036, .403]		
$c^2$	.000 [.000, .000]	.000 [.000, .000]	.000 [.000, .124]	.000 [.000, .000]	.001 [.000, .236]	.000 [.000, .000]		
$e^2$	.409 [.142, .753]	.404 [.225, .771]	.395 [.203, .628]	.326 [.178, .528]	.332 [.159, .557]	.399 [.230, .666]		
Regression Coefficients								
Total model								
$b_{phen}$	.003 [-.057, .064]	-.590 <sup>†</sup> [-1.072, -.305]	.092 [-.124, .643]	-.332 <sup>†</sup> [-.509, -.224]	-.245 [-1.073, .812]	.024 [-.063, .113]		
Mediated model								
$b'_{phen}$	—	.117 <sup>†</sup> [-.293, .863]	—	-.326 <sup>†</sup> [-.754, -.067]	—	—		
$b_A$	—	-1.761 <sup>†</sup> [-3.435, -.967]	—	-.067 <sup>†</sup> [-1.311, 1.171]	—	—		
$b_C$	—	[0]	—	[0]	—	—		
Covariates								
Effect on marital status								
Wave III score <sup>†</sup>	-.428 [-.851, -.234]	-.027 [-.081, .274]	-.306 [-.560, -.150]	-.261 [-.370, -.145]	-.013 [-.026, -.005]	.136 [.045, .240]		
Gender <sup>†</sup>	-.116 [-.228, .028]	-.149 [-.267, -.001]	-.132 [-.231, .019]	-.121 [-.219, -.004]	-.124 [-.226, -.002]	-.132 [-.236, -.001]		
Ethnicity <sup>†</sup>	-.186 [-.286, -.078]	-.147 [-.265, -.002]	-.170 [-.268, -.074]	-.153 [-.239, -.062]	-.148 [-.239, -.053]	-.171 [-.265, -.071]		
Age <sup>†</sup>	.071 [.022, .123]	.055 [.007, .110]	.067 [.025, .112]	.060 [.021, .103]	.061 [.022, .105]	.066 [.023, .114]		
Effect on phenotype								
Wave III score	.601 [.467, .711]	-.314 <sup>†</sup> [-.125, 1.130]	1.592 [1.107, 2.280]	.225 <sup>†</sup> [.055, .401]	.668 [.509, .866]	.527 [.440, .594]		
Gender	-.124 [-.200, -.060]	-1.042 <sup>†</sup> [-1.559, -.613]	.770 [.571, .987]	.352 <sup>†</sup> [.173, .479]	1.596 [.666, 2.478]	.025 [-.093, .167]		
Ethnicity	.069 [.017, .125]	-.038 <sup>†</sup> [-.506, .417]	-.294 [-.497, -.110]	.140 <sup>†</sup> [-.004, .280]	-1.539 [-2.482, -.644]	-.230 [-.348, -.110]		
Age	-.016 [-.042, .008]	-.015 <sup>†</sup> [-.193, .136]	-.048 [-.132, .024]	-.030 <sup>†</sup> [-.087, .025]	.079 [-.254, .383]	-.019 [-.061, .026]		
Goodness of Fit								
RMSEA (CFI/TLI)	.027 (.864/.872)	.041 (.960/.965)	.061 (.724/.749)	.074 (.841/.867)	.078 (.841/.870)	.064 (.880/.902)		

Note. Bolded values are significant at  $p < .05$ ;  $a^2$ ,  $c^2$ , and  $e^2$  are the additive genetic (A), shared environmental (C), and non-shared environmental (E) variance components of marital status;  $b_{Phen}$  and  $b'_{Phen}$  are the total and direct effects, respectively, of marital status on the phenotype;  $b_A$  and  $b_C$  are the indirect effects of marital status on the phenotype due to the A and C components of marital status.

<sup>†</sup> Probit regression weights.