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Marital Strain, Support, and Alcohol Use: Results from a Twin Design Statistically Controlling for Genetic Confounding

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ABSTRACT

Background: Marriage is one of the most frequently examined sources of social support and has been shown to protect against alcohol use and abuse. This study examines the relationship between perceived marital strain and support, and alcohol use controlling for additive genetic influence. **Methods:** Data from monozygotic (MZ) ($n = 320$) and dizygotic (DZ) ($n = 464$) twin pairs from the second wave of the National Survey of Midlife Development in the United States (MIDUS II) were used to test whether past year marital strain and support were associated with recent alcohol use. Generalized linear mixed models (GLMM) were estimated, allowing us to control for additive genetic and shared environmental influences as variance components. **Results:** Marital strain and support had positive, statistically significant associations with alcohol use. However, only the relationship between marital strain and alcohol use remained after controlling for variance in alcohol use attributed to genetics. **Conclusions:** After accounting for genetics, midlife adults still appear to cope with marital strain *via* alcohol use. However, this coping is unlikely to result in heavy episodic drinking or alcohol use disorder without other compounding factors.

KEYWORDS

Marriage; strain; support; alcohol use; genetics twins

Introduction

Heavy episodic drinking (HED) is prevalent and costly in our society. On average, alcohol consumption in the United States has increased steadily since the early 1900s (Holmes & Anderson, 2017). During 2015, an estimated 26.9% of individuals aged 18 and older reported binge drinking in the past month (SAMHSA, 2015). In 2010, the annual cost of binge drinking in the United States was estimated at \$188 billion (Bouchery, Harwood, Sacks, Simon, & Brewer, 2011). Grant and colleagues (2015) estimated the lifetime prevalence of alcohol use disorder (AUD) to be 29.1%, noting its high comorbidity with other substance use disorders, as well as antisocial and borderline personality disorders, major depressive and bipolar disorders, and generalized anxiety disorder. Understanding factors associated with alcohol consumption will aid in the prevention of HED/AUD.

Marital support, marital strain, and alcohol use

Studies show that marriage consistently protects against alcohol problems (cf. Berggren & Nystedt, 2006; Harford, Hanna, & Faden, 1994; Horwitz & White, 1991; Kretsch & Harden, 2014). Examining data from the National Longitudinal Study of Youth (NLSY) 1979–2000, Duncan, Wilkerson, and England (2006) found that marriage was associated with a 10–20% decrease in the odds of HED for men and women. Willoughby and Dworkin (2009) found

that even the desire to marry is associated with fewer instances of HED. While marital status and intention to marry has received much empirical attention, the association between marital strain and support, and problematic alcohol consumption has received less empirical attention. Fischer and colleagues (2005) found a weak association between general disagreements between partners and HED. However, after accounting for partner selection, Fleming, White, and Catalano (2010) found no relationship with heavy drinking. Moreover, disagreements do not necessarily proxy for all strains and supports unique to the marriage relationship.

Marriage is a trajectory and, upon entrance, can be defined by several dimensions, including the aforementioned supports and strains (Bookwala, 2005). Horwitz, White, and Howell-White (1996) observed that the strains which initiate divorce, and by extension strained marriages, could precipitate alcohol use as a coping behavior (Brennan, Moos, & Mertens, 1994), as observed for a range of other stressors (Cooper, Russell, Skinner, Frone, & Mudar, 1992; Howell, Leyro, Hogan, Buckner, & Zvolensky, 2010; Lennon 1987; Park, Armeli, & Tennen, 2004). Supporting this notion, Halford and Osbarby (1993) found heavy alcohol to be prevalent among couples in marital therapy, which could indicate that marital stressors precipitate both marital therapy and heavy drinking. However, the authors further note that in many cases heavy drinking precipitates the stressors which cause couples to engage in marital therapy (see also

Leonard & Roberts, 1998; Levinger, 1966; Wilsnack & Wilsnack, 1990). Ultimately, it is reasonable to assume the relationship between marital stress and alcohol use is complex and reciprocal.

The relationship between marital support and alcohol use is characterized by even greater complexity. Both Pearlman and Johnson (1977) and Leonard and Rothbard (1999) proposed that marital support and alcohol use should be negatively associated, arguing that the relationship between divorce and alcohol use could be partially attributable to a loss of intimate support. The empirical literature typically bears out this hypothesis that social support is negatively associated with alcohol use and abuse (Booth et al., 1992; Jennison, 1992; Moos, Fenn, Billings, & Moos, 1988; Pierce, Frone, Russell, Cooper, & Mudar, 2000; Sherbourne, Hays, & Wells, 1995; Steptoe, Wardle, Pollard, Canaan, & Davies, 1996). However, an important caveat is that research analyzing the second wave of the Midlife in the United States (MIDUS II) data has shown that support from romantic partners and family members is positively associated with a range of problematic health outcomes (Priest, Roberson, & Woods, 2019). This inversion of initial expectations could also apply to the relationship between marital support and problematic alcohol use. Indeed, Schutte, Brennan, and Moos (1994) observed that, later in life, remission in alcohol problems are associated with reduced spousal support for drinking. It may be the case that individuals with alcohol problems perceive their partner's vocal aversion to alcohol use as a lack of support, a supportive partner being one who is permissive of problematic alcohol use. Roberts and Leonard (1998) describe a "frequent intimate" pattern of alcohol use wherein romantic partners frequently consume alcohol while maintaining a positive, supportive relationship. Thus, while a majority of the social support literature implies a negative relationship with alcohol use, recent theory and research seems to imply that marital support could exacerbate alcohol use, especially during midlife.

Length of marriage is also a fundamental part of a marriage trajectory which has been related to alcohol use. Specifically, longer periods of marriage are associated with reductions in alcohol use (Harford et al., 1994; Horwitz, White, & Howell-White, 1996). Provided divorce is a strong negative correlate of HED (Salvatore et al., 2017), number of marriages may also be an important correlate of alcohol use. To this end, this study also accounts for marriage length and number of marriages.

The potential role of genetic confounding

Genetic heritability is often overlooked in studies examining the association between marriage, marital characteristics, and alcohol use. Studies have shown that alcohol use is influenced by genes. Meta-analyses and systematic reviews have concluded alcohol use is between 40 and 70% heritable (Tawa, Hall, & Lohoff, 2016; Verhulst, Neale, & Kendler, 2015). These estimates are consistent with the average estimated heritability of 49% in a meta-analysis of 50 years of twin studies analyzing 2748 articles examining a total of

17804 phenotypes (Polderman et al., 2015). Studies have further shown that various dimensions of a marriage trajectory are also influenced by genes. For example, McGue and Lykken (1996) estimated divorce to be approximately 53% heritable. Jerskey and colleagues (2010) found the heritability of marriage and divorce to be 41% and 32%, respectively. Finally, Salvatore and colleagues (2017) found that a substantial portion of covariation between divorce and alcohol use is explained by genetics.

Marriage characteristics are also influenced by marital homophily *via* assortative mating (Boutwell, Beaver, & Barnes, 2012). Individuals possessing similar traits are likely to attract each other. Sieving, Perry, and Williams (2000) suggest individuals choose peers whose behaviors and beliefs are similar to their own, an assertion which has been found to apply to views on alcohol use (Parra, Krull, Sher, & Jackson, 2007). Consequently, individuals are likely to select romantic partners who have similar reported levels of alcohol consumption (Fleming et al., 2010; Wiersma, Fischer, Cleveland, Reifman, & Harris, 2010). This seems to indicate the presence of a selection effect *vis-à-vis* marital homophily. Moreover, once in a relationship, romantic couples are likely to influence each other's alcohol use (Bartel et al., 2017).

Consequently, it stands to reason that heritability (or genetics) is a confounding factor in the association between marital strain and support, and alcohol use. Genetics may influence alcohol use, the likelihood of problem drinking, and the decision to marry. Genetics may also influence whom an individual marries and marital quality. Recent research examining the relationship between marriage and alcohol use have controlled for potential genetic confounding. For example, Kretsch and Harden (2014) control for genetic selection effects by nesting a growth curve analysis within sibling groups. However, a more direct approach to controlling genetic influence would be to continue biometric modeling traditions which utilize differences in genetic similarity between monozygotic (MZ, identical) twins, who share 100% of their genes, and dizygotic (DZ, fraternal) twins, who share approximately 50% of their genes (DeFries & Fulker, 1985; Rodgers & Kohler, 2005).

Indeed, Kendler, Lonn, Salvatore, Sundquist, and Sundquist (2016, 2017) continued this tradition in a population-level study of Sweden, modeling the relationship between marital status and the onset of AUD nested within a variety of genetic strata, including pairs of same-sex cousins, half-siblings, full-siblings, and monozygotic (MZ) twins. Similar to a parameterization proposed by Guo and Wang (2002), their approach separately examines within-pair covariance between the independent and dependent variable for a range of genetic strata. When taking this approach, the association between the independent and dependent variable will be weakened when confounded by either shared environmental or genetic factors. Consequently, a stronger relationship will be observed in higher genetic strata, those respondent dyads which share genetic and environmental backgrounds (Kendler et al., 2016). However, this approach is limited in two important ways: (1) it is difficult to ascertain how the parameters of the model are related to the

behavioral genetic parameters of interest (i.e. genetics, shared, and nonshared environments) (Rabe-Hesketh, Skrondal, & Gjessing, 2008); (2) it is unable to disentangle shared environmental and genetic variance.

This study addresses the potentially problematic, frequently implicit assumption found throughout studies on marriage and alcohol use. Namely that the confounding influence of genetics on the relationship(s) between social and behavioral variables is trivial. As we have attempted to demonstrate, this assumption is untenable given the extant body of research. We examine the relationship between marital strain/support and alcohol use among MZ and DZ twin pairs participating in MIDUS II. Our study employs a comprehensive statistical approach to controlling the influence of genetics, amending the methodological limitations of previous research by estimating generalized linear mixed models (GLMM) that allow us to capitalize on data collected from twin pairs to individually account for additive genetic confounding, shared environmental influences, as well as an array of other variables measuring individual-level differences within twin pairs (Rabe-Hesketh et al., 2008). We hypothesize that: (1) marital strain is positively associated with Alcohol Use; (2) marital support is negatively associated with Alcohol Use, consistent with the broader social support literature; and (3) while robust of genetic confounds, the magnitude of these relationships will decrease with the introduction of genetic and shared environmental variance components.

Methods

Sample and procedure

This study examines cross-sectional data from MIDUS II (2004–2006) to test the association between marriage strain/support and alcohol use among twin pairs participating in wave 2 of the National Study of Midlife in the United States (MIDUS II). A unique design characteristic of MIDUS was an extension made to the general population sample which added a subsample of 957 twin pairs. Beginning in 1995, data from a nationally representative sample of midlife adults were collected from participants ($N = 7108$), including a subsample ($N = 1914$) of MZ twins, who share 100% of their genes, and DZ twins, who share approximately 50% of their genes (Brim et al., 1995-6; Ryff et al., 2004-06). The subsample of twin pairs allows researchers to test biometric models estimating additive genetic, shared environmental, and nonshared environmental influences on health and well-being (see Cleveland & Almedia, 2013).

The MIDUS subsample of twins completed the same interviews and surveys as other MIDUS participants, and they were also asked to complete a short questionnaire that asked about their biological similarity (Kessler et al., 2004). This additional questionnaire was used to classify the zygosity of each twin pair (i.e. MZ identical and DZ fraternal twin pairs). Twins were asked if they looked almost exactly alike during childhood, had the same eye and hair color, complexion, height, weight, and facial features as their twin, the frequency which they were mistaken for their twin by

their parents during childhood, and if they believe themselves to be DZ or MZ. If a twin pair scored greater than 18 on the 10-item inventory contained within this screening survey they were assumed to be MZ. Research has demonstrated that this approach to determining zygosity is approximately 95% accurate (Reed et al., 2005; Rietveld et al., 2000).

Our analysis sample consisted of twin pairs whose zygosity could be determined with high certainty and who also reported being married or cohabiting in a marriage-like relationship during wave 2 interviews. Zygosity could not be determined in 25 cases and, of those remaining cases, 1136 reported being married or cohabiting. An additional 352 unpaired cases were dropped from our analysis. The remaining analysis sample consisted of 784 cases from 392 twin pairs within 383 families, of which 320 cases are MZ twins (i.e. 160 MZ twin pairs), 266 are same sex DZ twins (i.e. 133 DZ twin pairs), and 198 cases are opposite sex DZ twins (i.e. 99 opposite sex DZ twin pairs).¹ The analysis sample was 96.4% white, 47.3% male, averaged 52.9 years old [standard deviation (sd) = 10.85, min = 34, max = 81], with an average annual household income of \$79,392 (sd = \$52,852 min = \$0, max = \$300,000). These characteristics are similar to the full MIDUS II sample, which is 46.7% male, with an average age of 55.4 years (sd = 12.45, min = 28, max = 84), with an average annual household income of \$71,322 (sd = \$60,332 min = \$0, max = \$300,000). However, because the twin subsample does not overlap with MIDUS's metropolitan over-sample there is a greater proportion of white respondents when compared with the full sample's 90.6% white respondents.

It is important to briefly describe the logic of biometric models before discussing the statistical model used in this study. Biometric models (i.e. DF and ACE models) are commonly used to partition variance in a phenotype (e.g. alcohol use) into three primary components that include additive genetics (A), shared environment (C) (e.g. family of origin and family characteristics with twin pairs), and nonshared environment (E) (e.g. unique experience twins experience within pairs). The univariate biometric model, otherwise known as the ACE model, provides estimates for the proportions of variance due to each of these components. For example, 50% of the variance in phenotype is heritable, 10% is due to the shared environment, and 40% to the nonshared environment. As reported in a recent meta-analysis of 50 years of twin studies (Polderman et al., 2015), biometric models provide evidence to suggest that the most influential environmental influences on phenotypes are likely to be found in the nonshared environment, unique experiences between twins within pairs. However, it is often the case that such biometric models are unable to identify or estimate precisely which nonshared environmental variables

¹A multiple imputation (MI) algorithm in STATA 13 was used to retain missing observations. Since missingness approached 20% in some measures, including marital, familial, and peer strain/support, we imputed 50 data sets, and missing values were replaced with mean scores from across the 50 imputed multivariate normal (MVN) data sets, which was necessary as STATA 13's MI function cannot be combined with the GLLAMM package. (See Table 1 for post-imputation descriptive statistics by zygotic category).

are most relevant in predicting a phenotype (e.g. alcohol use). The GLMM used for this study allows us to address this problem.

This study uses Rabe-Hesketh and colleagues' (2008) GLMM to examine whether measured marital characteristics (i.e. indicators of nonshared environmental influences) are significantly associated with alcohol use while statistically controlling for additive genetic and shared environmental influences as measured by random effects in the multilevel model. The GLMM ACE model is parameterized as follows:

$$y_{ij} = \beta_0 + \sum \beta_k^{(1)} X_{ij} + \left\{ a_{ij}^{(2)} \left[\sqrt{\frac{1}{2} M_j} \right] + a_j^{(3)} \left[M_j + \sqrt{\frac{1}{2} M_j} \right] \right\} + c_j^{(3)} + e_{ij}^{(1)} \quad (1)$$

Where β_0 is the intercept and $\beta_k^{(1)}$ are the fixed effects, the change in respondent's alcohol use estimates for a one-unit change in an independent variable, and the random effects are interpreted as additive genetic and shared environment variance components, as in univariate biometric ACE models. The random effects $a_{ij}^{(2)}$ and $a_j^{(3)}$ are a pair of variance components which, after imposing equality constraints, both estimate the sample variance attributable to genes (A). The $c_j^{(3)}$ random effect estimates the proportion of variance attributable to the shared environment (C), while the remaining error, represented by the $e_{ij}^{(1)}$ parameter, is assumed to be the nonshared environmental variance (E). This parameterization allows for both (1) the estimation of ACE models while employing statistical controls for individual-level covariates, and (2) the estimation of fixed effects (i.e. individual-level covariates) while controlling for the influences of additive genetics and the shared environment. Since nonshared environmental variance is captured at the first level, (1), fixed effect estimates only take into account nonshared environmental variance. The AE model is nested in the ACE model; the parameterization only differs in that the shared environmental, $c_j^{(3)}$ term is dropped. The AE parameterization captures both shared and nonshared environmental variance at level 1. Rather than testing a hypothesis about genetic heritability we use Rabe-Hesketh et al.'s (2008) GLMM to control for additive genetic variance. The inclusion of additive genetic (A) and shared environment (C) random effects allows us to estimate the nonshared environmental fixed effects for indicators of marriage characteristics on alcohol use after accounting for the proportion of variance assigned to additive genetics and the shared environment influences on alcohol use.

Measures

Alcohol use

MIDUS II includes several composite scales assessing alcohol use. The problem with additive composites is that they necessitate the assumption that all component survey item responses are equally weighted, which is often not the case (Osgood, McMorris, & Potenza, 2002). As an example, frequently experiencing the effects of alcohol in the workplace

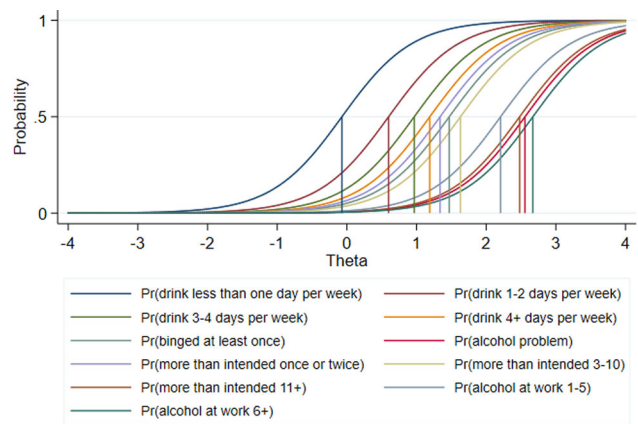


Figure 1. Boundary characteristic curves for an alcohol use continuum.

is probably much less common within the population than frequently drinking more alcohol than intended, making it a much stronger indicator of alcohol abuse—assuming the respondent does not work at a brewery or bar. Moreover, alcohol measures frequently assess alcohol use and abuse separately, when alcohol researchers have recently started to question this distinction, conceptualizing, and modeling alcohol use as a continuum (Hagman & Cohn, 2011; Krueger et al., 2004; Rehm et al., 2013; Saha, Chou, & Grant, 2006).

In an attempt to address these concerns, this study measured alcohol use with a continuous scale of five self-report items created from an Item Response Theory (IRT) measurement model. Survey items included: (1) average drinking frequency per month with 5 response categories ranging from “every day” to “less than one day a week” and “never drink” (which were collapsed into a single category); (2) HED, participants were asked if they consumed five or more drinks on one occasion in the past month (1 = yes, 0 = no); (3) alcohol problem, a binary measure derived from participants responses to four questions asking if they had suffered from alcohol-induced emotional problems, an irresistible desire/urge to use alcohol, a period of 1+ months using large quantities of alcohol, and diminished returns on the effect of alcohol use (1 = yes [alcohol problem], 0 = no); (4) number of times in past month suffered from alcohol's effects at work; and (5) number of times in past month used more alcohol than intended (ranging from 0 “never” to 6 “more than 20 times”). For each respondent, the IRT model produced logit scale scores, theta scores forthwith, which ranged from -1.531 to 2.849 . Higher scores indicate increasingly problematic alcohol use.

Figure 1 depicts the boundary characteristic curves associated with each individual survey item that informed the alcohol use theta scores used as our dependent variable. Each theta score is associated with an array of probabilities predicting the likelihood a respondent positively endorsed one of the aforementioned survey items. A respondent with a high theta score is more likely to positively endorse survey items which most other respondents would find it much more difficult to endorse (i.e. predictors of alcohol abuse/dependency). Although theta is somewhat right-skewed, as evidenced by these boundary characteristic curves tending

toward higher difficulties, the distribution of theta is sufficiently normal that a Gaussian (identity) link function is appropriate for our GLMMs.

Marital strain and support

Marital strain and support were measured using two six-item composite scales assessing spouse/partner strain and support. Spouse/partner support included items which asked respondents to what extent (1) their partner really cares about them, (2) their partner understands the way they feel, (3) their partner appreciates them, (4) they are able to rely on their partner vis-à-vis serious problems, (5) they are able to open up and talk about worries with their partner, and (6) they are able to relax around their partner ($\alpha=0.91$). Each item had four response categories ranging from “a lot” to “not at all” so that higher values represent greater support. The spouse/partner strain scale included items asking participants how often their partner (1) makes too many demands, (2) argues with them, (3) makes them feel tense, (4) criticizes them, (5) lets them down, (6) gets on their nerves. These items include four response categories ranging from “often” to “never” and were coded such that higher values represent greater strain ($\alpha=0.86$). Both marital strain and support measures are grand mean-centered in our statistical models.

Statistical control variables

Self-reported length of respondents’ marriage/relationship was measured in years ranging from 0 to 60, and number of prior marriages, ranged from 0 to 5. A binary variable for current marital status was included to distinguish between officiated marriages and marriage-like relationships

(1 = unmarried, 0 = married). Studies have shown that female drinking appears contingent upon their spouse while male drinking does not, the result being that males tend to benefit from a marriage-induced reduction in alcohol use regardless of their spouse where the same is not true of females (Hanna, Faden, & Harford, 1993). As such, we include binary variables for self-reported gender (1 = male, 0 = female) and spousal alcoholism (1 = lived with an alcoholic spouse in last 12 months, 0 = have not lived with alcoholic spouse in the last 12 months). Given that peer and family strain and support may covary with marital strain and support, we include four composite scales controlling for peer strain, family strain, peer support, and family support each constructed using a subset of the questions posed regarding marital support and strain (how much they (1) really care, (2) understand the way the respondent feels, (3) can be relied upon, (4) can be opened up to, and how often they (1) make too many demands, (2) criticize the respondent, (3) let the respondent down, and (4) get on the respondent’s nerves). All four extramarital support/strain controls range from 0 (low strain/support) to 3 (high strain/support). Self-reported number of children, ranging from 0 to 17, and a self-reported binary measure of pregnancy (1 = pregnant, 0 = not pregnant) also served as control variables given that the transition to motherhood is associated with decreases in alcohol use (Matusiewicz, Ilgen, & Bohnert, 2016). Since parents are the source of both inherited and modeled behavior, we account for parental alcohol problems during childhood using a self-reported binary measure (1 = parental alcohol problem, 0 = no parental alcohol problem). We include four items which asked participants to self-evaluate their physical and mental/emotional health from 1 (poor) to 5 (excellent) for both themselves

Table 1. Descriptive statistics by zygotic category.

Variables	DZ					MZ				
	μ	σ	Min	Max	ICC	μ	σ	Min	Max	ICC
Alcohol use	-0.017	0.817	-0.815	2.920	0.262	0.025	0.824	-0.815	2.244	0.608
Marital strain	1.167	0.545	0	3	0.142	1.167	0.557	0	3	0.252
Marital support	2.62	0.495	0	3.023	0.162	2.67	0.475	0	3	0.269
Unmarried cohabiting	0.0323	0.177	0	1	0.107	0.0437	0.205	0	1	0.256
Length of marriage	26.21	13.81	0	58	0.604	25.32	13.18	1	60	0.464
Number of marriages	1.265	0.603	0	5	0.129	1.212	0.547	0	4	0.249
Spousal Alc. Prob.	0.0302	0.153	-0.084	1	0.153	0.0326	0.157	-0.0943	1	-0.004
Peer strain	0.826	0.455	0	2.25	0.048	0.789	0.443	0	2.75	0.098
Family strain	1.037	0.551	0	2.75	0.204	1.021	0.49	0	3	0.306
Peer support	2.336	0.576	0	3	0.021	2.327	0.584	0	3	0.420
Family support	2.586	0.475	0.5	3	0.206	2.68	0.387	0.75	3	0.287
Number of children	2.694	1.704	0	14	0.248	2.575	1.733	0	17	0.410
Pregnant	0.00392	0.0475	-0.0422	1	-0.002	0.00647	0.0793	-0.0355	1	-0.002
Parental Alc. Prob.	0.218	0.413	0	1	0.710	0.204	0.403	0	1	0.634
Resp. physical health	3.534	1	1	5	0.204	3.65	0.968	1	5	0.460
Sp. physical health	3.475	0.929	1	5	0.042	3.667	0.934	1	5	0.151
Resp. mental health	3.884	0.879	1	5	0.013	3.919	0.892	1	5	0.255
Sp. mental health	3.894	0.918	1	5	0.094	4.037	0.884	1	5	0.145
Depression	0.483	1.55	0	7	-0.063	0.444	1.468	0	7	0.348
Anxiety	0.0711	0.621	0	9	-0.011	0.0656	0.53	0	6	-0.012
Religiosity	3.004	0.733	1	4	0.204	2.916	0.821	1	4	0.369
Work satisfaction	7.57	1.837	0	10	0.030	7.699	1.866	0	10	0.214
Household income	76.47	53.05	0	300	0.324	83.63	52.64	0	300	0.307
Education level	6.06	2.481	0	11	0.406	6.327	2.285	1	11	0.647
Age	52.82	10.91	34	81	0.999	52.89	10.78	35	80	0.999
Non-white	0.0275	0.153	-0.048	1	0.737	0.0486	0.212	-0.0175	1	0.790
Male	0.468	0.499	0	1	0.145	0.481	0.5	0	1	1.000

and their spouse. Given their established relevance to the marriage–alcohol use relationship (Horwitz & White, 1991; Sharma et al., 2016), we specifically control for depression and anxiety using 13- and 10-item composite scales ranging from 0 to 7 and 0 to 9 respectively, where greater scores indicate increased frequency and severity of symptoms. We control a range of demographic variables including religiosity, assessed using an ordinal variable asking respondents how religious they are ranging from 1 (not religious) to 4 (highly religious), work/job satisfaction, asking respondents how they rate their current work situation (regardless of employment) on a 11-point ordinal variable ranging from 0 (unsatisfied) to 10 (satisfied), education, asking respondents to report their highest level of completed education on a single 12-point ordinal variable ranging from 0 (no schooling) to 11 (Ph.D or equivalent), race, assessed *via* self-report and recoded into a binary variable where 1 = non-white and 0 = white, age, assessed in years by subtracting date of birth from the survey date producing a continuous variable ranging from 34 to 81, and finally annual household income, using a self-reported continuous variable measured in thousands of 2004 USD ranging from \$0 to \$300,000+. All continuous and ordinal variables were grand mean-centered in our statistical models, standardized with means of 0 and standard deviations of 1.

Analysis

A series of multivariate models were estimated to determine the association between marital strain/support variables and alcohol use. First, estimates from multivariate generalized linear models (GLM) with identity link functions are presented to show the relationship between marital strain/support variables and alcohol use without controls for additive genetic (A) and shared environmental (C) influences. Second, results from GLMMs are presented which reassess the marital strain/support–alcohol use relationship after statistically controlling for additive genetic and shared environmental influences.

Results

Table 2 shows results from GLMMs using identity link functions. Models A through D introduce individual-level covariates (i.e. fixed effects), beginning with the marriage variables, next introducing controls for marital strain and support, family and health, and finally demographics. Results in Models E and F are estimates from GLMMs which introduce random effects that control for genetic (A) and shared environment (C) influences on alcohol use.

Models A through D show that marital strain has a positive and statistically significant association with theta (alcohol use), and remains statistically significant with a consistent magnitude when controlling for marital support, family and health covariates, and demographic characteristics. The association between marital support and alcohol use was marginally significant ($p < .10$) after the inclusion of family, health, and demographic controls, its coefficient

reduced by 40% when compared with model A. Of the control variables, number of children and spouse's physical health were significantly related to alcohol use ($b_{\text{children}} = -0.028$; $b_{\text{physical health}} = 0.067$), albeit weakly ($p = .08-.092$). Pregnancy was negatively associated with alcohol use ($b = -0.531$), while physical health was positively ($b = 0.080$) associated with alcohol use.

The fully specified model (model D) predicts that each one-unit increase in marital strain is associated with a 0.223 increase in alcohol use ($p = .004$). Each one-unit increase in marital support is associated with a 0.146 increase in alcohol use. Although it bears repeating that this coefficient is not statistically significant by conventional standards ($p = .086$). Prior to the introduction of demographic controls (model C), length of marriage is negatively associated with alcohol use ($b = -0.007$). Once demographic controls are included (model D), length of marriage becomes nonsignificant. Whether or not a respondent's marriage-like relationship was officiated or not and number of marriages both appear inconsequential. Model D also shows that religiosity ($b = -0.194$, $p < .001$), household income ($b = 0.001$, $p = .065$), and sex ($b = 0.136$, $p = .026$) are, to various degrees, statistically significant predictors of alcohol use—these fixed effects are subsumed under “demographic controls” in Table 2.

Models E and F show results for the associations between marital characteristics and alcohol use controlling for additive genetic (A) and shared environment (C) random effects, respectively. Results from model E show that when additive genetic influence on alcohol use is controlled, the coefficient sizes for marital strain, marital support, and number of marriages on alcohol use were substantially reduced. The coefficient for marital strain was marginally significant and decreases by 45%, ($b = 0.223$, $p = .004$ to $b = 0.122$, $p = .083$). That is, when statistically controlling for marital support, health and family, demographics, and additive genetics, each unit increase in marital strain is associated with a 0.122 increase in alcohol use. The coefficient for marital support decreases by 65%, ($b = 0.146$, $p = .086$ to $b = 0.039$, $p = .434$).

Introducing the additive genetic random effect (model E) significantly improves model fit (compared to model D), resulting in a 34-point reduction in the log-likelihood ($\chi^2 = 68.01$, $p < .001$). This reveals that genes account for a substantial portion of the variance in alcohol use, with an estimated heritability (h^2) of approximately 54% ($A = 0.322$). Comparing predicted and actual alcohol use scores, Figure 2 illustrates visually how accounting for the additive genetic effect on alcohol use substantially improves model fit when compared with a fixed effects model. Introducing the shared environmental random effect (C) on alcohol use does not improve model fit when compared with model E (AE model for additive genetic effect), indicating that a negligible amount of variance in alcohol use is attributable to the shared environment net of the other variables in the model. Moreover, when all fixed effects are constant, a negligible amount of variance is attributable to the shared environment.

Table 2. GLMMs estimating the fixed effects of marriage quality on alcohol use appending genetic and shared environmental random effects.

	Model A b (SE)	Model B b (SE)	Model C b (SE)	Model D b (SE)	Model E b (SE)	Model F b (SE)	Model G b (SE)
Marital strain	0.233 (0.068)***	0.256 (0.070)***	0.225 (0.075)**	0.223 (0.077)**	0.122 (0.070) [†]	0.122 (0.070) [†]	0.128 (0.071) [†]
Marital support	0.246 (0.080)**	0.244 (0.082)**	0.198 (0.085)*	0.146 (0.085) [†]	0.039 (0.078)	0.039 (0.078)	0.039 (0.076)
Unmarried cohabiting	0.088 (0.145)	0.106 (0.148)	0.118 (0.143)	0.153 (0.154)	0.129 (0.144)	0.129 (0.144)	0.137 (0.144)
Length of marriage	-0.010 (0.002)***	-0.010 (0.002)***	-0.007 (0.002)**	-0.003 (0.004)	-0.004 (0.003)	-0.004 (0.003)	-0.003 (0.003)
Number of marriages	-0.039 (0.051)	-0.044 (0.052)	0.033 (0.055)	0.066 (0.058)	0.040 (0.059)	0.040 (0.059)	0.048 (0.058)
Spousal Alc. Prob.	0.004 (0.201)	0.006 (0.196)	0.073 (0.195)	0.081 (0.193)	-0.052 (0.174)	-0.052 (0.174)	-0.082 (0.176)
Peer strain		0.074 (0.072)	0.102 (0.072)	0.069 (0.070)	0.045 (0.067)	0.045 (0.067)	0.046 (0.067)
Family strain		-0.131 (0.071) [†]	-0.109 (0.070)	-0.044 (0.069)	-0.019 (0.066)	-0.019 (0.066)	-0.030 (0.066)
Peer support		-0.032 (0.055)	-0.055 (0.056)	-0.020 (0.056)	0.022 (0.052)	0.022 (0.052)	0.034 (0.052)
Family support		0.012 (0.076)	-0.017 (0.076)	0.061 (0.079)	0.048 (0.076)	0.048 (0.076)	0.046 (0.075)
Family and health controls							
Number of children			-0.047 (0.017)**	-0.028 (0.017) [†]	-0.039 (0.017)*	-0.039 (0.017)*	-0.039 (0.017)*
Pregnant			-0.780 (0.141)***	-0.531 (0.162)***	-0.564 (0.408)	-0.564 (0.408)	-0.058 (0.371)
Parental Alc. Prob.			-0.091 (0.071)	-0.089 (0.071)	-0.092 (0.073)	-0.092 (0.073)	-0.109 (0.073)
Resp. physical health			0.075 (0.035)*	0.080 (0.035)*	0.088 (0.034)**	0.088 (0.034)**	0.085 (0.033)**
Sp. physical health			0.089 (0.039)*	0.067 (0.038) [†]	0.046 (0.036)	0.046 (0.036)	0.041 (0.036)
Resp. mental health			-0.010 (0.041)	-0.025 (0.040)	-0.026 (0.037)	-0.026 (0.037)	-0.024 (0.037)
Sp. mental health			-0.036 (0.049)	-0.006 (0.048)	-0.009 (0.041)	-0.009 (0.041)	-0.007 (0.041)
Depression			-0.004 (0.021)	0.000 (0.021)	-0.003 (0.019)	-0.003 (0.019)	-0.002 (0.019)
Anxiety			-0.027 (0.059)	-0.029 (0.059)	-0.021 (0.045)	-0.021 (0.045)	-0.024 (0.047)
Demographic controls							
Constant	-0.003 (0.030)	-0.004 (0.030)	0.016 (0.033)	-0.047 (0.041)	-0.064 (0.048)	-0.064 (0.048)	-0.069 (0.048)
Genetic variance (A)					0.322 (0.041)	0.322 (0.041)	0.325 (0.041)
Marital strain							0.221 (0.091)
Marital support							0.184 (0.111)
Shared variance (C)							
Nonshared variance (E)							
Log likelihood	-939.408	-937.177	-923.861	-903.6149	-869.612	-869.612	-866.684

All continuous and nominal variables are mean centered. Binary variables remain uncentered. Variance inflation factors (VIF) for fixed effects ranged between 1.07 and 3.43, below the acceptable threshold of five described by James, Witten, Hastie, and Tibshirani (2013). Robust standard errors are in parentheses.

*** $p < .001$. ** $p < .01$. * $p < .05$. [†] $p < .1$.

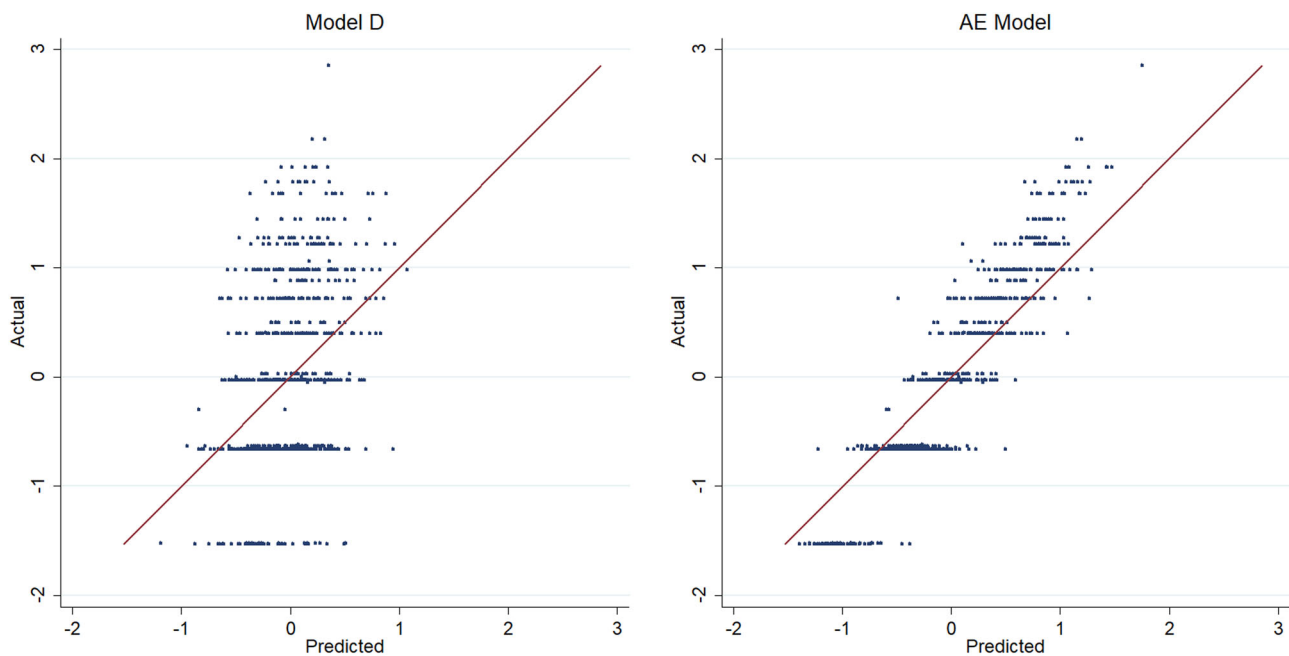


Figure 2. Scatterplot of actual and predicted alcohol use before and after appending statistical controls for genetic confounding.

Results reported in models A through F only control genetic variance in alcohol use, not genetic variance in marital strain and support. Thus far we have treated marital strain and support as individual-level covariates. By doing so they were allowed to retain all of their variance, both genetic and environmental. We next introduced random effects for marital strain and support, our predictors of interest, at the genetic level of analysis. Doing so marginally improved model fit ($\chi^2 = 5.86$, $p = .054$). However, regression coefficients and standard errors remained largely unchanged.

Discussion

This study's primary aim was to build on the extant body of research that examines influences of marital strain and support on alcohol use in a sample of married adults. Analyzing data collected from twin pairs participating in MIDUS II, genetically informative analyses were conducted using a specific type of GLMM, which allowed this study to take into account for genetic and shared environmental influences on recent alcohol use.

Our findings are somewhat consistent with past research (Berggren & Nystedt, 2006; Harford et al., 1994; Horwitz & White, 1991; Kendler et al., 2016, 2017). We found a relationship between alcohol use and marital strain and support when controlling for family/peer strain and support, family factors, and demographics. Consistent with Fischer et al. (2005), midlife adults who experience greater levels of marital strain drink more frequently and are likely to engage in problematic alcohol use. This could indicate that alcohol use is a coping behavior utilized in response to marital strain. Conversely, adults who perceive their marriage to have less strain tend to use alcohol less frequently. Our results, however, may indicate that midlife adults who perceived greater levels of support from their spouse also appear to use and abuse alcohol more frequently than those who perceive less

support, albeit to a lesser extent. This undermines the hypothesis that marriage is protective due to the social support it provides (Jerskey et al., 2010). This could suggest that relationships wherein romantic partners drink alcohol together tend to be perceived as more supportive. However, the relationship between marital support and alcohol use did not retain statistical significance ($p > .05$) by academic convention once accounting for demographic characteristics, and should be interpreted with caution.

After accounting for genetic confounding, marital strain continued to show a positive relationship with alcohol use. However, the magnitude of this relationship was substantially reduced and demonstrated marginal statistical significance ($p = .083$). Conversely, the weaker positive relationship between spousal support and alcohol use was almost entirely explained by genetics. One possible interpretation for this is that marital support is an artifact reflecting marital homophily. Romantic partners are selected based on similar heritable traits and behaviors (Fowler, Settle, & Christakis, 2011). This behavioral genetic similarity may then result in greater levels of perceived support, engaging in activities such as alcohol use together.

Several previous studies have taken care to note a lack of evidence that genes confound the effect of marriage on alcohol use (Kendler et al., 2016; Kretsch & Harden, 2014). Whereas our findings suggest that genes account for varying amounts of the covariance between alcohol use and marital characteristics, consistent with Dinescu et al. (2016). One possible explanation for this inconsistency is quite simple: the relationship between marital *status* and alcohol (mis)use is *not* substantially confounded by genetics, whereas the relationship between marital *characteristics* and alcohol (mis)use is confounded by genetics. Supporting this explanation, Kendler et al. (2017) found evidence of genetic confounding in the relationship between divorce, a marital outcome characterized by a reduced marital support and heightened

marital strain, and AUD. Granted, the method employed by Kendler et al. (2016, 2017) cannot distinguish between shared environmental and genetic confounding. However, we found that the shared environment had little influence, and there is consistent evidence that the shared environment tends to explain the least amount of variance on most traits (Polderman et al., 2015). Thus, it stands to reason that the confounding observed by Kendler et al. (2017) is largely genetic.

These inconsistencies could also be methodological. The majority of research addressing the issue of genetic confounding in the relationship between marriage and alcohol use simply compares discord within MZ and DZ pairs (Dinescu et al., 2016; Osler, McGue, Lund, & Christensen, 2008; Prescott & Kendler, 2001). Kendler et al. (2017) employ a more sophisticated approach, wherein shared environmental and genetic confounding is concluded to be present when the magnitude of the relationship between marital status/divorce and alcohol use is increased within clustered dyadic subsamples of increased relatedness. This study, aiming to produce a clearer representation of how genetics confound the relationship between marital characteristics and alcohol use, supposes that genetic confounding is meaningful if, once accounted for with appropriately specified random effects, the effect size of the relationship between marital characteristics and alcohol use is reduced to practical or statistical insignificance. Put simply, while these methods address the same substantive topic, genetic confounding, they do so using different criteria. To address these inconsistencies future research might consider: (a) comparing both methods to examine the extent which they agree; (b) employing the GLMM used in this study to test the robustness of other established relationships between a wider variety of traits and behaviors, including the relationship between marital status and AUD.

Ultimately, our results suggest that less perceived strain in marriages may protect midlife adults from problematic alcohol use. Therefore, attempts to defuse sources of interpersonal strain in romantic relationships, e.g. emotionally focused therapy (see Johnson, Hunsley, Greenberg, & Schindler, 2006), may help reduce alcohol use and misuse (e.g. frequent, heavy use), regardless of inherited traits and behaviors. Conversely, our findings imply that cultivating marital support would be ineffective and potentially iatrogenic.

Findings from this study should be interpreted with some caution due to several methodological limitations. First, while MIDUS is a panel study, it is decennial, with data collection only occurring approximately once per decade. In this instance we do not believe it theoretically justifiable to assume that characteristics of a marriage 10 years prior would influence current alcohol use. Consequently, our findings are from analyses of cross-sectional data and, therefore, the temporal order of the variables marital strain/support and alcohol use may be questionable. With that being said, the questions used to measure marital strain and support required participants to recall experiences during the past year, whereas the alcohol use measure was based on past

weeks and months. Thus it remains possible, however (un)likely (Greene, 1986),² that respondents' appraisal of marital stress/support is framed by events prior to their alcohol use.

Additionally, though somewhat robust of genetics, our results suggest that marital strain alone cannot produce alcohol problems. On average individuals with the highest score on our marital strain measure are only slightly more likely to engage in problematic alcohol use (e.g. HED or AUD). Other compounding factors are necessary to produce problem use.

Our analysis sample consisted of adult twins who are predominantly white, upper-middle class, each twin reporting that they were married or cohabiting in a marriage-like relationship during data collection for MIDUS II. A consequence is that it would be disingenuous to suggest that the mechanisms we have described in this study apply to other populations. However, while most of these sample characteristics are a feature of the MIDUS study and should simply be considered when framing these findings, the use of a twin sample is necessary for this type of statistical model, a potential limitation of this statistical approach to accounting for genetics. Research has demonstrated that twin samples represent the general population (Barnes & Boutwell, 2013). However, we would still recommend caution when making broader generalizations of results in this study.

Due to our decision to include both opposite and same-sex twins, it is possible that MZ and DZ twin pairs are not completely comparable because additional differences in the nonshared environment experienced by opposite sex pairs. However, given that the influence of this limitation is on the individual level (i.e. nonshared environment) our inclusion of gender as an individual-level covariate should account for the majority of bias that would have otherwise been introduced. With that being said, research indicates that the strength of the marital strain/support and alcohol use relationship may depend on gender (Li, Wilsnack, Wilsnack, & Kristjanson, 2010). Future research should further explore our study's findings to examine this possibility.

Finally, it should be noted that the shared environmental variance component does not necessarily define an experience as shared if both members of a twin pair were simply in the same room or household. If an environment or experience was experientially shared by a pair of twins, it can still produce nonshared environmental variance if said twins appraise that experience differently. For this reason, it is still important to control (a) measured family characteristics (e.g. parents' actual alcohol use), and (b) individual perceptions of the shared environment (e.g. twins' perception of their parents' alcohol use). These factors, determining which behaviors are modeled, learned, and adopted by respondents, are characteristics of family of origin socialization (Simons, Lin, & Gordon, 1998). Future research should

²Even though it is possible that these measures of marital strain/support precede alcohol use, it is more likely that respondents appraised all survey items using the most recent, relevant memories (Greene, 1986). It is therefore unlikely that responses to these survey items reflect the time ordering inherent to the survey items.

attempt to disentangle family of origin socialization and the shared/nonshared environment by examining the extent which measured, shared experiences in the family of origin are appraised (dis)similarly by siblings and twins.

Ultimately, Rabe-Hesketh and colleagues (2008) biometric model is a powerful tool that can be used in future research to answer important and informative research questions about how genetic influence and specific environmental variables are related to outcomes of importance across various disciplines. As we have demonstrated, it can be used as a method for controlling additive genetic and shared environmental influences on alcohol use. While alternative parameterizations for this model have required custom software (see Pawitan, Reilly, Nilsson, Cnattingius, & Lichtenstein, 2004), Rabe-Hesketh et al. (2008) note that this parameterization can be employed using STATA, allowing wider dissemination and model application. Future research employing family, twin, or sibling data can benefit from using this biometric model to identify specific nonshared environmental predictors of behaviors or other phenotypes net of additive genetic and shared environment influences.

Declarations of interest

The authors report no conflicts of interest.

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