

# Sex Differences in the Relative Influence of Marital Status and Parenthood on Alcohol Use Disorder Symptoms: A Multilevel Discordant Twin Design

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Marriage and parenthood are associated with alcohol use and use disorder (AUD), although they are confounded such that many studies struggle to identify their unique and/or causal effects. The present study utilized a genetically informed discordant twin design that strengthens the putative causal role of marital and parental status in the presentation of AUD symptoms by using each individual's cotwin as their own control while simultaneously modeling both predictors among men and women. Participants were 980 complete same-sex twin pairs from the Australian Twin Registry ( $M_{\text{age}} = 31.70$  [ $SD = 2.48$ ]; 71% women). Marital status, parental status, and past year AUD symptoms were assessed via semistructured interview. Three random-intercept generalized linear mixed models were fit in men and women including (a) marital status only, (b) parental status only, and (c) both marital and parental status; demographics, past year pregnancy, age of first drink, age of regular drinking, personality traits, and antisociality were included as covariates. Models tested for quasi-causal and familial effects. The sole-predictor marital status model (Model 1) provided the best fit among men, while the simultaneous-predictor marital and parental status model (Model 3) provided the best fit among women. Sole-predictor models showed familial effects of both predictors among men and quasi-causal and familial effects of both predictors among women; the simultaneous-predictor model revealed familial effects of marital status only among men and quasi-causal effects of parental status only among women. The present study elucidates important sex differences in the presentation of AUD among midlife adults in the context of notable developmental milestones.

### General Scientific Summary

Marriage, but not parenthood, influences alcohol use disorder (AUD) symptoms among adult men, while parenthood primarily influences AUD symptoms among adult women. Evidence for potentially causal influences of parenthood on AUD symptoms among women suggests that the closing of the “alcohol consumption gap” may be partially explained by declining birth rates. Initiatives to increase paternal involvement in childcare could serve to expand the protective effect of parenthood to fathers at risk for AUD.




**Keywords:** alcohol use disorder, marriage effect, parenthood, sex differences, twin study

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There is a robust literature examining the effects of developmentally salient tasks, such as marriage and parenthood, on alcohol use disorder (AUD) in adults (Chilcoat & Breslau, 1996; Dick et al., 2006; Gotham, Sher, & Wood, 2003; Kendler, Lönn, Salvatore, Sundquist, & Sundquist, 2016; Leonard & Eiden, 2007;

Leonard & Rothbard, 1999; Waldron et al., 2011). This literature has generally found a consistent effect of marital status on reduction of risk for AUD onset and mitigation of AUD symptoms, above and beyond “maturing out” effects that typically occur from the early through late 20s (Lee, Chassin, & MacKinnon, 2010;

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Miller-Tutzauer, Leonard, & Windle, 1991; O'Malley, 2004; Power, Rodgers, & Hope, 1999). However, the causal nature of this association remains equivocal despite longitudinal, twin, and longitudinal twin studies on the topic (Chilcoat & Breslau, 1996; Horwitz, White, & Howell-White, 1996; Kendler et al., 2016; Kretsch & Harden, 2014; Leonard & Rothbard, 1999; Little, Hاندley, Leuthe, & Chassin, 2009; Prescott & Kendler, 2001). These mixed findings are in part due to conflicting evidence regarding selection effects (Leonard & Rothbard, 1999), but may also be due to many studies omitting the potential role of parental status from their determination of marriage's role in mitigation of risk for AUD onset or persistence (Gotham et al., 2003; Heath, Jardine, & Martin, 1989; Kendler et al., 2016; Kretsch & Harden, 2014; Lee, Chassin, & MacKinnon, 2015), despite evidence that parenthood is associated with reduced alcohol consumption and AUD risk (Chilcoat & Breslau, 1996; Fergusson, Boden, & Horwood, 2012; Little et al., 2009) and strongly confounded with marital status. While some prior studies on the protective effects of marriage have included examination of parental status to further tease apart uniquely contributing effects (Bachman, Wadsworth, O'Malley, & Johnston, 1997; Chilcoat & Breslau, 1996; Christie-Mizell & Peralta, 2009; Oesterle, Hawkins, & Hill, 2011; Power et al., 1999), many have not.

There also remains debate about differential effects among men and women (Leonard & Eiden, 2007). Previous studies have found that marriage serves as a protective factor for AUD among both men and women (Chilcoat & Breslau, 1996; Kretsch & Harden, 2014; Leonard & Rothbard, 1999). Others have found patterns of enhanced protective effects for men (Barr et al., 2019; Duncan, Wilkerson, & England, 2006; Kiecolt-Glaser & Newton, 2001; Power et al., 1999; Umberson, 1992), and still others have found stronger protective effects for women (Horwitz et al., 1996; Kendler et al., 2016). One longitudinal twin study found potentially causal effects of marriage on alcohol use among men (Salvatore, Gardner, & Kendler, 2019), while another suggested that reductions in alcohol use after marriage may be due to familial, rather than causal, factors among women (Prescott & Kendler, 2001). With respect to parental status, protective effects of parenthood in the context of AUD tend to be more consistently magnified among women (Fergusson et al., 2012). The reduction in heavy drinking between the early 20s and early 30s observed among women is primarily associated with parental status over and above marital status, while parental status does not appear to influence heavy drinking among married men (Christie-Mizell & Peralta, 2009; Power et al., 1999); rather, fathers' declines in heavy drinking are better explained by marriage (Bachman, Wadsworth, O'Malley, Johnston, & Schulenberg, 2013; Leonard & Eiden, 2007). Similarly, having a pregnant spouse exerts no influence over men's drinking behavior (Leonard & Eiden, 2007; O'Malley, 2004); in fact, one quarter of men are at risk for AUD both during their partner's pregnancy and through at least the first postnatal year (Condon, Corkindale, & Boyce, 2004). This points toward substantive sex differences in the influence of salient developmental tasks on AUD symptoms.

### Present Study

The roles of marriage and parenthood in the presentation of AUD symptoms have received empirical attention for decades.

However, their potential simultaneous influence, and their differing effects in men and women, has, to our knowledge, not yet been examined in a genetically informed design. The present study sought to expand on the developmental alcohol use literature by implementing a discordant twin design to disentangle potential causal ("quasi-causal") influences of marital and parental statuses on AUD symptoms in midadulthood, with a focus on how these factors may operate differently among men and women. In light of previous literature, it was expected that (a) marital status, but not parental status, would exert a quasi-causal effect on AUD symptoms among men, (b) marital status would exert a familial effect and parental status would exert a quasi-causal effect on AUD symptoms among women, and (c) among women, the effect of parental status would eclipse that of marital status when both predictors were modeled simultaneously.

## Method

### Participants and Procedure

Participants were 980 complete same-sex twin pairs of known zygosity (monozygotic [MZ] pair  $N = 565$ ; dizygotic [DZ] pair  $N = 415$ ) from the Australian Twin Registry Cohort III ( $M_{\text{age}} = 31.70$  [ $SD = 2.48$ ], range = 27–37 [one twin pair was age 40]; 71% women; see Lynskey et al. (2012) for more information about participants). Participants were surveyed by computer-assisted telephone interview (CATI) in 2005–2009 (participation rate = 76%) and a follow-up survey administered via the Internet or a mailed paper-and-pencil questionnaire (completion rate = 94%). The original data collection was approved by the Institutional Review Boards at Washington University and Queensland Institute of Medical Research-Berghofer, and secondary analysis of these data was determined to be exempt by the University of Missouri Institutional Review Board.<sup>1</sup>

### Measures

**Marital and parental status.** As part of the CATI interview, participants were asked if they were currently never married, married, widowed, separated, or divorced. Never married (40% of the sample) and married (55% of the sample) were used as comparison groups for analysis of marital status; those who reported being widowed, separated, and divorced (5% of the sample) were excluded from analyses, leaving a final analytic sample of 935 pairs ( $N = 1,870$ ;  $M_{\text{age}} = 31.65$  [ $SD = 2.47$ ]; 70% women).

Women were asked if they had ever been pregnant; if endorsed, they were asked if they were currently pregnant and how old they were the first time they got pregnant. Women who reported being currently pregnant and/or whose age of first pregnancy was equal to their current age were considered to have been pregnant in the past year. All participants were asked if they had children; those who endorsed this item were coded positively for parental status.

**Past year AUD symptoms.** Assessment of past year AUD symptoms was based on the Australian version of the Semi-Structured Assessment of the Genetics of Alcoholism (SSAGA-

<sup>1</sup> "Secondary Data Analyses of Australian Twin Registry Cohorts II and III"; IRB 1209709 C, Exempt Application 119185.

OZ; Bucholz et al., 1994; Heath et al., 1997) and administered via CATI. Alcohol abuse and dependence were assessed using *DSM-IV* criteria and scored according to the *DSM-5* criteria for AUD (absent the criterion of craving, which was not included in the *DSM-IV*). A cumulative symptom score was created by summing endorsed symptoms.

#### Covariates.

**Demographics.** Participants were asked to report their highest educational level attained (primary incomplete, primary completed, year 8 completed, year 9 completed, year 10 completed, year 11 completed, year 12 completed, technical college, undergraduate degree, and graduate degree). Due to low prevalence of the 6 lowest categories, they were collapsed into a “high school or less” group (24% of the sample); the rest of the sample was distributed across technical college (28%), undergraduate degree (28%), and graduate degree (20%). Neighborhood disadvantage served as an index of socioeconomic status and was calculated using data from participants’ postal codes matched to census data containing indicators of disadvantage (e.g., low income, subsidized housing; Slutske, Piasecki, Deutsch, Statham, & Martin, 2019).

**Alcohol use history.** As part of the SSAGA-OZ, participants were asked how old they were when they consumed their first drink of alcohol (“age of first drink” [AFD]) and how old they were when they began drinking regularly (i.e. at least once per month for 6 months or more; “age of regular drinking” [ARD]).

**Big Five personality traits.** Big 5 personality traits were assessed using an adapted NEO PI-R (Costa & McCrae, 1992a, 1992b; Dash et al., 2019) and administered via self-report survey within 2 weeks of the CATI interview. The questionnaire consisted of 74 items scored on a 1 (strongly disagree) to 5 (strongly agree) scale. Scores were generated by computing the item means for each scale. Internal consistency reliabilities (coefficient alphas) for neuroticism, extraversion, openness to experience, agreeableness, and conscientiousness were .89, .85, .77, .81, and .85, respectively.

**Adult antisocial behavior (AAB).** As part of the CATI interview, participants were administered a *DSM-IV* diagnostic assessment for antisocial personality disorder (ASPD), which queried behaviors that occurred since age 15. Participants were considered to display AAB if they met the adult criteria for ASPD. A majority of participants meeting criteria for AAB (89%) also displayed conduct disorder prior to age 15, and therefore met full criteria for ASPD.

## Analytic Plan

Analyses were conducted using SAS Version 9.4 (SAS Inc., 2014). To examine the effect of marital and parental status on past year AUD symptoms, two-level generalized linear mixed models (GLMMs) were run using PROC GLIMMIX. GLMMs are a statistical procedure used for the analysis of clustered data with nonnormally distributed outcome variables (Hedeker, 2005). Twin data are clustered, with individual twins (level 1) nested within twin pairs (level 2). Random intercept models were used to estimate level 1 and level 2 variances. Predictors were coded to test within-pair (i.e. comparison of twin and cotwin’s deviations from their pair average; quasi-causal) and between-pair (i.e. comparison of twin pair averages across twin pairs; familial) effects (Slutske et al., 2014). A negative binomial distribution and log link function were used due to the positive skewness of the past year AUD symptom variable. Coefficients from the multi-

level models were exponentiated to produce incidence rate ratios (IRR; Slutske et al., 2019).

First, preliminary models were fit in the full sample to probe for evidence of potential sex differences (see [online supplemental materials](#)). Because these models provided evidence for sex differences, particularly with respect to parental status, we proceeded to model the data in men and women separately. At the first step, models were run at the individual level. These models accounted for the clustering of twin pair data so as to approximate independent data. Never married individuals served as the reference group for the marital status variable, and nonparents served as the reference group for the parental status variable. Base models for (a) marital status only, (b) parental status only, and (c) marital and parental status together were fit with zygosity as a covariate; the simultaneous-predictor model (Model 3) also included a marital status by parental status interaction term. Significant interactions were carried forward to the fully adjusted model. Fully adjusted models for each set of predictors included zygosity, past year pregnancy, age, educational attainment, neighborhood disadvantage, personality traits, age of first drink, age of regular drinking, and AAB as covariates. Models were also run with covariates entered in blocks (i.e. predictor[s] and demographics, predictor[s] and personality traits, predictor[s] and alcohol use, and predictor[s] and AAB; “partially-adjusted models”) to determine if the inclusion of all covariates in a single model was producing misleading results.

Next, cotwin control models were fit to examine potential quasi-causal and familial effects of marital status and parental status on past year AUD symptoms (McGue, Osler, & Christensen, 2010). Such discordant twin designs have the advantage of controlling for the potential confounding factors of genes (completely for MZ twins and partially for DZ twins) and familial environment (completely for both MZ and DZ twins), using each individual’s cotwin as their own control and thereby permitting stronger causal inference even in cross-sectional data. First, base models including each set of predictors, zygosity, and a zygosity by predictor interaction term were fit (a significant zygosity by predictor interaction term would indicate the presence of genetic confounding); the simultaneous-predictor model also included a within-pair marital status by within-pair parental status interaction term. Significant interactions were carried forward to the fully adjusted model. Fully adjusted cotwin control models for each set of predictors were run first using data from both MZ and DZ pairs and subsequently in MZ pairs only. MZ-only models fully control for both genetic and shared environmental factors, thereby providing stronger causal inference and a more stringent test of genetic confounding; a reduction in effect in the MZ model compared to the MZ-DZ model would indicate the presence of such confounding. Fully adjusted models included zygosity (for the MZ-DZ model), past year pregnancy, age, educational attainment, neighborhood disadvantage, personality traits, age of first drink, age of regular drinking, and AAB as covariates; partially adjusted models (as described above) were also run for all cotwin control analyses.

The fits of the sole-predictor models were compared using the Akaike’s information criterion (AIC), the sample size adjusted Akaike’s information criterion (AICC), and Bayesian information criterion (BIC). The fits of nested models (i.e. each fully adjusted sole-predictor model compared to the fully adjusted model with quasi-causal effects of both marital and parental status) were compared using log-likelihood ratio tests.

## Results

### Sample Characteristics

Forty-one percent of twin pairs were discordant for marital status, and 33% of pairs were discordant for parental status. Forty-three percent of the analytic sample was married with children, 14% was married without children, 7% was never married with children, and 35% was never married without children. Nineteen percent of men and 8% of women met criteria for at least two AUD symptoms in the past year (i.e. clinically significant disorder). Descriptive statistics and effect sizes for past year AUD symptoms among the different marital and parental status groups are available in Table 1. Among men, moderate effects emerged for marital status overall, and within the two parental status groups (i.e. never married nonparents vs. married nonparents [ $d = 0.35$ ], never married parents vs. married parents [ $d = 0.54$ ]; see Table 1). The effect of parental status was negligible within both never married ( $d = 0.16$ , 95% CI [-0.09, 0.41]) and married ( $d = 0.11$ , 95% CI [-0.12, 0.33]) men. Effects for marital and parental status overall were small to moderate among women. Marital status appeared to have a small effect among nonmothers ( $d = 0.27$ ), but no effect among mothers ( $d = 0.12$ ; see Table 1). However, there was an effect of parental status within never married women ( $d = 0.29$ , 95% CI [0.12, 0.46]), such that never married mothers averaged fewer AUD symptoms than never married nonmothers.

### Model Fit

Comparing the sole-predictor cotwin control models, the marital status model outperformed the parental status model among both men and women. However, nested model comparisons with the simultaneous-predictor model revealed sex differences (see Table 2). Among men, the model including marital status only did not

differ in fit from the model including both marital and parental status, indicating that the sole-predictor marital status model was the more parsimonious model for this group. Among women, the model including both marital and parental statuses appeared to best fit the data. The same results were obtained in the more stringent MZ-only models. Model results for the three sets of models (marital status, parental status, and marital and parental status) fit at the individual and twin pair levels are detailed below.

### Sole-Predictor Model: Marital Status

**Individual-level models.** Marital status significantly predicted AUD symptoms in the base model for both men ( $IRR = 0.46$ , 95% CI [0.34, 0.63],  $p < .0001$ ) and women ( $IRR = 0.42$ , 95% CI [0.32, 0.55],  $p < .0001$ ). After adjusting for covariates, marital status remained significant, with being married reducing the expected number of AUD symptoms by 47% in both men and women ( $IRR = 0.53$ ; see Table 3, column a). Results did not differ across partially and fully adjusted models.

**Cotwin control models.** The familial effect of marital status was significant in the base model among men ( $IRR = 0.34$ , 95% CI [0.22, 0.54],  $p < .0001$ ), but the quasi-causal effect was not ( $IRR = 0.57$ , 95% CI [0.15, 2.09],  $p = .39$ ). Among women, both quasi-causal and familial effects were significant ( $IRR = 0.43$ , 95% CI [0.20, 0.93],  $p = .03$ ;  $IRR = 0.43$ , 95% CI [0.28, 0.65],  $p < .0001$ ). The interactions with zygosity were nonsignificant ( $p = .85-.99$ ) and therefore not carried forward. The same pattern of significance held after adjusting for covariates in the fully adjusted model, although the magnitude of the familial effect among women was notably reduced (from  $IRR = 0.43$  to  $IRR = 0.61$ ; see Table 3, column b). Results did not differ across partially and fully adjusted models.

In the unadjusted MZ-only model, the magnitude of the familial effect for marital status was retained among men

Table 1  
Descriptive Statistics of AUD Symptoms by Sex, Marital Status, and Parental Status

Characteristic/status	<i>N</i>	<i>M</i> [95% CI]	<i>SD</i>	Range	Cohen's <i>d</i> [95% CI]
<b>Men</b>					
Never married	252	1.32 [1.07, 1.57]	2.01	0-10	0.44 [0.27, 0.61]
Married	300	0.60 [0.46, 0.74]	1.19	0-8	
Nonparent	298	1.13 [0.92, 1.33]	1.80	0-9	0.27 [0.10, 0.44]
Parent	254	0.69 [0.52, 0.87]	1.43	0-10	
Never married nonparent	222	1.27 [1.02, 1.53]	1.93	0-9	0.35 [0.12, 0.58]
Married nonparent	76	0.70 [0.40, 0.99]	1.29	0-6	
Never married parent	30	1.63 [0.69, 2.58]	2.53	0-10	0.54 [0.29, 0.79]
Married parent	224	0.57 [0.41, 0.72]	1.16	0-8	
<b>Women</b>					
Never married	538	0.36 [0.31, 0.41]	0.91	0-8	
Married	780	0.24 [0.18, 0.29]	0.73	0-8	0.31 [0.20, 0.42]
Nonparent	626	0.50 [0.42, 0.59]	1.09	0-8	0.31 [0.20, 0.42]
Parent	692	0.22 [0.17, 0.28]	0.69	0-8	
Never married nonparent	432	0.59 [0.48, 0.69]	1.15	0-7	0.27 [0.11, 0.43]
Married nonparent	194	0.31 [0.19, 0.44]	0.91	0-8	
Never married parent	106	0.30 [0.14, 0.46]	0.82	0-5	0.12 [-0.03, 0.27]
Married parent	586	0.21 [0.16, 0.26]	0.67	0-8	

Note. AUD = alcohol use disorder. Effect size conventions: 0.20 = small, 0.50 = moderate, 0.80 = large. Shading denotes groups to which effect sizes correspond.



Table 2

Model Fit Statistics and Information Criteria for Fully Adjusted Cotwin Control Models Predicting AUD Symptoms

Predictor(s)	MZ-DZ models				
	AIC	AICC	BIC	-2 Log likelihood	Nested model comparison*
Men (N = 552)					
<b>Marital status</b>	<b>1132.66</b>	<b>1133.99</b>	<b>1193.12</b>	<b>1098.66</b>	LR(2) = 1.96, <i>p</i> = .38
Parental status	1221.78	1223.03	1283.39	1187.78	LR(2) = 180.20, <i>p</i> < .0001
Marital & parental statuses	1135.68	1137.34	1203.26	1097.68	
Women (N = 1,318)					
Marital status	1619.96	1620.56	1699.49	1583.96	LR(3) = 29.56, <i>p</i> < .0001
Parental status	1774.56	1775.56	1856.01	1738.56	LR(3) = 169.38, <i>p</i> < .0001
<b>Marital &amp; parental statuses</b>	<b>1611.18</b>	<b>1611.99</b>	<b>1703.96</b>	<b>1569.18</b>	
Predictor(s)	MZ-only models				
	AIC	AICC	BIC	-2 Log likelihood	Nested model comparison*
Men (N = 329)					
<b>Marital status</b>	<b>698.68</b>	<b>700.66</b>	<b>747.58</b>	<b>666.68</b>	LR(2) = 3.08, <i>p</i> = .21
Parental status	745.66	747.53	795.45	713.66	LR(2) = 97.04, <i>p</i> < .0001
Marital & parental statuses	701.14	703.65	756.16	665.14	
Women (N = 749)					
Marital status	892.94	893.90	958.33	858.94	LR(2) = 27.12, <i>p</i> < .0001
Parental status	988.30	989.15	1055.64	954.30	LR(2) = 217.84, <i>p</i> < .0001
<b>Marital &amp; parental statuses</b>	<b>883.38</b>	<b>884.57</b>	<b>956.46</b>	<b>845.38</b>	

Note. Lower value indicates better fit. Bold indicates preferred solution. AUD = alcohol use disorder; MZ = monozygotic; DZ = dizygotic; AIC = Akaike's information criterion; AICC = sample size adjusted Akaike's information criterion; BIC = Bayesian information criterion; LR = likelihood ratio test.

\* Nested model comparison = sole predictor model compared to simultaneous predictor model.

(*IRR* = 0.29, 95% CI [0.16, 0.53], *p* < .0001) and the quasi-causal effect remained nonsignificant (*IRR* = 0.62, 95% CI [0.35, 1.10], *p* = .10). Among women, both quasi-causal and familial effects were significant and similar in magnitude to those in the unadjusted MZ-DZ model (*IRR* = 0.42, 95% CI [0.25, 0.70], *p* = .001; *IRR* = 0.41, 95% CI [0.22, 0.77], *p* = .006). In the fully adjusted MZ-only model, effects among men were retained while effects among women were rendered nonsignificant (see Table 3, column c). Results did not differ across partially and fully adjusted models, with the exception of the quasi-causal effect of marital status among women retaining significance in the partially adjusted models (*IRR* = 0.47–0.53, *p* = .006–.02).

### Sole-Predictor Model: Parental Status

**Individual-level models.** Parental status significantly predicted AUD symptoms in the base model for both men (*IRR* = 0.64, 95% CI [0.47, 0.87], *p* = .004) and women (*IRR* = 0.43, 95% CI [0.33, 0.56], *p* < .0001). After adjusting for covariates, parental status remained significant, with being a parent reducing the expected number of AUD symptoms by 40% in men and 53% in women (see Table 4, column a). Results did not differ across partially and fully adjusted models.

**Cotwin control models.** The familial effect of parental status was significant in the base model among men (*IRR* = 0.53, 95% CI [0.34, 0.83], *p* = .006), but the quasi-causal effect was not (*IRR* = 0.75, 95% CI [0.20, 2.76], *p* = .66). Among women, both quasi-causal and familial effects were significant (*IRR* = 0.28, 95% CI [0.13, 0.60], *p* = .001; *IRR* = 0.42, 95% CI [0.28, 0.62], *p* < .0001). The interactions with zygosity were nonsignificant

(*p* = .17–.99) and therefore not carried forward. The same pattern of significance held after adjusting for covariates, although the magnitude of the quasi-causal effect among women was reduced (from *IRR* = 0.28 to *IRR* = 0.45; see Table 4, column b). Results did not differ across partially and fully adjusted models.

In the base MZ-only models, neither the quasi-causal nor familial effect of parental status was significant among men (*IRR* = 0.74, 95% CI [0.41, 1.35], *p* = .33; *IRR* = 0.67, 95% CI [0.37, 1.22], *p* = .19), while both exerted an influence among women (*IRR* = 0.36, 95% CI [0.22, 0.58], *p* < .0001; *IRR* = 0.47, 95% CI [0.26, 0.85], *p* = .01). In the fully adjusted MZ-only models, the effects were of the same pattern and approximate magnitude (see Table 4, column c). Results did not differ across partially and fully adjusted models.

### Simultaneous-Predictor Model: Marital and Parental Status

**Individual-level models.** Marital status, but not parental status, predicted AUD symptoms in the base model among men (*IRR* = 0.47, 95% CI [0.31, 0.69], *p* = .0002; *IRR* = 0.99, 95% CI [0.67, 1.46], *p* = .94). Among women, both marital and parental status predicted AUD symptoms (*IRR* = 0.56, 95% CI [0.41, 0.76], *p* = .0002; *IRR* = 0.57, 95% CI [0.41, 0.78], *p* = .0004). The marital by parental status interactions (*p* = .27–.29), were nonsignificant and therefore not carried forward. After adjusting for covariates, parental status remained significant among women, and marital status was marginally, but significantly, associated with AUD symptoms in both men and women (see Table 5, column a). Results did not differ across partially- and fully adjusted models.

Table 3  
Incident Rate Ratios (IRR) for Fully Adjusted Models of Marital Status Predicting AUD Symptoms

Model variables	(a) Individual-level			(b) MZ-DZ			(c) MZ-only		
	IRR	[95% CI]	<i>p</i>	IRR	[95% CI]	<i>p</i>	IRR	[95% CI]	<i>p</i>
Men									
Predictor									
Marital status	<b>0.53</b>	<b>[0.38, 0.74]</b>	<b>.0002</b>	—	—	—	—	—	—
BP marital status	—	—	—	<b>0.41</b>	<b>[0.26, 0.65]</b>	<b>.0001</b>	<b>0.35</b>	<b>[0.19, 0.63]</b>	<b>.0005</b>
WP marital status	—	—	—	0.72	[0.45, 1.16]	.18	0.63	[0.32, 1.23]	.17
Covariate									
Zygoty	1.17	[0.83, 1.65]	.38	1.22	[0.85, 1.74]	.28	—	—	—
Age	1.00	[0.93, 1.08]	.98	1.01	[0.94, 1.09]	.73	1.02	[0.92, 1.13]	.70
Education	<b>0.76</b>	<b>[0.66, 0.88]</b>	<b>.0003</b>	<b>0.74</b>	<b>[0.64, 0.86]</b>	<b>.0001</b>	0.82	[0.67, 1.01]	.06
Neighborhood disadvantage	1.00	[0.94, 1.07]	.88	1.00	[0.94, 1.07]	.93	0.99	[0.91, 1.08]	.90
Neuroticism	<b>1.82</b>	<b>[1.39, 2.37]</b>	<b>&lt;.0001</b>	<b>1.80</b>	<b>[1.38, 2.35]</b>	<b>&lt;.0001</b>	<b>1.66</b>	<b>[1.14, 2.41]</b>	<b>.008</b>
Extraversion	1.31	[0.95, 1.81]	.10	<b>1.42</b>	<b>[1.02, 1.97]</b>	<b>.04</b>	1.13	[0.72, 1.78]	.59
Openness to experience	0.97	[0.66, 1.42]	.88	0.93	[0.63, 1.38]	.73	0.80	[0.48, 1.35]	.41
Agreeableness	<b>0.71</b>	<b>[0.50, 1.00]</b>	<b>.05</b>	0.72	[0.51, 1.01]	.06	0.68	[0.42, 1.10]	.12
Conscientiousness	0.98	[0.71, 1.36]	.92	1.00	[0.73, 1.39]	.98	1.11	[0.72, 1.71]	.64
Age of first drink	<b>0.89</b>	<b>[0.82, 0.95]</b>	<b>.002</b>	<b>0.90</b>	<b>[0.83, 0.96]</b>	<b>.004</b>	0.92	[0.82, 1.02]	.11
Age of regular drinking	1.04	[0.99, 1.09]	.16	1.04	[0.98, 1.09]	.17	<b>1.08</b>	<b>[1.01, 1.16]</b>	<b>.03</b>
AAB	1.71	[0.95, 3.08]	.08	1.79	[0.99, 3.24]	.06	<b>2.58</b>	<b>[1.21, 5.52]</b>	<b>.01</b>
Women									
Predictor									
Marital status	<b>0.53</b>	<b>[0.40, 0.70]</b>	<b>&lt;.0001</b>	—	—	—	—	—	—
BP marital status	—	—	—	<b>0.61</b>	<b>[0.40, 0.91]</b>	<b>.02</b>	0.64	[0.36, 1.15]	.13
WP marital status	—	—	—	<b>0.50</b>	<b>[0.34, 0.74]</b>	<b>.0005</b>	0.62	[0.36, 1.07]	.09*
Covariate									
Zygoty	0.83	[0.61, 1.13]	.24	0.830	[0.61, 1.13]	.32	—	—	—
Past year pregnancy	0.67	[0.39, 1.15]	.14	0.67	[0.39, 1.15]	.14	0.47	[0.20, 1.11]	.08
Age	0.97	[0.91, 1.03]	.27	0.96	[0.91, 1.03]	.25	0.96	[0.88, 1.05]	.40
Education	1.02	[0.90, 1.16]	.72	1.02	[0.90, 1.16]	.75	0.92	[0.77, 1.10]	.37
Neighborhood disadvantage	1.00	[0.94, 1.05]	.87	0.99	[0.94, 1.05]	.82	1.04	[0.96, 1.13]	.38
Neuroticism	<b>1.76</b>	<b>[1.40, 2.21]</b>	<b>&lt;.0001</b>	<b>1.74</b>	<b>[1.38, 2.20]</b>	<b>&lt;.0001</b>	<b>1.98</b>	<b>[1.41, 2.77]</b>	<b>&lt;.0001</b>
Extraversion	<b>1.78</b>	<b>[1.30, 2.45]</b>	<b>.0004</b>	<b>1.74</b>	<b>[1.26, 2.39]</b>	<b>.0007</b>	<b>1.96</b>	<b>[1.25, 3.06]</b>	<b>.003</b>
Openness to experience	1.00	[0.73, 1.36]	.99	0.99	[0.72, 1.35]	.93	1.05	[0.67, 1.64]	.82
Agreeableness	0.86	[0.62, 1.18]	.34	0.87	[0.63, 1.21]	.40	0.71	[0.44, 1.16]	.17
Conscientiousness	<b>0.73</b>	<b>[0.55, 0.96]</b>	<b>.03</b>	<b>0.74</b>	<b>[0.55, 0.98]</b>	<b>.04</b>	0.71	[0.47, 1.09]	.12
Age of first drink	<b>0.83</b>	<b>[0.78, 0.89]</b>	<b>&lt;.0001</b>	<b>0.83</b>	<b>[0.78, 0.89]</b>	<b>&lt;.0001</b>	<b>0.84</b>	<b>[0.76, 0.93]</b>	<b>.0005</b>
Age of regular drinking	<b>1.04</b>	<b>[1.00, 1.07]</b>	<b>.04</b>	1.03	[1.00, 1.07]	.06	1.04	[0.99, 1.09]	.12
AAB	1.99	[0.79, 5.01]	.15	2.11	[0.82, 5.38]	.12	2.08	[0.59, 7.32]	.25

Note. Reference categories: sex = female, marital status = never married. Bold indicates significance,  $p < .05$ . AUD = alcohol use disorder; MZ = monozygotic; DZ = dizygotic; BP = between-pair; WP = within-pair; AAB = adult antisocial behavior.

\* Effect was significant in partially-adjusted models ( $IRR = .47-.53$ ,  $p = .006-.02$ ).

**Cotwin control models.** Only familial effects of marital status predicted AUD symptoms in the base model among men ( $IRR = 0.32$ , 95% CI [0.18, 0.59],  $p = .0002$ ). The zygoty by predictor interactions ( $p = .61-.64$ ), as well as the marital by parental status interaction ( $p = .61$ ), were nonsignificant and were therefore not carried forward. Among women, the familial effects of marital status predicted AUD symptoms ( $IRR = 0.59$ , 95% CI [0.35, 0.98],  $p = .04$ ), as did quasi-causal and familial effects of parental status ( $IRR = 0.23$ , 95% CI [0.09, 0.55],  $p = .001$ ;  $IRR = 0.60$ , 95% CI [0.36, 0.99],  $p = .05$ ); the weakness of both familial effects should be noted. The zygoty by within-pair parental status interaction was significant among women ( $p = .02$ ) and therefore carried forward to the full model; the marital by parental status interaction was nonsignificant and therefore not carried forward ( $p = .37$ ). In the fully adjusted model, only the familial effect of marital status was significant among men, and only the quasi-causal effect of

parental status was significant among women; the zygoty by within-pair parental status interaction was reduced to marginality ( $p = .08$  see Table 5, column b). Results did not differ across partially and fully adjusted models, with the exception of the quasi-causal effect of marital status retaining significance in the partially adjusted models ( $IRR = 0.51-0.66$ ,  $p = .001-.04$ ).

In the base MZ-only model, the familial effect of marital status was significant among men ( $IRR = 0.18$ , 95% CI [0.07, 0.42],  $p = .0001$ ) and, marginally, women ( $IRR = 0.47$ , 95% CI [0.22, 1.01],  $p = .05$ ). The quasi-causal effect of parental status was also significant among women ( $IRR = 0.38$ , 95% CI [0.21, 0.67],  $p = .0001$ ). The marital by parental status interaction was nonsignificant and therefore not carried forward ( $p = .42-.62$ ). The magnitude of the familial marital status effect among men was retained in the fully adjusted MZ-only model, but was rendered nonsignificant among women ( $\Delta IRR = 0.29$ ). The quasi-causal effect of parental status among women was re-

Table 4  
Incident Rate Ratios (IRR) for Fully Adjusted Models of Parental Status Predicting AUD Symptoms

Model variables	(a) Individual-level			(b) MZ-DZ			(c) MZ-only		
	IRR	[95% CI]	<i>p</i>	IRR	[95% CI]	<i>p</i>	IRR	[95% CI]	<i>p</i>
Men									
Predictor									
Parental status	<b>0.60</b>	<b>[0.43, 0.84]</b>	<b>.003</b>	—	—	—	—	—	—
BP parental status	—	—	—	<b>0.51</b>	<b>[0.32, 0.79]</b>	<b>.003</b>	0.66	[0.37, 1.20]	.18
WP parental status	—	—	—	0.73	[0.46, 1.18]	.20	0.65	[0.32, 1.34]	.24
Covariate									
Zygosity	1.16	[0.83, 1.63]	.39	1.18	[0.84, 1.66]	.35	—	—	—
Age	1.03	[0.96, 1.11]	.42	1.04	[0.97, 1.12]	.30	1.04	[0.94, 1.16]	.43
Education	<b>0.78</b>	<b>[0.68, 0.90]</b>	<b>.0007</b>	<b>0.78</b>	<b>[0.67, 0.89]</b>	<b>.0005</b>	0.83	[0.68, 1.03]	.09
Neighborhood disadvantage	0.99	[0.93, 1.05]	.74	0.99	[0.93, 1.05]	.71	0.98	[0.90, 1.07]	.64
Neuroticism	<b>1.84</b>	<b>[1.42, 2.38]</b>	<b>&lt;.0001</b>	<b>1.81</b>	<b>[1.39, 2.35]</b>	<b>&lt;.0001</b>	<b>1.75</b>	<b>[1.19, 2.56]</b>	<b>.004</b>
Extraversion	<b>1.42</b>	<b>[1.03, 1.94]</b>	<b>.03</b>	<b>1.44</b>	<b>[1.04, 1.97]</b>	<b>.03</b>	1.11	[0.70, 1.74]	.66
Openness to experience	0.95	[0.65, 1.39]	.80	0.95	[0.65, 1.38]	.79	0.81	[0.48, 1.37]	.43
Agreeableness	<b>0.66</b>	<b>[0.47, 0.93]</b>	<b>.02</b>	<b>0.66</b>	<b>[0.47, 0.92]</b>	<b>.02</b>	<b>0.61</b>	<b>[0.38, 1.00]</b>	<b>.05</b>
Conscientiousness	0.90	[0.66, 1.23]	.52	0.90	[0.66, 1.23]	.53	0.97	[0.62, 1.50]	.88
Age of first drink	<b>0.88</b>	<b>[0.82, 0.95]</b>	<b>.0007</b>	<b>0.88</b>	<b>[0.82, 0.95]</b>	<b>.001</b>	<b>0.89</b>	<b>[0.79, 0.99]</b>	<b>.04</b>
Age of regular drinking	1.04	[0.99, 1.10]	.11	1.04	[0.99, 1.10]	.11	<b>1.09</b>	<b>[1.01, 1.17]</b>	<b>.02</b>
AAB	1.45	[0.82, 2.58]	.20	1.50	[0.85, 2.67]	.16	<b>2.21</b>	<b>[1.01, 4.86]</b>	<b>.05</b>
Women									
Predictor									
Parental status	<b>0.47</b>	<b>[0.35, 0.62]</b>	<b>&lt;.0001</b>	—	—	—	—	—	—
BP parental status	—	—	—	<b>0.48</b>	<b>[0.32, 0.72]</b>	<b>.0003</b>	<b>0.56</b>	<b>[0.32, 0.98]</b>	<b>.04</b>
WP parental status	—	—	—	<b>0.45</b>	<b>[0.31, 0.67]</b>	<b>&lt;.0001</b>	<b>0.36</b>	<b>[0.22, 0.61]</b>	<b>.0001</b>
Covariate									
Past year pregnancy	0.89	[0.66, 1.19]	.42	0.89	[0.66, 1.19]	.42	<b>0.35</b>	<b>[0.15, 0.81]</b>	<b>.01</b>
Zygosity	<b>0.49</b>	<b>[0.29, 0.84]</b>	<b>.009</b>	<b>0.49</b>	<b>[0.29, 0.84]</b>	<b>.009</b>	—	—	—
Age	0.99	[0.93, 1.05]	.73	0.99	[0.93, 1.05]	.70	0.99	[0.91, 1.08]	.82
Education	1.00	[0.89, 1.13]	.96	1.00	[0.89, 1.13]	.95	0.92	[0.78, 1.09]	.33
Neighborhood disadvantage	0.98	[0.93, 1.03]	.44	0.98	[0.93, 1.03]	.45	1.00	[0.93, 1.08]	.97
Neuroticism	<b>1.70</b>	<b>[1.36, 2.12]</b>	<b>&lt;.0001</b>	<b>1.70</b>	<b>[1.36, 2.12]</b>	<b>&lt;.0001</b>	<b>1.90</b>	<b>[1.41, 2.57]</b>	<b>&lt;.0001</b>
Extraversion	<b>1.94</b>	<b>[1.43, 2.65]</b>	<b>&lt;.0001</b>	<b>1.94</b>	<b>[1.43, 2.65]</b>	<b>&lt;.0001</b>	<b>2.17</b>	<b>[1.44, 3.29]</b>	<b>.0003</b>
Openness to experience	1.04	[0.77, 1.41]	.79	1.04	[0.77, 1.41]	.79	1.10	[0.73, 1.67]	.64
Agreeableness	0.83	[0.60, 1.13]	.23	0.83	[0.60, 1.13]	.24	0.77	[0.49, 1.21]	.26
Conscientiousness	<b>0.67</b>	<b>[0.51, 0.88]</b>	<b>.004</b>	<b>0.67</b>	<b>[0.51, 0.88]</b>	<b>.004</b>	<b>0.61</b>	<b>[0.42, 0.89]</b>	<b>.01</b>
Age of first drink	<b>0.82</b>	<b>[0.77, 0.88]</b>	<b>&lt;.0001</b>	<b>0.82</b>	<b>[0.77, 0.88]</b>	<b>&lt;.0001</b>	<b>0.84</b>	<b>[0.77, 0.92]</b>	<b>.0001</b>
Age of regular drinking	1.03	[1.00, 1.07]	.07	1.03	[1.00, 1.07]	.07	1.04	[0.99, 1.09]	.15
AAB	<b>2.64</b>	<b>[1.12, 6.24]</b>	<b>.03</b>	<b>2.63</b>	<b>[1.12, 6.22]</b>	<b>.03</b>	2.89	[0.94, 8.83]	.06

Note. Reference categories: sex = female, parental status = no kids. Bold indicates significance, *p* < .05. AUD = alcohol use disorder; MZ = monozygotic; DZ = dizygotic; BP = between-pair; WP = within-pair; AAB = adult antisocial behavior.

tained (see Table 5, column c). Results did not differ across partially and fully adjusted models.

Figure 1 summarizes and compares the results of the fully adjusted models; they provide strong evidence for the relative roles of marital status and parental status in men versus women.

### Discussion

The present study examined the joint influence of two major developmental tasks known to be associated with AUD symptoms (Leonard & Eiden, 2007), as well as differences in their influence among men and women. The series of models presented here aimed to disentangle the effects of marriage and parenthood on AUD by (a) examining these factors in isolation versus simultaneously and (b) using individual-level versus genetically informed approaches. When modeled as sole predictors, both marital and parental status appeared to exert influence on AUD symptoms, and with apparent quasi-causal

influence for both among women. However, when examined as simultaneous predictors, a pattern emerged whereby familial effects of marital status predicted AUD symptoms among men, while quasi-causal effects of parenthood emerged most robustly among women.

It is argued that the marriage effect persists independent of parental status (Bachman et al., 2013; Leonard & Eiden, 2007), which may be an accurate assertion when examining the effect at the population level. The individual-level model of marital and parental statuses as simultaneous predictors presented here reflected a similar finding (see Table 5, column a), as did estimates from individual-level analyses with data from men and women modeled together (see Table S5, column a in the online supplemental materials). However, a different picture begins to emerge when applying a more rigorous test of effects in a cotwin control framework and when parsing out these effects in men and women separately. In contrast to the sole-

Table 5  
Incident Rate Ratios (IRR) for Fully Adjusted Models of Marital and Parental Status Predicting AUD Symptoms

Model variables	(a) Individual-level			(b) MZ-DZ			(c) MZ-only		
	IRR	[95% CI]	<i>p</i>	IRR	[95% CI]	<i>p</i>	IRR	[95% CI]	<i>p</i>
Men									
Predictor									
Marital status	<b>0.61</b>	<b>[0.41, 0.93]</b>	<b>.02</b>	—	—	—	—	—	—
BP marital status	—	—	—	<b>0.48</b>	<b>[0.28, 0.84]</b>	<b>.01</b>	<b>0.24</b>	<b>[0.11, 0.55]</b>	<b>.001</b>
WP marital status	—	—	—	0.77	[0.42, 1.43]	.41	0.60	[0.23, 1.52]	.28
Parental status	0.78	[0.51, 1.21]	.27	—	—	—	—	—	—
BP parental status	—	—	—	0.75	[0.42, 1.34]	.34	1.67	[0.74, 3.78]	.22
WP parental status	—	—	—	0.90	[0.48, 1.69]	.74	1.12	[0.41, 3.09]	.83
Covariate									
Zygoty	1.18	[0.84, 1.67]	.34	1.23	[0.87, 1.76]	.24	—	—	—
Age	1.01	[0.94, 1.09]	.79	1.02	[0.95, 1.11]	.57	1.00	[0.90, 1.11]	.99
Education	<b>0.76</b>	<b>[0.66, 0.88]</b>	<b>.0002</b>	<b>0.74</b>	<b>[0.64, 0.86]</b>	<b>&lt;.0001</b>	0.84	[0.68, 1.04]	.11
Neighborhood disadvantage	1.00	[0.94, 1.07]	.98	1.00	[0.94, 1.07]	1.00	1.00	[0.92, 1.09]	.99
Neuroticism	<b>1.81</b>	<b>[1.39, 2.36]</b>	<b>&lt;.0001</b>	<b>1.79</b>	<b>[1.37, 2.34]</b>	<b>&lt;.0001</b>	<b>1.67</b>	<b>[1.15, 2.43]</b>	<b>.007</b>
Extraversion	1.33	[0.97, 1.84]	.08	<b>1.44</b>	<b>[1.03, 2.00]</b>	<b>.03</b>	1.12	[0.71, 1.76]	.62
Openness to experience	0.95	[0.65, 1.40]	.81	0.92	[0.62, 1.36]	.67	0.82	[0.49, 1.38]	.46
Agreeableness	<b>0.69</b>	<b>[0.49, 0.98]</b>	<b>.04</b>	<b>0.70</b>	<b>[0.49, 0.99]</b>	<b>.05</b>	0.71	[0.44, 1.16]	.17
Conscientiousness	0.97	[0.70, 1.34]	.86	0.99	[0.72, 1.37]	.97	1.10	[0.72, 1.70]	.66
Age of first drink	<b>0.89</b>	<b>[0.82, 0.95]</b>	<b>.002</b>	<b>0.90</b>	<b>[0.83, 0.96]</b>	<b>.004</b>	0.92	[0.83, 1.02]	.12
Age of regular drinking	1.04	[0.99, 1.09]	.15	1.04	[0.98, 1.09]	.17	<b>1.08</b>	<b>[1.01, 1.16]</b>	<b>.03</b>
AAB	1.62	[0.89, 2.95]	.11	1.73	[0.95, 3.17]	.07	<b>2.81</b>	<b>[1.30, 6.08]</b>	<b>.009</b>
Women									
Predictor									
Marital status	<b>0.73</b>	<b>[0.53, 1.00]</b>	<b>.05</b>	—	—	—	—	—	—
BP marital status	—	—	—	0.83	[0.51, 1.36]	.46	0.76	[0.38, 1.53]	.44
WP marital status	—	—	—	0.66	[0.43, 1.01]	.06*	1.06	[0.58, 1.95]	.85
Parental status	<b>0.53</b>	<b>[0.38, 0.74]</b>	<b>.0002</b>	—	—	—	—	—	—
BP parental status	—	—	—	0.56	[0.34, 0.93]	.59	0.73	[0.35, 1.54]	.41
WP parental status	—	—	—	<b>0.84</b>	<b>[0.44, 1.59]</b>	<b>.03</b>	<b>0.33</b>	<b>[0.18, 0.62]</b>	<b>.0006</b>
Interaction									
WP Parental Status × Zygoty	—	—	—	0.49	[0.22, 1.09]	.08	—	—	—
Covariate									
Zygoty	0.84	[0.62, 1.14]	.27	0.84	[0.61, 1.15]	.27	—	—	—
Past year pregnancy	0.61	[0.36, 1.05]	.08	0.62	[0.36, 1.06]	.08	0.44	[0.19, 1.02]	.06
Age	0.99	[0.93, 1.05]	.72	0.98	[0.92, 1.05]	.64	0.97	[0.88, 1.07]	.55
Education	1.00	[0.88, 1.13]	.97	1.00	[0.88, 1.14]	.96	0.92	[0.77, 1.10]	.34
Neighborhood disadvantage	0.99	[0.94, 1.04]	.63	0.99	[0.93, 1.04]	.61	1.04	[0.96, 1.13]	.38
Neuroticism	<b>1.72</b>	<b>[1.37, 2.16]</b>	<b>&lt;.0001</b>	<b>1.73</b>	<b>[1.37, 2.17]</b>	<b>&lt;.0001</b>	<b>2.00</b>	<b>[1.45, 2.78]</b>	<b>&lt;.0001</b>
Extraversion	<b>1.79</b>	<b>[1.31, 2.44]</b>	<b>.0003</b>	<b>1.74</b>	<b>[1.27, 2.39]</b>	<b>.0006</b>	<b>1.99</b>	<b>[1.29, 3.08]</b>	<b>.002</b>
Openness to experience	0.98	[0.72, 1.34]	.91	0.96	[0.70, 1.32]	.82	1.02	[0.66, 1.58]	.93
Agreeableness	0.88	[0.64, 1.21]	.44	0.90	[0.65, 1.25]	.55	0.81	[0.50, 1.30]	.38
Conscientiousness	<b>0.69</b>	<b>[0.53, 0.92]</b>	<b>.01</b>	<b>0.71</b>	<b>[0.53, 0.94]</b>	<b>.02</b>	0.71	[0.47, 1.08]	.11
Age of first drink	<b>0.83</b>	<b>[0.78, 0.89]</b>	<b>&lt;.0001</b>	<b>0.83</b>	<b>[0.78, 0.89]</b>	<b>&lt;.0001</b>	<b>0.85</b>	<b>[0.77, 0.93]</b>	<b>.0006</b>
Age of regular drinking	1.03	[1.00, 1.07]	.08	1.03	[0.99, 1.06]	.11	1.03	[0.98, 1.08]	.22
AAB	2.28	[0.91, 5.72]	.08	2.37	[0.93, 6.00]	.07	2.24	[0.65, 7.70]	.20

Note. Reference categories: sex = female, marital status = never married, parental status = no kids. Bold indicates significance,  $p < .05$ . AUD = alcohol use disorder; MZ = monozygotic; DZ = dizygotic; BP = between-pair; WP = within-pair; AAB = adult antisocial behavior.

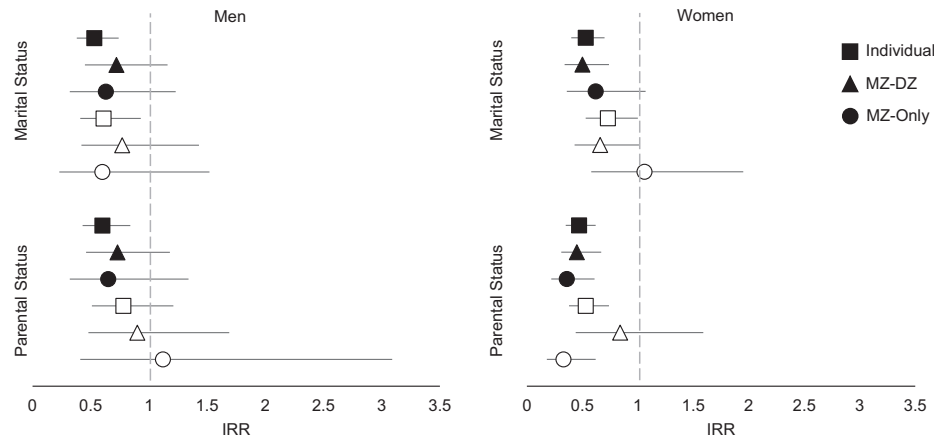
\* Effect was significant in partially-adjusted models ( $IRR = .51$ – $0.66$ ,  $p = .001$ – $.04$ ).

predictor models that approximated independent data, which showed roughly equivalent significant effects of both marital and parental status, the cotwin control and zygoty-limited models reflected that the effect of marriage was eclipsed among women when parental status was included in the model. This finding is in line with past longitudinal and twin studies that, respectively, found parental status to eclipse marital status (Power et al., 1999) and suggested that associations between women's drinking and marital status is likely not causal (Prescott & Kendler, 2001). Juxtaposition of these models highlights that examining marital or parental status in isolation,

testing only individual-level models, and/or neglecting to examine sex differences may hamper the ability to grasp a fuller picture of the potentially causal association between these roles and AUD symptoms.

In line with previous research, the present study identified that marital status, and not parenthood, drives AUD symptoms among men, albeit not causally. However, contrary to some previous research, the results presented here do not support the role of marital status in mitigated risk for AUD symptoms among women. There are two reasons why past studies may have found an effect of marital status among women: (a) parental status was not in-





*Figure 1.* Forest plot of incidence rate ratios (*IRR*) for fully adjusted models in men and women. Black markers represent effects of sole predictor models, white markers represent effects of the simultaneous predictor model. A predictor is significant if its confidence interval does not pass through the vertical line denoting an *IRR* of 1.00. Effects for cotwin control models are within-pair (quasi-causal) effects. MZ = monozygotic; DZ = dizygotic.

cluded as a concurrent predictor, and/or (b) the effects for men and women were not disaggregated. That is, an effect was attributed to marriage that may have been due to parenthood (as many married individuals are also parents) and may have appeared significant when modeling these effects in a mixed-sex sample (as was also observed in our full sample analyses; see [Table S5](#) in the online supplemental materials).

Cultural and societal norms, divergent social role expectations of men and women in midadulthood, and the contraindication of drinking with biological processes of childbearing may in part contribute to a difference in the potentially causal role of parenthood in AUD symptoms among men and women (Nolen-Hoeksema, 2004). For example, women not uncommonly take on the primary role in childcare and associated responsibilities. Carrying these responsibilities may play a causal role in minimizing heavy drinking patterns that themselves can increase risk for development or persistence of AUD (Laborde & Mair, 2012), perhaps due to having less time available to drink, be under the influence of alcohol, or to recover from a drinking episode (O'Malley, 2004). Additionally, alcohol use, and particularly problematic alcohol use, is often stigmatized and viewed as negligent in the context of child rearing, with these attitudes disproportionately directed toward women (Lyons & Willott, 2008), and consuming alcohol while pregnant and/or breastfeeding is actively targeted in public health efforts. These factors may influence mothers' decisions to refrain from consuming alcohol, while men are necessarily precluded from having to consider many of them when making decisions about alcohol use.

It has been suggested that these effects of marital status and parenthood on drinking behaviors could be due to self-selection into these roles. That is, individuals who take on these roles are also those who are less likely to experience AUD symptoms, while those who experience AUD symptoms are also those less likely to take on these roles (Leonard & Eiden, 2007; Leonard & Rothbard, 1999). Selection effects for marriage are well-documented, and are in part reflected in the between-pair effects among men found in

the present study (stronger between-pair as compared to within-pair effects indicate the presence of selection; Burt et al., 2010). However, it is unlikely that selection effects fully explain the disproportionately strong quasi-causal effects of parenthood on AUD symptoms among women in the present sample, particularly in light of the general lack of between-pair effects that would indicate selection. While selection effects may explain the association between marriage and reduction of AUD risk among men, the results presented here provide robust evidence that a potentially causal mechanism can at least partially explain the association between parenthood and reduction of risk for AUD among women.

## Limitations

This study presents with limitations. First, it is unclear how results from this Australian sample will generalize to other groups. Second, these data were cross-sectional. As such, we were unable to observe changes in AUD symptoms prior to and post role transition or conclusively determine causal ordering of AUD and marital or parental status. We also did not have data on how long participants had been married or been parents, which may play a role in these associations with AUD symptoms. Finally, our analyses may have been underpowered to detect effects in the sex-stratified models, and particularly in the sex-stratified MZ-only models, as reducing the sample by sex and zygosity substantially reduced the sample size; as such, the differences in observed effects across men and women may be, at least in part, attributable to power issues. However, the results of this study are consistent with longitudinal research demonstrating effects of parental status among women, but not men (Power et al., 1999), and twin research demonstrating that marital status is not a causal factor in reduced drinking among women (Prescott & Kendler, 2001). Despite limitations, the present study adds an important piece to the puzzle of the divergent associations of marriage, parenthood, and AUD among men and women.

## Conclusions

This is the first study to use a quasi-causal, genetically informed design to identify simultaneous marital and parental status effects on AUD symptoms in men and women. The findings presented here support the scant literature that has concurrently examined the influence of marital and parental statuses on midadulthood heavy drinking (Chilcoat & Breslau, 1996), finding that noncausal effects of marital status, rather than parenthood, are the primary drivers of symptom reduction among men while parental status exerts substantive, quasi-causal influence on AUD symptoms among women (Power et al., 1999). Initiatives to increase paternal involvement in childcare and parental responsibilities typically shouldered by mothers may serve to expand the protective effect of parenthood to fathers at risk for AUD. It will be important to continue to monitor these effects, and to do so with a closer eye to the unique effects of developmental tasks on women's health as it relates to AUD, particularly as fewer women are having children and, potentially relatedly, as women's alcohol consumption rates increase (Hamilton, Martin, Osterman, & Rossen, 2019; Richter, 2019; Slade et al., 2016).

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