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Comparing the Potential Causal Influence of Two Indicators of Early Alcohol Use on Later Alcohol Use Disorder Symptoms

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Age of first drink (AFD) has repeatedly been found to be associated with alcohol use disorder (AUD); however, some studies suggest this is a noncausal effect that may be due to childhood risk factors or familial influences. In contrast to indicators of any early alcohol use, such as AFD, indicators of a pattern of repeated drinking may be more likely to be causally associated with later problematic alcohol use. The current study examined AFD and age of onset of regular drinking (ARD; defined as drinking at least once a month for 6 or more months) as quasicausal predictors of lifetime AUD symptoms. Participants were 3,005 adult Australian twins who reported having been regular drinkers in their lifetime. Semistructured interviews were conducted to assess AFD, ARD, AUD, externalizing symptomatology, and other substance use. Personality traits were assessed via questionnaire. Unadjusted and adjusted multilevel discordant twin models were conducted using data from 1,041 complete twin pairs; adjusted models included socioeconomic status, personality, conduct disorder, and early initiation of regular smoking and marijuana use as covariates. Results from fully adjusted models controlling for familial confounds provided evidence for a causal influence of ARD on AUD symptoms, whereby twins with an earlier age of regular drinking than their cotwin had more lifetime AUD symptoms. However, AFD did not significantly predict AUD symptoms after adjusting for confounds. These results suggest that early regular drinking may serve as a causal risk factor for future problems, while early initiation of any alcohol use may indicate genetic liability.

General Scientific Summary

Early onset of regular drinking may be a causal risk factor for the development of alcohol use disorder, whereas age of first drink may instead serve as an indicator of familial risk. Interventions targeting early patterns of alcohol use, therefore, may be more effective at reducing future drinking-related harms than are those targeting initiation of any alcohol use.

Keywords: age of regular drinking, age of first drink, alcohol use disorder, multilevel model, discordant twin design

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Individuals vary considerably in their age of onset of alcohol use as well as their early patterns of use, and this variation might be important for understanding risk for developing an alcohol use disorder (AUD). Age of first drink (AFD) has been repeatedly found to be associated with AUD and other problematic alcohol use behaviors (Aiken et al., 2018; DeWit, Adlaf, Offord, & Ogborne, 2000; McGue, Iacono, Legrand, Malone, & Elkins, 2001). However, it is unclear whether this is a causal relation, with some research suggesting that early onset of drinking is not associated with risk after controlling for childhood conduct problems or taking into account familial influences (Prescott & Kendler, 1999; Rossow & Kuntsche, 2013; Sartor et al., 2009). For example, two recent studies have found minimal evidence of a causal effect of AFD on later substance use, mental health, and antisocial behavior after accounting for childhood risk factors (Newton-Howes, Cook, Martin, Foulds, & Boden, 2019; Waldron, Malone, McGue, & Iacono, 2018). Instead, the association between AFD and later negative outcomes could largely be explained by confounding

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factors (Newton-Howes et al., 2019; Waldron et al., 2018). The effects of AFD on problematic outcomes, therefore, might be noncausal, suggesting that prevention efforts should be focused elsewhere.

Researchers have indeed begun to question the value of AFD as an indicator of alcohol-related risks (Kuntsche, Rossow, Engels, & Kuntsche, 2016; Ward, Snow, & Aroni, 2010). Although AFD indicates onset of alcohol use to some degree, individuals may have just one drink of alcohol at a young age and then not have another drink for a number of years or may never proceed to a regular pattern of drinking. Instead, indicators of more involved patterns of alcohol use may be more strongly associated with later hazardous substance use than initiation of any use (Baggio, Studer, Mohler-Kuo, Daeppen, & Gmel, 2013; Woodcock, Lundahl, Stoltman, & Greenwald, 2015). For example, age of onset of intoxication is a better predictor of progression to "hard" drug use (e.g., heroin, crystal methamphetamine) than onset of any alcohol use (Baggio et al., 2013). Similarly, in a sample of over 500 heroin users the best predictor of progression to regular heroin use was age of initiation of regular alcohol and tobacco use (defined as using 3+ times per week; Woodcock et al., 2015). A potential explanation for this finding is that individuals who initiate regular alcohol use or intoxication early in adolescence or as children may disrupt critical social, psychological, and neurological developments that occur during this period (DeWit et al., 2000; Zeigler et al., 2005). These effects are likely to be a function of cumulative exposure, with greater use resulting in increased impairment compared to more limited or sporadic alcohol consumption. Although these studies did not assess alcohol-specific outcomes, they provided evidence that early, involved alcohol use may predict a variety of substance use outcomes better than early initiation.

Previous studies have used discordant twin analysis to examine the effects of early regular drinking on later alcohol use outcomes. These analyses control for unmeasured genetic and shared environmental confounders by examining twins who differ from one another on an environmental exposure of interest (e.g., initiation of regular drinking). If the twin with an earlier age of initiation develops more severe alcohol problems than their cotwin (with the same genes and rearing environment) who initiated regular alcohol use at a later age, this would be consistent with a causal effect of regular drinking initiation on later alcohol problems. Using data from a sample of male Vietnam veteran twins, researchers conducted regression analyses of twin pairs discordant for early initiation of regular drinking (defined as drinking at least once a month for 6+ months) to determine whether this predicted adult substance use outcomes, including alcohol use and dependence, after accounting for genetic and shared environmental influences (Grant et al., 2006). There was evidence for a quasicausal effect of early regular alcohol use on adult alcohol dependence, as well as marijuana and other drug abuse/dependence (Grant et al., 2006). This line of research provides evidence for the importance of drinking patterns in adolescence on later problematic alcohol and other substance use.

Another study used multilevel Cox proportional hazard frailty models to examine the developmental trajectory of stages of alcohol use (i.e., from less problematic to more problematic drinking) to determine causal effects of age of initiation of early drinking milestones on time to the next drinking milestone in a sample of adult Australian twins[1] (Deutsch et al., 2017). Each drinking milestone was examined as a causal predictor of the time to the next milestone in a cascade-like process (e.g., AFD predicting age of first intoxication, which then predicts age of onset of regular drinking [ARD], which then predicts onset of tolerance, and so forth). The findings suggested that earlier drinking milestones (e.g., AFD) had potentially causal effects on more proximal milestones (e.g., ARD), which, in turn, had potentially causal effects on more distal drinking milestones (e.g., age of first AUD symptom onset; Deutsch et al., 2017). This study highlights the importance of intermediate drinking milestones (like ARD) on AUD development.

Discordant twin designs used in previous studies control for genetic and shared environmental influences but cannot control for unique environmental influences that may affect early alcohol use and lifetime AUD symptom counts. A variety of other factors (socioeconomic status [SES], personality, conduct disorder, early initiation of regular smoking, and early repeated marijuana use) have been shown to be associated with AUD and have the potential to influence the age at which an individual initiates alcohol use. For example, research suggests individuals with lower SES may be more likely to experience alcohol-related problems, even after controlling for consumption (Grittner, Kuntsche, Graham, & Bloomfield, 2012; van Oers, Bongers, van de Goor, & Garretsen, 1999). Personality may be associated with alcohol use through a variety of mechanisms, including selection effects (Park, Sher, Wood, & Krull, 2009), whereby individuals who are high in extraversion or Openness may self-select into early social groups that include heavy drinking environments. This may influence the age at which an individual initiates use and may also have consequences for the development of risky drinking behaviors. Similarly, conduct disorder is a powerful predictor of both substance use disorders and early initiation of substance use (Elkins, McGue, & Iacono, 2007). Finally, according to the stepping-stone hypothesis (Cohen, 1972), which suggests that substance use initiation is associated with an increased probability of the use of another substance, early tobacco and marijuana use may increase liability for early initiation of other substances, like alcohol, and may be associated with later problem use (Brook, Brook, Zhang, Cohen, & Whiteman, 2002; Chen et al., 2002). However, previous studies implicating a causal influence of early drinking have not always accounted for these factors, which may help explain the association of early initiation of alcohol use and later problems (Kuntsche et al., 2016).

The current study aimed to expand upon previous research by examining potential causal relations between AFD, ARD, and lifetime AUD symptom counts using a multilevel discordant twin design. While causal influences of AFD and ARD on AUD have been examined separately within discordant twin analyses, these effects have not yet been directly compared within the same study. By examining both AFD and ARD as quasicausal predictors of lifetime alcohol problems, the current study was able to make comparisons of the effects of these alcohol use onset phenotypes on the development of disordered drinking. Additionally, the current study included a number of covariates (SES, personality, conduct disorder, early initiation of regular smoking, and early repeated marijuana use) that allowed for a more rigorous exami-

[1] The sample in Deutsch et al. (2017) partially overlaps with the present study sample.

nation of causality, with the aim of avoiding improper causal inferences (McGue, Osler, & Christensen, 2010).

Method

Participants

Participants were recruited from the Australian Twin Registry (ATR), which is a large population-based volunteer registry of twins and siblings (Hopper, 2002), to participate in an interview-based study of substance use and mental health. Data for the current study were collected via computer-assisted telephone interviews conducted between 2005 and 2009 (76% response rate). Participants were also mailed a questionnaire following completion of the interview to assess personality traits; 93% of those interviewed completed the questionnaires, usually within 2 weeks. The sample included 3,292 twins of known zygosity who completed the interview; those who did not report being regular drinkers (e.g., drinking at least once a month for 6 months or more) at some point in their lifetime were not included (n = 287). The resulting sample consisted of 3,005 individual twins. Multilevel models were conducted using data from complete twin pairs with complete data (n = 1,041 pairs).

Potential sampling bias was examined by comparing mean AFD, ARD, and AUD symptom counts among twins from pairs concordant for participation in the survey ("complete pairs") to means among twins whose cotwin did not participate in the survey ("incomplete pairs"). Incomplete pairs provide a window into characteristics of nonparticipating twins (including those in which neither twin from a pair participated). That is, if twins with earlier ages of AFD and ARD and more AUD symptoms were systematically undersampled, lower means for AFD and ARD and higher AUD symptoms would be expected among twins whose cotwin did not participate than among twins concordant for participation in the interview (assuming that AFD, ARD, and AUD are correlated in twin pairs). Twins from incomplete twin pairs differed slightly from those from complete pairs in their reported AFD, ARD, and AUD symptoms. On average, twins from incomplete pairs reported earlier AFD (15.53 vs. 15.94 years old), ARD (18.03 vs. 18.28 years old), and more AUD symptoms (1.88 vs. 1.56 symptoms) than twins from complete pairs. Though significant, each of these differences represented a small effect size (Cohen's ds = 0.09–0.15) and suggests minimal sampling bias.

Of complete pairs, 330 were monozygotic (MZ) females, 153 were MZ males, 252 were dizygotic (DZ) females, 107 were DZ males, and 199 were DZ opposite sex twins. Participants were between the ages of 27 and 37 at the time of data collection, with a mean age of 31.84 years (SD = 2.45). 63.9% of the sample identified as female. For additional details regarding sample recruitment and characteristics, see Lynskey et al. (2012).

Measures

The interview was based on the Australian version of the Semi-Structured Assessment of the Genetics of Alcoholism (Bucholz et al., 1994) and included assessments of drinking behaviors, alcohol-related consequences, conduct disorder symptoms, use of other substances, and demographics. Participants also completed a self-report questionnaire assessing personality traits.

AFD. To assess initiation of drinking, respondents were asked, "How old were you the first time you had a full drink of beer, wine or spirits?" In order to reduce the potential influence of outlying values, individuals who reported ages of less than 5 years were equated to 5 years. The 4-year test–retest reliability of AFD in a similar cohort from the ATR was excellent, $r(215) = .77$, $p < .0001$ (Slutske, 2019).

Age of initiation of regular drinking. To assess age of initiation of regular drinking, respondents were asked, "At what age did you start to drink regularly—that is, drinking at least once a month for 6 months or more?" This question was used to restrict the sample for the current study to regular drinkers. Participants reported initiating regular drinking, on average, at 18.21 years old (SD = 2.67). The 4-year test–retest reliability of ARD in a similar cohort from the ATR was also excellent, $r(200) = .80$, $p < .0001$ (Slutske, 2019).

Lifetime AUD symptoms. AUD symptom counts were based on participants' responses to questions pertaining to the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev.; *DSM–IV–TR*; American Psychiatric Association [APA], 2000) diagnostic criteria for alcohol abuse and dependence. Participants' lifetime symptoms were summed across abuse and dependence criteria. The resulting lifetime AUD symptom count score demonstrated high internal consistency reliability (α = .76). On average, participants who endorsed AUD symptoms reported experiencing the most symptoms when they were about 25 years old (M = 24.87, SD = 4.59; range = 9–36).

Household income. Participants were asked, "What is your current combined household gross income, that is before tax?" The 11 response options ranged from *AU$0–9,999/year* to *AU$150,000 or more/year*. Participants reported a median household income of AU$75,000–AU$99,999/year. This is representative of the general Australian population, with the Australian Bureau of Statistics (2017) reporting a mean household income of AU$89,076/year around the time of data collection.

Educational attainment. Participants were asked, "What is the highest educational level you have completed?" Response options ranged from not completing primary school to obtaining a postgraduate degree. Responses were coded into a five-level variable, where scores of 1 indicated that a participant did not complete Year 12 (equivalent to not completing high school in the United States; 10.38% of sample), 2 indicated completion of Year 12 (high school diploma; 13.88% of sample), 3 indicated completion of technical college (similar to community college; 28.22% of sample), 4 indicated obtaining an undergraduate degree (28.02% of sample), and 5 indicated obtaining postgraduate education (19.50% of sample).

Conduct disorder symptomatology. The interview assessed each of the 15 *DSM–IV–TR* (APA, 2000) criteria for conduct disorder. Criteria for conduct disorder include symptoms within four broad domains: (a) aggression to people and animals, (b) destruction of property, (c) deceitfulness or theft, and (d) serious violations of rules. Participants were asked to only consider those behaviors that occurred prior to age 18. Although the *DSM–IV–TR* (APA, 2000) requires that the two conduct disorder criteria "often stays out at night" and "often truant from school" occur before age 13, this age requirement was not imposed in the current study. Criteria were summed across the four domains, with the resulting

conduct disorder symptom count score having acceptable internal consistency reliability ($\alpha = .67$).

Early regular smoking. In line with the study's hypotheses regarding the importance of an established pattern of substance use, regular smoking was used in analyses rather than initiation of smoking. To assess early regular smoking, participants were first asked, "Was there ever a time in your life when you smoked cigarettes at least once a week for at least two months in a row?" Participants who endorsed having smoked regularly during their lifetime were asked the age at which they first began smoking regularly. Age of initiation of regular smoking ranged from 7–33 years in the current sample. Those who reported being regular smokers by age 14 (approximately 1 *SD* below the sample mean age of initiation) were coded as 1, while those who were not were coded as 0.

Early repeated marijuana use. The marijuana use assessment differed from that of smoking, and age of onset of regular marijuana use was not assessed. However, it was possible to characterize early repeated marijuana use. Participants were first categorized according to whether they had tried marijuana by age 15 (which is approximately 1 *SD* below the sample mean age of initiation). Following this, participants who reported using marijuana in their lifetime were asked, "How soon after you first tried marijuana did you try it again?" Again, in order to capture a repeated pattern of use beyond mere initiation (in line with the study's hypotheses regarding the importance of an established pattern of substance use), participants who reported never using marijuana and those who only used marijuana once and never tried it again (regardless of their age of initiation) were coded as 0. Those who reported first using marijuana by age 15 and who continued to use were coded as 1. Therefore, this measure indexes early repeated marijuana use.

Personality traits. Participants who completed the interview were subsequently invited to complete a questionnaire that included a 74-item adapted Revised NEO Personality Inventory (Costa & McCrae, 1992; Few et al., 2014) assessing the Big Five personality traits of Neuroticism, Extraversion, Openness to Experience, Agreeableness, and Conscientiousness. Responses were coded on a 1 (*strongly disagree*) to 5 (*strongly agree*) scale. Scale scores were the average of all responses for a particular personality trait and also ranged from 1 to 5. Each scale demonstrated high internal consistency reliability, with coefficient alphas for the scales ranging from 0.77–0.89.

Analytic Plan

All analyses were conducted using SAS (Version 9.4). Descriptive analyses were conducted to examine sample characteristics. Correlations were calculated accounting for clustering of twin pairs. To examine the effect of AFD and ARD on lifetime 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). Both Level 1 and Level 2 variances were estimated, along with a random intercept. A negative binomial distribution and log link function were used due to the skewness of the lifetime AUD symptom count variable, with about a third of respondents (34.43%) reporting zero lifetime symptoms. Coefficients from the multilevel models were exponentiated to produce incidence rate ratios (IRRs), which index relative risk. For each unit increase in the predictor variable, IRRs represent the percent change in the predicted rate of AUD symptoms (e.g., an IRR of 1.40 would indicate a 40% increase in AUD symptoms with each unit increase in the predictor).

Models were initially run at the individual level taking into account nonindependence of twin-pair observations. A base model was fit including sex, zygosity, AFD, and ARD as predictors of lifetime AUD symptoms. Evidence for sex differences was evaluated by including an AFD × Sex interaction term and an ARD × Sex interaction term; if significant, interaction terms were carried forward into subsequent analyses. Fully adjusted models were fit including all covariates (income, education, personality traits, conduct disorder symptoms, early regular smoking, and early repeated marijuana use). These individual level analyses examine evidence for an overall effect of early drinking and approximate analyses conducted using unrelated individuals.

Following this, cotwin control models were fit in an attempt to identify potential sources of confounding that might contribute to the overall effect (McGue et al., 2010). Models included a random intercept to account for the nonindependence of twin data (β_{0i}). The cotwin control design models environmental variables of interest (i.e., AFD and ARD) in terms of a within-twin pair regression coefficient (β_W) and a between-twin pair regression coefficient (β_B):

$$Y_{ij} = \beta_0 + \beta_{W1}(X_{1ij} - \overline{X}_{1i}) + \beta_{W2}(X_{2ij} - \overline{X}_{2i}) + \beta_{B1}\overline{X}_{1i} + \beta_{B2}\overline{X}_{2i} + \beta_{0i} + e_{ij}$$

where Y_{ij} is the number of lifetime AUD symptoms for individual j within the ith twin pair, X_{1ij} is the AFD for individual j within the ith twin pair, X_{2ij} is the ARD for individual j within the ith twin pair, $\overline{X}_{1i}$ is the mean AFD for the ith twin pair, and $\overline{X}_{2i}$ is the mean ARD for the ith twin pair.

Compared to analysis of unrelated individuals, the discordant twin analysis controls for familial environmental and genetic factors (partially for DZ twins and completely for MZ twins), permitting stronger causal inference. If between-twin pair effects of a predictor are significant, this suggests that genetic or shared environmental factors associated with the predictor contribute to lifetime AUD symptom development. On the contrary, if within-twin pair effects are significant, this suggests that the associated environmental exposure (such as AFD or ARD) may causally contribute to AUD symptom development. Base and fully adjusted models were fit including both MZ and DZ twin pairs. To examine potential sources of confounding, zygosity by within-twin pair interactions were included. Significant zygosity by within-twin pair interactions would indicate potential genetic confounding. Any significant interactions were carried forward into fully adjusted models. Zygosity-limited analyses including only DZ twins and only MZ twins were also conducted. Models including only MZ twins allow for the strongest causal interpretation, whereby a predictor may be isolated as having a unique environmental effect on lifetime AUD symptomatology.

The relative causal influence of AFD and ARD on lifetime AUD symptoms was examined by conducting log-likelihood ratio tests comparing model fit for a fully adjusted cotwin control model

including quasicausal effects of both AFD and ARD to nested models including quasicausal effects for just one early drinking indicator (either AFD or ARD). In addition, the fits of sole predictor models (i.e., that included quasicausal effects of either AFD or ARD) were compared using the Bayesian information criterion (BIC), which is a criterion for evaluating model fit that favors parsimonious solutions and has been shown to perform consistently, particularly with large sample sizes (Dziak, Coffman, Lanza, Li, & Jermiin, 2019).

Results

Sample Characteristics

Participants typically had their first drink around age 16 (M = 15.70, SD = 2.22), and 15.33% of twins were concordant for AFD. Discordant twins differed in their AFD by 2.29 years, on average (SD = 2.01, range = 1–15 years). Participants typically became regular drinkers around age 18 (M = 18.21, SD = 2.67), and 18.08% of twins were concordant for ARD. Twins who were discordant for ARD differed in their ages of initiation by 2.68 years, on average (SD = 2.43; range = 1–16 years).

Participants reported being, on average, 14.29 years old when they first used tobacco (SD = 3.55) and were 17.85 years old (SD = 3.33) when they first used marijuana. About half (52.46%) of the sample reported smoking tobacco by age 14. However, just 14.71% of these users progressed to regular smoking and were classified as early regular smokers for the current study. One-fifth (20.94%) of the sample tried marijuana by age 15, and of these early users, 96.29% were classified as early repeated marijuana users.

Approximately two thirds (65.57%) of the sample reported at least one AUD symptom in their lifetime, and just under half (42.54%) met criteria for lifetime AUD based on endorsing two or more AUD symptoms. On average, participants reported 1.76 lifetime AUD symptoms (SD = 2.02). Additionally, almost half (45.49%) of participants endorsed at least one conduct disorder symptom, and 9.61% endorsed three or more conduct disorder symptoms, the number required for a lifetime diagnosis.

Correlations Between Study Variables

AFD was negatively correlated with AUD symptoms, $r = -0.25$, $p < .001$, such that those with an earlier AFD had significantly more lifetime AUD symptoms. ARD and lifetime AUD symptoms were also significantly negatively correlated, $r = -0.24$, $p < .001$. Lower income and educational attainment were associated with endorsing more lifetime AUD symptoms (Table 1). Higher levels of Neuroticism were associated with more AUD symptoms, as were lower levels of Agreeableness, Extraversion, and Conscientiousness. Conduct disorder symptoms, early regular smoking, and early repeated marijuana use were all associated with increased AUD symptoms.

Many covariates that were associated with AUD, such as the personality traits of Agreeableness, Extraversion, and Conscientiousness, conduct disorder symptoms, early regular smoking, and early repeated marijuana use were also correlated with AFD and ARD (see Table 1). Individuals higher in Extraversion and Openness and lower in Agreeableness and Conscientiousness were more likely to have earlier ages of alcohol use, as were individuals who reported early regular smoking, early repeated marijuana use, and more conduct disorder symptoms.

Multilevel Models

Individual level models. The base GLMM run at the individual level indicated that AFD significantly predicted lifetime AUD symptoms (IRR = 0.94, 95% CI [0.91, 0.97]), with each additional age at which drinking initiation was postponed contributing to an expected 6% decrease in AUD symptom counts. Similarly, ARD significantly predicted AUD symptoms (IRR = 0.90, 95% CI [0.88, 0.93]), with each additional year that regular drinking was postponed being associated with a 10% decrease in expected AUD symptoms. Sex was a significant predictor of AUD symptoms in this base individual level model, as well as all additional models,

Table 1
Correlation Matrix and Means of Study Variables

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	*M* (*SD*) or % (*N*)
1. Age															31.86 (2.48)
2. Sex	−.01														64% (1,919)
3. Age of first drink	.04	**.16**													15.70 (2.22)
4. Age of regular drinking	**.05**	**.10**	**.52**												18.21 (2.68)
5. AUD symptoms	−.02	**−.27**	**−.25**	**−.24**											1.76 (2.02)
6. Household income	.03	**−.07**	−.03	**−.05**	**−.08**										8.73 (2.02)
7. Educational attainment	.02	**.08**	.02	.01	**−.16**	**.25**									3.32 (1.23)
8. Neuroticism	**−.06**	**.12**	.02	.02	**.22**	**−.22**	**−.10**								2.60 (.73)
9. Extraversion	.01	.02	**−.05**	**−.06**	**−.07**	**.21**	**.13**	**−.53**							3.54 (.55)
10. Agreeableness	**.05**	**.24**	**.09**	**.08**	**−.28**	.03	**.12**	**−.34**	**.28**						3.75 (.47)
11. Openness	**−.04**	**.04**	**−.09**	**−.06**	.03	.01	**.26**	**.06**	**.13**	**.07**					3.31 (.47)
12. Conscientiousness	**.08**	**.13**	**.08**	**.04**	**−.22**	**.16**	**.16**	**−.42**	**.31**	**.26**	**−.05**				3.84 (.54)
13. Early regular smoking	−.01	**−.05**	**−.20**	**−.18**	**.24**	**−.12**	**−.28**	**.11**	**−.08**	**−.13**	−.02	**−.12**			7% (208)
14. Early marijuana use	**−.08**	**−.12**	**−.35**	**−.32**	**.27**	−.03	**−.14**	**.06**	**−.06**	**−.14**	**.11**	**−.14**	**.40**		15% (459)
15. CD symptoms	−.03	**−.22**	**−.30**	**−.21**	**.42**	**−.10**	**−.25**	**.11**	**−.08**	**−.28**	.03	**−.18**	**.35**	**.32**	.88 (1.35)

Note. Table entries are Pearson correlations (phi coefficients for dichotomous variables—sex, early regular smoking, and early marijuana use). Sample sizes for calculating correlations ranged from 2,793–3,005 twins, depending on data missingness. For the variable of sex, females were coded as 1, males were coded as 0. Bold indicates significance ($p < .05$). AUD = alcohol use disorder; CD = conduct disorder.

with men having higher lifetime AUD symptomatology. However, there was no evidence for significant sex differences in the effects of AFD or ARD, as indicated by nonsignificant interaction terms (AFD: IRR = 1.04, 95% CI [0.99, 1.08]; ARD: IRR = 1.00, 95% CI [0.96, 1.04]).

After adjusting for covariates, the protective effects of postponing alcohol initiation and regular drinking remained (Figure 1). Postponing alcohol initiation was associated with an expected 3% reduction in lifetime AUD symptoms (IRR = 0.97, 95% CI [0.95, 0.99]), while postponing regular drinking was associated with an expected 6% reduction in symptoms (IRR = 0.94, 95% CI [0.92, 0.96]). Additionally, all personality traits except Openness predicted AUD symptoms in the fully adjusted individual-level model. Conduct disorder symptoms (IRR = 1.11, 95% CI [1.08, 1.14]), early regular smoking (IRR = 1.12, 95% CI [1.02, 1.22]), and early repeated marijuana use (IRR = 1.26, 95% CI [1.15, 1.37]) were also associated with AUD symptoms.

Cotwin control models. Cotwin control models were then fit to the data including both MZ and DZ twins (Table 2). In the base cotwin control model, the quasicausal influence of AFD was not significant (IRR = 1.00, 95% CI [0.95, 1.06]). By contrast, the within-twin pair effect indexing the quasicausal influence of ARD on AUD symptoms was significant (IRR = 0.92, 95% CI [0.88, 0.96]). The between-twin pair effect of AFD, which indexes familial influences, was significant (IRR = 0.91, 95% CI [0.86, 0.95]), as was the between-twin pair effect of ARD (IRR = 0.90, 95% CI [0.85, 0.94]). Within-twin pair influences of AFD and ARD did not significantly differ by zygosity (AFD: IRR = 0.94, 95% CI [0.88, 1.01]; ARD: IRR = 1.04, 95% CI [0.97, 1.11]), indicating no evidence of genetic confounding; therefore, these interaction terms were not carried forward into the fully adjusted cotwin control analysis.

After including covariates in the cotwin control model, the within-twin pair effect of AFD was not a significant predictor (IRR = 0.98, 95% CI [0.94, 1.01]) of AUD symptoms. However, the significant quasicausal effect of ARD on AUD symptoms remained (IRR = 0.95, 95% CI [0.92, 0.98]), with each year that regular drinking was postponed being associated with a predicted 5% decrease in AUD symptoms. Familial level influences of both AFD (IRR = 0.94, 95% CI [0.91, 0.97]) and ARD (IRR = 0.91, 95% CI [0.89, 0.94]) were significant. Similar to the individual level model, all personality traits except Openness were significantly associated with AUD symptoms. Conduct disorder symptoms (IRR = 1.15, 95% CI [1.10, 1.19]) were also associated with increased lifetime AUD symptoms. Because the within-twin pair influences of AFD and ARD did not significantly differ by zygosity, the results of zygosity-limited analyses including only DZ twins and only MZ twins are not presented here; in brief, the results from the MZ-limited analyses were similar to those presented for the full twin sample (see the online supplemental materials for more detail).

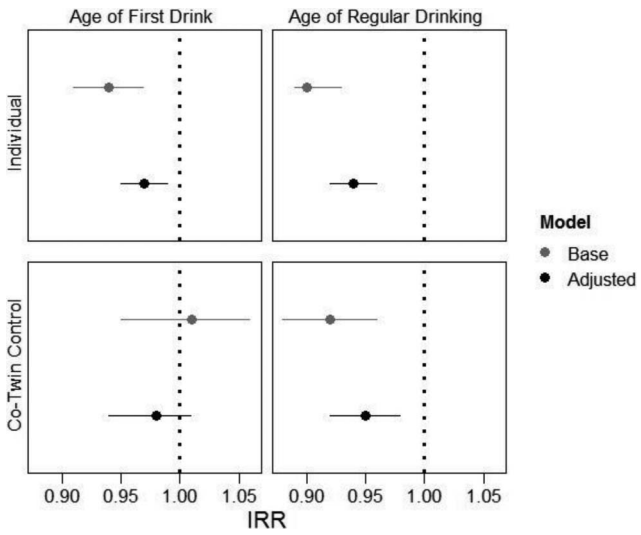

Figure 1. Forest plot of incidence rate ratios (IRRs) for lifetime alcohol use disorder symptoms predicted by age of first drink and age of regular drinking initiation. A predictor is significant if the confidence interval does not pass through the vertical dotted line indicating an IRR of 1.00. Adjusted models refer to models including all covariates.

Table 2
Results From the Fully Adjusted Cotwin Control Model Predicting Alcohol Use Disorder Symptoms

Predictor	IRR [95% CI]
Sex	**1.51 [1.36, 1.67]**
Zygosity	1.03 [.93, 1.14]
WP age of first drink	.98 [.94, 1.01]
BP age of first drink	**.94 [.91, .97]**
WP age of onset of regular drinking	**.95 [.92, .98]**
BP age of onset of regular drinking	**.91 [.89, .94]**
Household income	.99 [.97, 1.02]
Educational attainment	.97 [.93, 1.01]
Neuroticism	**1.35 [1.25, 1.45]**
Agreeableness	**.82 [.74, .91]**
Extraversion	**1.23 [1.11, 1.35]**
Conscientiousness	**.88 [.81, .97]**
Openness	.96 [.87, 1.07]
Conduct disorder symptoms	**1.15 [1.10, 1.19]**
Early regular smoking	.90 [.76, 1.07]
Early repeated marijuana use	.96 [.84, 1.10]

Note. Bold indicates significance. IRR = incidence rate ratio; CI = confidence interval; WP = within-twin pair (i.e., quasicausal effect); BP = between-twin pair (i.e., familial effect).

Model fit for the fully adjusted cotwin control model was compared to that of nested models including quasicausal effects of either AFD or ARD using log-likelihood ratio tests. Dropping the quasicausal effect of AFD resulted in significantly poorer model fit ($\Delta\chi^2(1) = 5.04$, $p = .02$). Similarly, dropping ARD from the model resulted in significant deterioration in model fit ($\Delta\chi^2(1) = 8.71$, $p = .003$), suggesting that a model including causal effects for both predictors performed better than a model including either alone. Finally, comparing the two sole predictor models against each other, the BIC indicated that a model including just ARD as a quasicausal predictor (BIC = 6,494.11) performed better than a model including only quasicausal effects of AFD (BIC = 6,458.8).

Discussion

This study compared the effects of AFD and ARD on lifetime AUD symptom counts using a genetically informed research design. The cotwin control design can be used to determine whether an effect is truly environmental and even potentially causal. The

results of the current study suggest that ARD may have an important causal effect on lifetime AUD symptoms, with the twin who began to regularly drink earlier than their cotwin going on to experience more symptoms of AUD. These effects persisted even after controlling for the effects of SES, personality, conduct disorder symptomatology, and early regular tobacco and marijuana use.

Developing a pattern of alcohol use in adolescence, which is a time associated with a number of developmental and social milestones, may be indicative of poor coping skills or stress management. For example, research suggests those who begin substance use during this stressful life period are more likely to continue using alcohol frequently as a way to cope with problems (Buchmann et al., 2010; McCubbin, Needle, & Wilson, 1985). Drinking to cope has been found to be associated with alcohol problems (Carpenter & Hasin, 1999; Holahan, Moos, Holahan, Cronkite, & Randall, 2001), and adolescents who begin drinking during this period may be more likely to develop an association between drinking and removal of negative affect than those who begin in later years. This is in line with the social learning theory of alcohol use, which suggests that those who develop alcohol problems differ from healthy drinkers in their ability to cope with stressful life events and their expectancies of alcohol use (Abrams & Niaura, 1987). If this is the case, drinking to cope may partially or completely explain the effect of ARD on lifetime AUD symptoms. A formal mediation analysis should support an indirect effect of within-twin pair ARD on AUD via drinking to cope. Because the current study did not assess drinking motives, this remains an important direction for future research.

An additional possibility is that of dose-dependent effects of alcohol on the brain, which may explain why early regular use is potentially more hazardous for lifetime AUD development compared to initiation or experimental use of alcohol. Research using animal models suggests there are dose- and age-dependent effects of alcohol on brain functioning, such that repeated or binge exposure to alcohol during adolescence is associated with greater susceptibility to alcohol's memory-impairing effects (White, Ghia, Levin, & Swartzwelder, 2000). Research with humans has supported these conclusions; accelerated declines in frontal and temporal cortical volumes were found among adolescent heavy drinkers compared to light/nondrinkers (Squeglia et al., 2015). These impairments may be critical to risk for developing AUD, as learning and memory play important roles in the development of addiction (Torregrossa & Taylor, 2016). Therefore, indices of a pattern of regular drinking during adolescence may have greater implications than initiation or experimentation for understanding adolescents' risk for experiencing harmful cognitive effects of alcohol, including impairments in memory formation and unhealthy habit development.

The current study also found evidence for a familial, or between-twin pair, effect of regular drinking initiation on later AUD symptoms, suggesting that genetic or shared environmental factors confer risk for early regular drinking initiation and AUD symptom development. This is consistent with work finding overlapping genetic influences on onset of drinking and AUD (Grant et al., 2006; Ystrom, Kendler, & Reichborn-Kjennerud, 2014) and suggests that the relationship between onset of regular drinking and later alcohol use problems is complex and cannot be explained solely by a causal effect.

Unlike the results for ARD, within-twin pair effects of AFD did not remain significant in the cotwin control analyses, which account for genetic and shared environmental influences. Although the associations of AFD and ARD with AUD symptoms were of similar magnitude, the current analyses suggested that the two indicators might provide different sources of meaningful information about an individual's risk for AUD development. AFD may operate as an indicator of familial influences, signifying genetic liability for AUD. Research findings have shown that the overlap between AFD and AUD is due largely to genetic factors, with almost no evidence for overlap of nonshared environmental influences, indicating the absence of a causal mechanism (Sartor et al., 2009; Ystrom et al., 2014). By contrast, in addition to indicating genetic risk, ARD may serve as a causal environmental risk factor, potentially by producing neurocognitive impairments and habit development that confers risk for subsequent alcohol use problems. Both of these early drinking indicators are important and likely work together to influence adult outcomes; however, being able to disentangle the nature of risk factors for AUD will aid in the development of more effective preventive and treatment interventions (Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001). For example, if ARD (and not AFD) acts as a causal environmental risk factor for AUD, this would provide more opportunities for intervention, as there are more points at which a pattern of drinking can be interrupted than a single drinking event (such as one's first drink). Parents, teachers, or mental health professionals who become aware that a child or adolescent has initiated early alcohol use may be able to intervene to halt the development of a pattern of drinking among those who may be most vulnerable to developing alcohol use problems.

Finally, given the similarity of effect sizes for AFD and ARD in the current study, additional research will be needed to confirm these findings. Disentangling the nature of risk factors for AUD and other forms of psychopathology is a difficult but much needed avenue of research. AUD is a highly complex disorder and no doubt results from a number of potential causal chains with both familial and environmental mechanisms operating to influence an individual's outcomes. However, by investigating the nature of risk factors, it may be possible to build more accurate models and better understand the development and course of AUD among individuals with heterogeneous causal pathways to the disorder.

Limitations

The current study presents with a number of limitations. Participants' reports of AFD and ARD were retrospective, and, therefore, subject to bias. Research suggests that reported ages of substance initiation tend to increase over time, especially for individuals with the earliest ages of onset (Parra, O'Neill, & Sher, 2003). Therefore, prospective reports would have been preferable. However, research has also found that age at assessment does not moderate the relationship between age of substance initiation and later problems, suggesting that this relationship may be robust to small inconsistencies in reporting (Parra et al., 2003). Additionally, in the current study, AFD and ARD were minimally associated with age at the time of assessment (rs = 0.04 and 0.05, respectively), which indicates little evidence for age-related bias in reporting ages of initiation. Furthermore, test–retest reliability in a similar cohort from the ATR was excellent (Slutske, 2019). De-

spite this, any measurement error in AFD and ARD may attenuate within-twin pair estimates due to compounding of error when using difference scores (Ashenfelter & Krueger, 1994). Additionally, as MZ twin correlations are typically higher than DZ twin correlations, any within-twin pair attenuation would be larger among MZ twin pairs (McGue et al., 2010).

A second limitation is the cross-sectional nature of the study, which did not allow for the establishment of temporal precedence for all covariates included in analyses. However, these covariates were included to provide a more rigorous test of the quasicausal effect of early drinking on AUD symptoms by accounting for a variety of potential third variables associated with AUD that may have also played a role in discordance for onset of drinking and onset of regular drinking.

Another limitation is related to inconsistencies in the literature in how "regular drinking" is defined. For the current project, regular drinking was defined as drinking at least once a month for 6 or more months. Though widely used in previous research (Dick et al., 2006; Grant et al., 2006; Jackson, 2010; Johnson & Gerstein, 1998), other definitions of regular drinking that have been used in the literature, including drinking at least once a week (Reifman, Barnes, Dintcheff, Farrell, & Uhteg, 1998), would have likely resulted in reports of different ages of initiation for some respondents. Another limitation is the use of an Australian, mostly White, sample. It is unclear whether the findings of the current study would generalize to those of other nationalities or ethnicities. For example, research shows that Australia is a particularly heavy drinking country, ranking in the top 20 countries for alcohol consumption (Organization for Economic Cooperation and Development, 2018).

Although regular drinking was quasi-causally associated with lifetime AUD symptoms, the current study did not assess contextual factors related to adolescents' drinking. Importantly, drinking safely with adults has been found to reduce risk of problematic drinking (Bellis et al., 2007; Foley, Altman, Durant, & Wolfson, 2004; Strunin et al., 2010). Research with adolescents and young adults in Italy, the United States, and England indicates that having early alcohol experiences in the context of family dinners may be protective against later alcohol use problems, including binge drinking (Bellis et al., 2007; Foley et al., 2004; Strunin et al., 2010). This suggests that early regular drinking might not always increase risk for AUD. Future research should incorporate contextual information beyond whether or not regular drinking is occurring to better understand risk for AUD.

Research has also suggested that cultural norms influence the alcohol socialization process, and these cultural factors are important for understanding risk associated with early drinking (Rolando, Beccaria, Tigerstedt, & Törrönen, 2012). Contextual factors associated with early drinking may differ across cultural identities, including nationality and gender. This relationship should be considered in a number of different populations, as cultural differences in the experience and meaning of drinking among adolescents from various backgrounds may influence the generalizability of these findings to other groups.

Conclusions

Despite the limitations of the current study, it represents a rigorous attempt to detect causal influences of ARD and AFD on lifetime AUD symptoms. Results suggested that the two early drinking indicators may provide unique information about an individual's risk for subsequent alcohol use problems, with AFD being an indicator of genetic liability, and ARD serving as both an indicator of genetic liability and as a causal environmental risk factor. An important implication of the finding that age of regular drinking onset may have a causal environmental influence on AUD symptom development is that there may be more opportunities for intervening to disrupt this pattern of drinking than there would be to intervene for a single drinking event (like one's first drink). As both AFD and ARD appear important to understanding an individual's risk for AUD, future research should continue to consider the ways in which early drinking operates to confer risk for the development of alcohol use problems.

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Correction to Gruber and Joormann (2020)

In the article "Best Research Practices in Clinical Science: Reflections on the Status Quo and Charting a Path Forward," by June Gruber and Jutta Joormann (*Journal of Abnormal Psychology*, 2020, Vol. 129, No. 1, pp. 1–4, http://dx.doi.org/10.1037/abn0000497), an incomplete sentence in the abstract read "This special section aims to take stock of current practices in our field and to reflect on them by providing user-friendly articles on common practices across a variety of methodologies in." The complete sentence is as follows: "This special section aims to take stock of current practices in our field and to reflect on them by providing user-friendly articles on common practices across a variety of methodologies in clinical science." The online version of this article has been corrected.

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