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The overall effect of parental supply of alcohol across adolescence on alcohol-related harms in early adulthood – a prospective cohort study

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Abstract

Background and Aims Recent research suggests parental supply of alcohol is associated with more risky drinking and alcohol-related harm among adolescents. However, the overall effect of parental supply across adolescence remains unclear because parental supply of alcohol varies over adolescence. Due to the complexity of longitudinal data, standard analytic methods can be biased. This study examined the effect of parental supply of alcohol on alcohol-related outcomes in early adulthood using robust methods to minimise risk of bias.

Design Prospective longitudinal cohort study.

Setting Australia

Participants Cohort of school students (n=1906) recruited in the first year of secondary school (average age 12.9yrs) from Australian schools in 2010-11, interviewed annually for 7 years.

Measurements The exposure variable was self-reported parental supply of alcohol (including sips/whole drinks) across five years of adolescence (waves 1-5). Outcome variables were self-reported binge drinking, alcohol-related harm, and symptoms of alcohol use disorder, measured in the two waves after the exposure period (waves 6-7). To reduce risk of bias, we used Targeted Maximum Likelihood Estimation to assess the (counterfactual) effect of parental supply of alcohol in all five waves versus no supply, on alcohol-related outcomes.

Findings Parental supply of alcohol across adolescence saw greater risk of binge drinking (RR:1.53; 95% CI:1.27-1.84) and alcohol-related harms (RR:1.44; 95% CI:1.22-1.69) in the year following the exposure period compared with no supply in adolescence. Earlier initiation of parental supply also increased risk of binge drinking (RR:1.10; 95% CI:1.05-1.14), and any alcohol-related harm (RR:1.09; 95% CI:1.05-1.13) for each year earlier parental supply began compared with later (or no) initiation.

Conclusions Adolescents whose parents supply them with alcohol appear to have an increased risk of alcohol-related harm compared with adolescents whose parents do not supply them with alcohol. The risk appears to increase with earlier initiation of supply.

Keywords: Alcohol, adolescence, epidemiology, targeted maximum likelihood estimation, confounding, cohort studies

Introduction

The use of alcohol and other drugs cause a substantial health burden worldwide (1, 2). Alcohol consumption in early life is an important global health issue, recognised as the leading risk for disability adjusted life years in 10-24-year-olds (3), as well as being associated with non-communicable diseases (4) and other health burden (5). Despite the potential for harm caused by alcohol consumption, parents remain a major provider of alcohol to adolescents (6). While this is sometimes attributed to a desire to mitigate harm, there is little evidence for a beneficial effect of parental supply of alcohol. In fact, recent research has shown that parental supply of alcohol is associated with significantly higher odds of risky drinking, alcohol-related harm, and symptoms of alcohol use disorders (AUD) (7, 8).

Previous analyses have focussed on point estimates of exposure, typically using a relatively proximal outcome (e.g. measuring the association between exposure in one year and outcome in the following year). Thus, longer-term relationships such as joint effects of repeated exposure over time, or the cumulative effects of long-term exposure, remain unclear. In addition, parental supply of alcohol is not static, potentially varying for each individual over the course of adolescence. Consequently, the overall effect of parental supply of alcohol on later harm is uncertain. To date there has been no attempt to consider the overall, cumulative effect of parental supply of alcohol across adolescence, nor to examine the effect of different 'courses' of supply, that is, to examine the effect of initiating parental supply earlier in adolescence.

When examining the joint effect of an exposure across multiple time-points, additional sources of bias must be addressed beyond those considered in standard analyses of observational data. One such source of bias is time-varying confounding, where confounders of the relationship between an exposure and an outcome are themselves effected by prior

exposures (9). This issue can bias standard analytic techniques (10), even to the point of leading to contradictory conclusions (11). A range of different statistical methods for handling time-varying confounding have been developed, with the most commonly used being inverse probability of treatment weighted marginal structural models (IPW) (12). These use the propensity score (the probability of exposure) to create a weighted outcome model which is a consistent estimator of causal effects such as average treatment effects, provided the propensity is correctly estimated. Alternative methods have focused on the outcome model rather than the propensity model. For example, G-computation (13, 14) models the outcome given exposure and covariates, and compares what would have been expected had everyone in the sample received specific levels of the exposure. More recently, a number of more robust techniques have been proposed, which utilise models of both exposure and outcome, but require only one of the two to be specified correctly to provide consistent estimates (15). One such method is targeted maximum likelihood estimation (TMLE) (16), which involves estimating initial models of both the outcome and the exposure of interest, and then updating the outcome model based on a function of the initial estimates (17, 18).

The aim of this study was to estimate the joint effect of parental supply of alcohol across adolescence on a number of alcohol-related outcomes in early adulthood, using observed data from the Australian Parental Supply of Alcohol Longitudinal Study (APSALS) cohort (8) of adolescents. In doing so, we compared the effects of a number of different trajectories of parental supply across five years of adolescence (waves 1-5 of the cohort) on the risk of five different alcohol-related outcomes in the two subsequent years (wave 6 and wave 7): binge drinking, alcohol-related harms, and symptoms of alcohol abuse, dependence, and alcohol use disorder (AUD). To adjust for exposure-affected time-varying confounding, we estimated effects using TMLE.

Methods

APSALS cohort

We analysed data from the APSALS cohort (registered as a longitudinal cohort with ClinicalTrials.gov: NCT02280551), a sample of n=1906 adolescents recruited in the first year of secondary school (average age 12.9 years) from Australian schools in 2010-11 and surveyed annually via either pen-and paper or online survey. Details of the sample are provided elsewhere (7, 8, 19). Signed consent was obtained from participating parents. Ethical approval was given by UNSW Sydney, Curtin University, and the universities of Tasmania, Newcastle, and Queensland. The STROBE checklist and recruitment/retention flow diagram are included in Supplementary Appendix A. The current analysis includes data from seven years of the study (waves 1- 7).

Primary outcome variables

We present analyses of five binary outcomes: binge drinking, alcohol-related harms, and three outcomes based on the Diagnostic and Statistical Manual of Mental Disorders (DSM) criteria for substance use disorders: two based on DSM-IV (abuse and dependence) (20) and one based on DSM-5 (AUD) (21).

Binge drinking

We defined binge drinking based on the Australian National Health and Medical Research Council (NHMRC) guidelines (22), which recommend that consumption of alcohol not exceed 40g (four standard drinks) on a single occasion to reduce the risk of alcohol-related injury arising from that occasion. As such, we defined binge drinking as consumption of more than four standard drinks on a single occasion in the previous year, coded as a binary variable (no/yes). Participants were also asked how frequently they drank four or more drinks in the past year, which was used for sensitivity analyses of the frequency of binge drinking.

Experience of alcohol-related harm

Based on questions from the School Health and Alcohol Harm Reduction Project (SHAHRP) (23), adolescents were asked how often (if at all) they had experienced a range of different alcohol-related harms in the last 12 months. The scale includes 17 different harms, which were then used to code into a binary (no/yes) variable indicating whether the participant had experienced any of the 17 harms, based on past research (8), as well as a scale score of the number of harms experienced at least once, ranging from 0-17. Because the first two items of the SHAHRP are less severe, and potentially overlap with binge and other risky drinking, we conducted a post-hoc sensitivity analysis excluding the first two items from the scale.

DSM outcomes

Symptoms of DSM-IV and DSM-5 disorders were assessed using the alcohol abuse items from the National Institute of Health Diagnostic Interview Schedule for Children Version IV (NIMH DISC-IV) (24), which includes self-reported experience of DSM-IV and DSM-5 AUD symptoms. Adolescents were considered to have experienced DSM-IV alcohol abuse if they reported experiencing at least one of the four abuse symptoms in the last 12 months. Similarly, they were considered to meet DSM-IV dependence criteria if they reported at least three of the seven dependence symptoms, and to meet DSM-5 AUD criteria if they reported experiencing at least two of the eleven alcohol use disorder symptoms. Each of the three variables was coded into a binary (no/yes) variable.

Exposure variable

The primary exposure considered in this analysis was parental supply of alcohol, as reported by the adolescent. Adolescents were asked who had supplied them alcohol in the past 12 months, with possible sources including mother, father, other adults, friends, siblings, self-supply and religious services, how often they received supply from those sources, and the

quantity supplied. Based on responses to these questions, a dichotomous exposure variable was coded indicating those who had received supply of alcohol from parents, and those who had not. For the purposes of these analyses, parental supply of alcohol included supply of whole drinks and sips. Because the analysis is intended to examine the joint effect of exposure across five years of adolescence, our primary analysis compares the two most extreme trajectories of exposure: no parental supply in any of the five years versus supply in all five years. This represents the maximum possible effect of parental supply. In addition, as a post-hoc secondary analysis, we also consider the effect of delaying supply to age 15 (in line with NHMRC guidelines to delay alcohol initiation until at least age 15)(22), but supplying thereafter, versus no supply at all. Finally, we also consider the effect of earlier initiation of supply, analysed as the additive effect of initiating supply in an earlier year and continuing to supply thereafter – that is, the effect of initiating supply at age 12 compared with age 13; age 13 compared with age 14; and so on – estimated by parameters from a working marginal structural model, in which wave of initiation was considered as a continuous predictor variable.

Confounding variables

We included a range of baseline (Wave 1) and time-varying (i.e. that vary with each wave) confounding variables in our analysis based on prior research with APSALS (7, 8) and identified from the broader literature (25-34). Wave 1 variables comprised parental factors (parental religiosity, parent employment and birth country, and parental responsiveness, demandingness and consistency), and familial factors (family conflict/positive relations, household income and area level socioeconomic status). Time-varying confounders comprised supply of alcohol from other sources (e.g., peers), parental factors (parent alcohol use, alcohol-specific rules and parental monitoring), family factors (one- or two-parent household, access to alcohol without parental knowledge), child factors (child gender, money

to purchase alcohol, tobacco use, externalising behaviour, anxiety and depression symptoms, and problems socialising), and peer factors (peer substance use, and peer disapproval of alcohol and tobacco use). More detail on the covariates is included in Supplementary Appendix B.

Statistical analysis

We analysed the joint effect of parental supply of alcohol from waves 1-5 (early-mid adolescence) on outcomes at wave 6 (average age 17.8 years) and wave 7 (average age 18.8 years), when participants were reaching the legal age to purchase alcohol. That is, the primary causal estimand was the effect of parental supply at all five waves, compared with no parental supply at any wave. Analysis was based on a counterfactual framework, using models to construct expected probabilities had everyone in the sample experienced those particular patterns of exposure. These counterfactual estimates are based on sum of the estimates of the effect of supply in each wave, based on all observations, regardless of their observed pattern of exposure. Thus, patterns of exposure can be compared based on models that use all data points, regardless of the number with that *observed* pattern of exposure, increasing the power of analyses. The assumed causal structure can be seen in Figure 1, where A_t is the exposure (parental supply of alcohol) at each time point, B is the set of wave 1, time-constant confounders, L_t is the set of time-varying confounders, and Y is the outcome.

In addition to the primary analysis, two post-hoc sensitivity analyses were also conducted. Firstly, to test for the possible effect of including covariates from the same wave as the exposure variable, the data were re-analysed using only covariates from previous waves. Secondly, to address the potential confounding effect of previous levels of the outcome variables, the data were re-analysed including the observations of the levels of the outcomes during the exposure period.

Because it is likely that at least some of the time-varying confounders are affected by exposure at previous waves (as indicated in Figure 1 by the arrows connecting A_{t-1} to L_t), analysis using standard regression techniques is likely to be biased. As such, analyses were conducted using targeted maximum likelihood estimation (TMLE). For comparison purposes, we also conducted naïve analyses using generalised linear models (GLMs).

TMLE (16) is a consistent, doubly robust method for estimating causal effects, estimated via a three step process. Firstly, initial models of the probability of exposure (i.e. the propensity, similar to IPW) and expectation of the outcome (similar to G-computation) are estimated. These initial estimates are then used to 'update' the initial outcome model. This model is then evaluated at different counterfactual exposure values, to provide a targeted estimated of the causal effect of exposure. Because counterfactual exposures are used, patterns of exposure can be compared based on models that use all data points, regardless of the number with that *observed* pattern of exposure, increasing the power of analyses. More detail of the method is included in Supplementary Appendix C. The two initial models used in TMLE can be estimated using GLMs, but are often estimated using machine learning, and in particular Super Learner (35), an ensemble machine learning algorithm. Implementation via Super Learner has the advantage over GLMs in that in addition to GLMs it can also include adaptive, flexible algorithms which allow for complex and non-linear relationships such as interactions and polynomial effects, without explicitly specifying those effects. The final, updated outcome model is then carried out using GLMs. We conducted all analyses using longitudinal TMLE (LTMLE), via the 'ltmle' (36) program in R 3.5.1 (37), with models estimated via SuperLearner using a range of prediction algorithms: means, generalised linear models, generalised additive models (38), and random forests (39). Analysis code is available at https://www.philipclare.com/code/apsals/. Because all outcomes were binary, results are reported as risk ratios with corresponding 95% confidence intervals, calculated based on the

ratio of the predicted probability of the outcome in the exposure vs the control group from the TMLE model. The specific analyses presented in this study were not pre-registered and thus should be considered exploratory.

<INSERT FIGURE 1 AROUND HERE>

Assumptions of TMLE

In order to produce valid inference, TMLE requires that a number of assumptions be met. The first assumption is that of *no interference*, which is that the association between exposure and outcome for each participant is independent of the exposure of other participants (40). In this case, the effect of parental supply of alcohol and subsequent alcohol-related outcomes is likely to be independent of the exposure of other participants. That is, while there is a possibility participants may be in the same peer group, the assumption of *no interference* would only be violated if participants both had peers in the study, and also affected the role of parental supply in those peers. The second assumption is *consistency*, which assumes that the counterfactual outcome associated with a given exposure, and the actual outcome observed if that exposure occurs are the same (41). This assumption can be violated if, for example, the exposure is defined ambiguously. In the case of this study, it is unlikely that the outcome could be affected by ambiguity in the definition of the exposure, although there is the possibility that the effect of supply differs based on things like the motivation of the parents, and even the drinking culture in which supply occurs. Thirdly, valid inference depends on the assumption that there is *no unmeasured confounding* (42). While effects of known and measured confounders can be eliminated via modelling, it is impossible to guarantee that there are no unknown factors that influence the relationship between parental supply and alcohol-related outcomes. As a sensitivity analysis, E-Value (43) analysis was conducted via the EValue package in R to test the strength of association required for unmeasured confounding to be likely. Finally, the analysis assumes *positivity* (44) (or *near-positivity*), that

is, that there are no participants for whom the probability of exposure is zero (or very close to zero). In this case, given the relatively high probability of exposure, the assumption of *positivity* is not violated, and near-positivity is unlikely to be. In addition, TMLE is more robust to near-positivity violations than other causal estimators (45).

Missing Data

Because the study is a longitudinal survey, there were a number of participants with missing data. To reduce potential bias from this missing data, analysis was conducted using multiple imputation. In order to take into account within-subject clustering/correlation in the data, based on past research (46) we initially conducted imputation using multivariate normal imputation, however the method failed to converge. Consequently, the final imputations were using joint modelling of latent normal distributions using multi-level models, via the 'mitml' package in R (47), which conducts multiple imputation using multilevel models. Imputation models included all variables used in the analyses, including all outcomes in the same imputation model. Analyses were then conducted on each imputed dataset, and combined using Rubin's rules with the R package 'Amelia' (48). Based on the proportion of missing data, we used M=50 imputations (49). Further details of the missing data and the imputation procedure are included in Supplementary Appendix D.

Results

Sample

Baseline socio-demographic characteristics of the sample are included in Table 1. In wave 1 when participants were an average age of 12.95 years, 16.1% of participants reported receiving alcohol from their parents in the past year. This increased steadily over the exposure period, peaking at just under half the sample (45.7%) by wave 5, when the average age of participants was 16.88 years. Around two in five received no parental supply in the

exposure period (39.4%), while 5.3% received alcohol from their parents in all five waves (Supplementary Table S3).

<INSERT TABLE 1 AROUND HERE>

Of the five outcomes considered, binge drinking and experience of alcohol-related harms were common, with over half of the sample reporting binge drinking in wave 6 (55.5%), and most reporting they had done so in wave 7 (80.6%). Report of individual types of harms varied, with some very common (e.g. hangovers), while more serious harms were rare, as seen in Supplementary Table S4. Similarly, the majority reported experiencing at least one alcohol-related harm in the past year in wave 6 (67.7%), which increased in wave 7 (85.3%). The three DSM-based outcomes were less common, but still reported by up to a quarter of the sample in wave 6 (23.6% for DSM-5 AUD), and just under half of the sample in wave 7 (42.0% for DSM-5 AUD; see Table 2). Predicted prevalence of each outcome associated with the counterfactual exposure patterns is included in Supplementary Table S5.

<INSERT TABLE 2 AROUND HERE>

Targeted Maximum Likelihood Estimation

When examining outcomes 1-year after the exposure period, when the average age of participants was 17.79 years, parental supply of alcohol across waves 1-5 led to greater risk of binge drinking (RR: 1.53; 95% CI: 1.27-1.84) and any alcohol-related harm (RR: 1.44; 95% CI: 1.22-1.69), but there was less evidence to suggest an effect on DSM-IV abuse (RR: 1.51; 95% CI: 0.72-3.17), DSM-IV dependence (RR: 1.94; 95% CI: 0.94-4.02), or DSM-5 AUD (RR: 1.66; 95% CI: 0.91-3.04), as seen in Figure 2a and supplementary Table S6. These results were similar in analyses of wave 7 outcomes, when average age was 18.77 years (Figure 2b), although the effect sizes for binge drinking and any harms were around half those of the wave 6 analyses, suggesting diminishing effect of adolescent exposure. In

contrast, secondary analyses of supply from age 15 (versus no supply) showed little evidence of any effect on either wave 6 or wave 7 outcomes, as seen in Supplementary Figure S2.

<INSERT FIGURE 2 AROUND HERE>

Consistent with the primary analysis, examination of the effect of earlier initiation of parental supply showed that the effect of parental supply on binge drinking and alcohol-related harms increased as age of initiation of parental supply decreased. That is, for each year earlier that initiation of parental supply of alcohol began, there was an approximately 10% increase in the risk of wave 6 binge drinking (RR: 1.10; 95% CI: 1.05-1.14), and any alcohol-related harm (RR: 1.09; 95% CI: 1.05-1.13). In contrast to the results of the primary analysis, there was also evidence that younger age of initiation of parental supply had an effect on DSM-IV dependence and DSM-5 AUD, with the risk increasing by 15% (RR: 1.15; 95% CI: 1.03-1.27) and 13% (RR: 1.13; 95% CI: 1.04-1.24) respectively for each year earlier parental supply was initiated. However, the same pattern was not observed for DSM-IV abuse (RR: 1.09; 95% CI: 0.98-1.21).

A similar pattern was observed for the wave 7 outcomes, with earlier initiation associated with an approximately 5% increase in the risk of binge drinking (RR: 1.06; 95% CI: 1.04-1.09), any alcohol-related harm (RR: 1.05; 95% CI: 1.03-1.08), and DSM-5 AUD (RR: 1.06; 95% CI: 1.00-1.12), although the effects on DSM-IV dependence (RR: 1.06; 95% CI: 0.98-1.15) and did not remain, as seen in Table 3. Importantly, these effects are additive, with greater increases in risk for earlier initiation, as seen in supplementary Figures S3-S7.

<INSERT TABLE 3 AROUND HERE>

Sensitivity analyses

Sensitivity analyses controlling only for lagged covariates (i.e. excluding covariates from the same wave as the exposure) were consistent with the primary analyses, as seen in

Supplementary Figure S8. Similarly, sensitivity analyses controlling for past observations of the outcomes were also consistent with the primary analyses, as seen in Supplementary Figure S9. Sensitivity analyses of frequency of binge drinking and the number of harms experienced were also generally consistent with the primary analyses, with parental supply associated with greater frequency of binge drinking, and greater numbers of harms experienced, as seen in Supplementary Figure S10. Sensitivity analyses of alcohol-related harms were broadly consistent with the primary analysis. When the first two items were excluded from the scale, the effect of parental supply on the wave 6 outcome remained, with marginally higher risk ratio than the primary analysis (RR: 1.53; 95% CI: 1.23-1.90), however the effect was not evident in the wave 7 outcome, due to marginally lower RR and wider confidence intervals (RR: 1.12; 95% CI: 0.95-1.31), as seen in Supplementary Figure S11. Finally, for comparison purposes, naïve analyses using GLMs are shown in Supplementary Figures S12-S13.

E-Value Analysis

E-value sensitivity analysis suggests that in order to alter the findings, unmeasured confounders would need to have a fairly strong average association with the exposure and outcome, with E-values ranging from a RR of 1.64 to 3.29, depending on the particular outcome (Supplementary Table S7).

Discussion

This study represents the first attempt to quantify the effect of parental supply of alcohol over the course of adolescence on alcohol-related harms in early adulthood. Novel, robust statistical techniques were used to account for the complex sources of bias that can be introduced by longitudinal analysis of observational data. Consistent with previous work (8), the results provide further evidence that parental supply of alcohol in adolescence has effects

on a number of negative outcomes in early adulthood, including binge drinking and alcohol-related harm, leading not only to increased risk of binge drinking and harm, but also increased frequency of binge drinking and number of harms experienced. Analysis of earlier initiation of supply showed that the magnitude of the effect of parental supply increased the earlier supply was initiated, but supply initiated in later adolescence was still associated with negative outcomes. While these effects are relatively proximal and diminish as the time between exposure and outcome increases, some associations remained two years after the exposure period. This may be due to the diminished influence of parents as adolescents reach adulthood, both because they are able to purchase alcohol themselves, but also because they are increasingly likely to move out of their family home. Analysis of initiation of supply at age 15 showed no significant differences to no supply at all across adolescence. This suggests that early adolescence in particular is important, and provides evidence supporting Australian guidelines to avoid supply of alcohol until age 15, although NHMRC's recently released revised guidelines are more conservative, and recommend avoiding alcohol until age 18 (50). Importantly however, there was no evidence that parental supply, either across adolescence, or initiated at age 15, led to any reduction in harm or other benefit with respect to adolescent drinking behaviour.

Unlike previous work, which found that parental supply of alcohol was associated with DSM-IV dependence and DSM-5 AUD symptoms (8), these results do not provide evidence for a robust association between parental supply of alcohol and DSM-IV or DSM-5 symptoms. However, it remains unclear whether this is due to a lack of effect, or if the study simply lacked the power to detect effects.

It is worth noting that the definition of binge drinking used in this study is based on Australian guidelines, and differs from those used in other jurisdictions (for example, the United States National Institute on Alcohol Abuse and Alcoholism, which defines binge

drinking as 4 or more drinks for women and 5 or more drinks for men). Thus, it may be that some of the effect is due to a lower threshold for binge drinking, particularly among males.

The mechanisms also remain to be established. For example, it may be that parental supply acts as an initiation to a local drinking culture in the area in which the family resides. Thus, where there is a hazardous drinking culture, parental supply may lead to negative outcomes. Further work is required to explore the influence of parental supply of alcohol in different cultures. This also raises the possibility that the findings may not generalise beyond the Australian context; the effects of parental supply may be distinct elsewhere. Alternatively, there may be biological mechanisms, particularly in light of heritable traits associated with addiction. That is, it may be that biological traits associated with addiction make it more likely both for parents to supply alcohol to their children, and for those children to experience later harm. While the analysis controlled for parental drinking as a marker of parent alcohol problems, the possibility remains, although E-value analysis suggests that the effects would need to be strong in order to explain the effects.

Limitations

We considered only a subset of the potential (counterfactual) patterns of exposure, with supply considered to continue once started. A number of other possible patterns of supply exist that could be considered. For example, future analyses might consider the effects of stopping parental supply after initiation, based on criteria (for example, parents who stop supply alcohol when their children begin to experience harm). In addition, our measure of supply is relatively coarse – in reality supply is likely to vary within years as well as between, as well as changing in terms of frequency and quantity of supply. Further work is therefore required to explore supply in more detail, although our study represents one of the most detailed studies of parental supply available to date.

Second, the study relies on self-report data. While there is evidence to suggest that self-report of alcohol behaviours and consequences is generally valid (51, 52), there are known to be problems with recanting of former reports in adolescent cohort studies (53). It is also possible that the reporting of parental supply is biased, for example after heavy drinking (54), although past research has shown relatively high agreement between child and parent report of supply (8). Also, while the prevalance of the DSM-based outcomes is likely inflated compared to those asssessed clinically, the levels of symptoms reported by the cohort is not unexpected given relatively high rates of alcohol use disorder in community samples in Australia (55) and other high-income countries (56, 57).

A strength of TMLE is that, provided structural assumptions hold, findings can be interpreted as causal effects. It is possible that these structural assumptions were violated, undermining causal interpretation. However, we do not believe that the assumptions of no interference, consistency or positivity are likely to have been violated, and E-value analysis suggests that relatively strong unmeasured confounding would be required to influence the findings. That is, while unmeasured confounding remains possible, the analysis suggests that in order to alter the conclusions the unmeasured confounding would need to have an effect as strong or stronger than parental supply, or any other exposure assessed previously in the cohort.

Conclusions

Consistent with previous research, the study provides evidence that parental supply of alcohol in adolescence can lead to higher risk of binge drinking and alcohol-related harms in the short-term. Further, there is no evidence to suggest that parental supply of alcohol acted as a protective factor for any of the adult harms examined. In addition, the study provides evidence that the risks associated with parental supply increase the earlier such supply is initiated. Thus, the results suggest that parental supply of alcohol should be avoided, but in particular that parents should not supply alcohol in early adolescence.

Table 1 Child and household socio-demographic characteristics of the sample at wave 1

	n (%) / mean (SD)
Age	12.9 (0.5)
Child gender	
Male	1,051 (55%)
Female	855 (45%)
Parent born in Australia	
No	501 (26%)
Yes	1,391 (74%)
Parent employment at wave 1	
Employed (full-time/part-time)	1,534 (81%)
Unemployed (in workforce)	237 (13%)
Unemployed (not in workforce)	121 (6%)
Relative socioeconomic disadvantage of area of residence	
Low	327 (17%)
Medium	414 (22%)
High	1,151 (61%)
Household income	
Up to $34,000	160 (9%)
$35,000 to $80,000	464 (25%)
$81,000 to $180,000	918 (49%)
$181,001 or more	340 (18%)

Table 2 Percentage of sample reporting parental supply in exposure waves (1-5) and outcomes in outcome waves (6-7)

	Percentage
Parental Supply of Alcohol	
Wave 1	16.1%
Wave 2	26.2%
Wave 3	28.2%
Wave 4	34.6%
Wave 5	45.7%
Binge Drinking	
Wave 6	55.5%
Wave 7	80.6%
Any alcohol-related harms	
Wave 6	67.7%
Wave 7	85.3%
DSM-IV Abuse	
Wave 6	8.1%
Wave 7	16.5%
DSM-IV Dependence	
Wave 6	12.6%
Wave 7	23.2%
DSM-5 Alcohol Use Disorder	
Wave 6	23.6%
Wave 7	42.0%

Table 3 Adjusted risk ratio of wave of initiation of parental supply versus no parental supply in any wave on five alcohol-related outcomes in wave 6 or wave 7

Outcome	Wave 6 RR (95% CI)	Wave 7 RR (95% CI)
Binge drinking	1.10 (1.05-1.14)	1.06 (1.04-1.09)
Any harms	1.09 (1.05-1.13)	1.05 (1.03-1.08)
DSM-IV Abuse	1.09 (0.98-1.21)	1.05 (0.97-1.13)
DSM-IV Dependence	1.15 (1.03-1.27)	1.06 (0.98-1.15)
DSM-5 Alcohol Use Disorder	1.13 (1.04-1.24)	1.06 (1.00-1.12)

Note: RRs represent effect of initiating supply one year earlier, and then continuing supply thereafter (e.g. the effect of initiating supply at age 12 versus age 13, or age 14 versus age 15). Effects are additive, increasing as supply is initiated earlier. RRs for differences of more than one year are included in Supplementary Figures S3-S7.

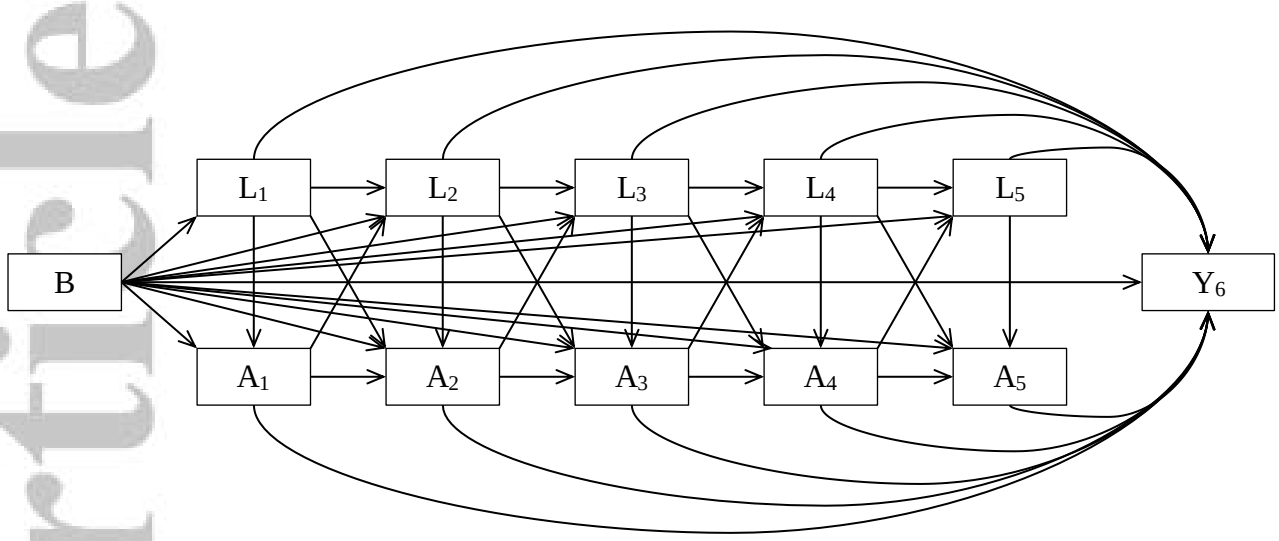
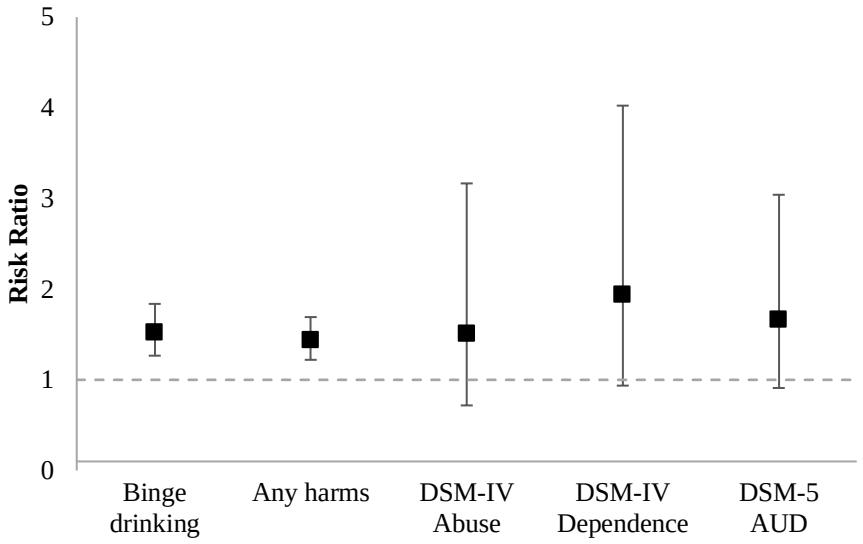


Figure 1 Directed Acyclic Graph of proposed causal structure

Note: B is the set of baseline, time-constant variables; A is the exposure variable measured at each time point; L is the set of exposure-affected time-varying confounders measured at each time point; and Y is the outcome variable measured at the final time point.

(a) Wave 6



(b) Wave 7

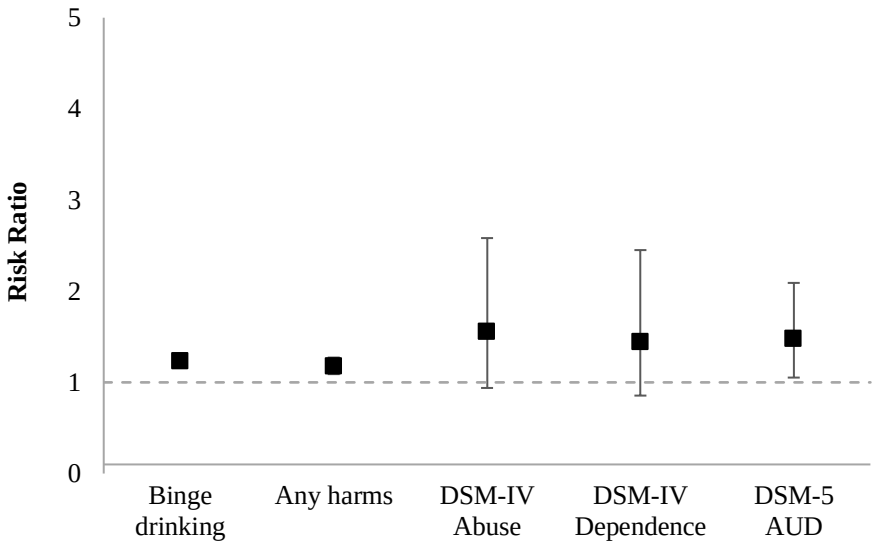


Figure 2 Adjusted risk ratios of supply in all five years of adolescence versus no supply in any wave on five alcohol-related outcomes in wave 6 or wave 7

Note: Null value shown as dotted line. Models controlled for supply of alcohol from other sources, parent alcohol use, parental religiosity, parent employment and birth country, alcohol-specific rules, parental monitoring, parent responsiveness, parent demandingness, parenting consistency, family conflict, family positive relations, household income, area level socioeconomic status, where live in one-parent household, access to alcohol without parental knowledge, child gender, money to purchase alcohol, tobacco use, externalising behaviour, anxiety/depression, problems socialising, peer substance use, and peer disapproval of alcohol and tobacco use.