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Parent hazardous drinking and their children's alcohol use in early and mid-adolescence: prospective cohort study

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Background: Why adolescents' drinking is associated with their parents' drinking remains unclear. We examined associations in a prospective cohort study, adjusting for socio-demographic characteristics and family factors. **Methods:** We recruited 1927 children from grade 7 classes (mean age 13 years), and one of their parents, in three Australian states, contacted participants annually from 2010 to 2014, and analysed data from assessments at ages 13, 14, 15 and 16 years. We used the Alcohol Use Disorders Identification Test-Consumption (AUDIT-C) subscale to identify hazardous drinking in parents (score $\geq$5) and children (score $\geq$3) and constructed mixed-effect logistic regression models, accounting for clustering within school and adjusting for likely confounders. We evaluated the sensitivity of estimates by imputing missing values assuming the data were missing at random vs. missing not at random. **Results:** Parent hazardous drinking predicted mid-adolescent hazardous drinking, e.g. 15 years olds whose parents [adjusted odds ratio (aOR) 2.00; 95% confidence interval 1.51–2.64] or parents' partners (aOR 1.94; 1.48–2.55) were hazardous drinkers had higher odds of being hazardous drinkers at age 16. The magnitude of univariate associations changed little after adjusting for covariates, and sensitivity analyses confirmed the robustness of the association, across a wide range of assumptions about the missing data. **Conclusions:** The associations between parents' and their adolescent children's hazardous drinking are unlikely to be due to confounding by socio-demographic and family factors. Parents should be encouraged, and supported by public policy, to reduce their own alcohol consumption in order to reduce their children's risk of becoming hazardous drinkers.

Introduction

Hazardous drinking in adolescence is a global health priority due to its adverse health and social outcomes, including cognitive impairment, lower educational attainment, substance dependence, injury and sexually transmitted infection.[1] Hazardous drinking is defined as a pattern of alcohol use that significantly increases the risk of harmful consequences.[2] In Australia, 16% of boys and 13% of girls aged 16–17 years report past-month hazardous drinking, and 15% of this age group experience alcohol-related harm at least annually.[3] Alcohol-related injury presentations to the emergency department peak among 15–19-year-olds and the incidence has increased in recent years, e.g. by 63% among girls from 2005 to 2012.[4]

Risk factors for adolescent hazardous drinking include impaired educational attainment,[5] poor family relations[6] and internalizing or externalizing psychological disorders.[5,7] Risk markers include male gender,[5] genetic factors[8] and family history.[9] Contextual factors include normative expectations based on the minimum legal drinking age,[5] the price of alcohol,[10] its accessibility,[5] peer influence,[7] parental influence[5] and socio-economic deprivation among adolescents and the communities in which they live.[11]

A major threat to the validity of causal inferences from observational data is the possibility of unadjusted confounding. A 2015 systematic review reported that four longitudinal studies (of 21 identified as eligible for inclusion), judged to have designs that provide capacity for causal inference, suggest that parents' hazardous drinking predicted hazardous drinking in their children.[12] However, inferences were constrained by the possibility of bias due to lack of adjustment for potential confounders.[12] A 2017 meta-analytic review of nine studies also found a positive association [mean effect size: $r = 0.15$; 95% confidence interval (CI) 0.09–0.22], but estimates were unadjusted, such that plausible competing explanations could not be excluded.[13] Thus, to what extent the association is causal remains unclear.

Prospective measurement of the exposure and confounders, with adjustment in multivariable models, is required to minimize bias in

non-randomized study designs. Identification of potential confounders should be based, where possible, on empirical observation in similar populations, and their hypothesized role specified in advance of the analysis.[14]

With data from a large cohort of parent-child dyads assessed annually, we are able to address critical gaps in the literature: (i) whether the association between parent hazardous drinking and the development and/or continuation of hazardous drinking in adolescence is confounded by parent socio-demographic factors and family factors; and (ii) the extent of association between parent hazardous drinking and later adolescent hazardous drinking after adjusting for possible confounders.

Methods

Design

We conducted a prospective cohort study, recruiting 1927 parent-child dyads from 49 schools (Grade 7 classes) in 2010, in New South Wales, Tasmania and Western Australia. Adolescents and one of their parents completed either paper-based or online questionnaires annually (see ClinicalTrials.gov: NCT02280551).[15] The protocol was approved by the University of New South Wales Human Research Ethics Committee (Reference HC 10144).

Sample

We used four waves of complete parent-child dyad data, from 2010 (T1) to 2014 (T4). Retention was >85% throughout the annual follow-ups. The sample size was 1896 dyads at T1–T2, 1806 at T2–T3 and 1741 at T3–T4. The mean age of adolescents and parents at T1 was 13 (SD = 0.5) and 44 (SD = 5.4) years, respectively, and 87% of responding parents were mothers. T1 demographic factors (table 1) were similar to those of the Australian population: 45% of adolescents were female (vs. 49% of the population); 80% lived in two-parent households (vs. 81%); 81% of parents were employed (vs. 88%); 74% of the parents' country of origin was Australia (vs. 72%); and the mean socio-economic status index for the sample was 1023 (SD = 80; Australian standardized mean = 1000; SD = 100).[16–18]

Analytic approach

We used directed acyclic graphs to hypothesize associations between exposure, confounder and outcome variables (Supplementary figures S1 and S2),[14] identified but did not adjust for potential intermediate variables, to avoid overadjustment, which usually biases estimates towards the null.[19]

The confounders we hypothesized have been found to be associated with parent drinking and/or adolescent drinking. For example, we adjusted for socio-demographic characteristics and household composition because previous research shows that single mothers and parents with low socio-economic status were more likely to drink hazardously.[20] Low parental socio-economic status[11] and single-parenthood[21] have also been found to be associated with adolescent hazardous drinking, so we adjusted for them in our models.

We adjusted for family history of alcohol use disorder because previous research shows that grandparents' hazardous drinking was associated with their children's hazardous drinking and grandchildren's problem behaviour, a precursor of adolescent hazardous drinking.[22] Research has also suggested that adolescents whose first-degree and second-degree relatives were hazardous drinkers are at increased risk of becoming hazardous drinkers.[9]

We adjusted for religiosity because it has been found to protect against children's alcohol use.[23] Additionally, Rossow *et al.*[12] noted in their review that existing studies seldom accounted for religious factors that may be prescriptive in relation to parent and adolescent drinking behaviour.

Table 1 Socio-demographic characteristics of the cohort of baseline (T1)

Demographics	Cohort (n = 1896), mean (SD) or %
Participating parent's demographic characteristics	
Reporting parent is female	1635 (87)
Age (years), mean (standard deviation)	44 (5.4)
Religiousness	
Very important	262 (14)
Pretty important	297 (16)
Not or little important	1336 (70)
Participating parent's education	
School Certificate or below	423 (22)
High School Certificate/Diploma	786 (41)
University degree	681 (36)
Participating parent's employment status	
Employed (full-time or part-time)	1538 (81)
Unemployed	347 (19)
Household income (Australian dollars)	
Up to $34 999	161 (9)
$35 000–$80 999	464 (25)
$81 000–180 999	920 (48)
$181 000 or more	341 (18)
Participating parent's country of origin	
Australia	1395 (74)
United Kingdom	230 (12)
New Zealand	62 (3.3)
Africa	70 (3.7)
Asia	65 (3.4)
Europe	34 (1.8)
Other	40 (1.8)
Household composition	
Two parent household	1525 (80)
Family conflict (Score 0–3)	3.8 (0.9)
Positive family relations (Score 0–3)	5.9 (0.5)
Family history of heavy drinking	
No	923 (48)
Yes	885 (47)
I don't know	83 (5)
Participating child's demographics	
Age, mean (range)	13 (0.5)
Child is female	851 (45)
School type	
Government	701 (37)
Catholic	341 (18)
Independent	845 (45)

We included positive family relationships and family conflict as potential confounders because in a US trial, the family intervention Brief Strategic Family Therapy programme was found to be effective in reducing parent and adolescent alcohol use.[6]

Measures

Outcome

We assessed adolescent hazardous drinking with the Alcohol Use Disorders Identification Test-Consumption (AUDIT-C), dichotomizing the cohort as 'non-hazardous drinkers' (AUDIT-C < 3) vs. 'hazardous drinkers' (AUDIT-C ≥ 3), relying on empirically-based recommendations for adolescents.[24]

Exposures

We dichotomized parents and their partners as 'non-hazardous drinkers' (AUDIT-C < 5), vs. 'hazardous drinkers' (AUDIT-C ≥ 5), in light of Australian research that identified this cut-off as optimal.[25] The partner's alcohol use was reported by the participating parent, who was the mother in 87% of cases.

Confounders

Parent socio-demographic factors At T1, we asked parents how important religion was in their lives ('very important', 'pretty

important' or 'not important or of little importance'). In the analyses, we categorized parent educational qualifications as 'school certificate or below', 'high school certificate or diploma' and 'university level degree'; household annual income as 'low (<$35 000)', 'middle ($35 000–$80 999)' or 'high (≥$81 000)'; and parent employment status as 'unemployed' or 'employed'.

Household composition We asked adolescents with which family members they lived most of the time, and categorized response options for analyses as 'one-parent household' (consisting of any lone parent, including a step-parent) or 'two-parent household' (consisting of father and mother, father and step-mother or mother and step-father).

Positive family relationships We evaluated the quality of family relationships from parent report using three items with yes/no response options: 'Family members support one another', 'There are feelings of togetherness in our house' and 'Family members get along well'.[26] Possible scores ranged from 0 to 3, with higher scores indicating more positive relationships (Cronbach's alpha = 0.71).

Family conflict We assessed levels of family conflict from parent report using three items with yes/no response options: 'Family members have big arguments over little things', 'Family members get angry with each other daily' and 'Family members get angry with each other three times a week'.[26] Possible scores ranged from 0 to 3, with higher scores indicating greater conflict in the family (Cronbach's alpha = 0.57).

Family history of heavy drinking At T1, we asked parents whether their children's grandparents, aunts or uncles, on either side, ever drank heavily, with response options yes/no/I don't know,[27] classifying 'I don't know' as missing.

Statistical analysis

We performed logistic mixed-effects regression modelling (accounting for clustering within schools using a random intercept for school), on complete case data, to estimate crude and adjusted associations. We analysed contiguous pairs of time periods: T1–T2, T2–T3 and T3–T4, regressing outcomes in the subsequent year on exposures and confounders measured in the earlier year. We used SAS version 9.4 (SAS Institute Inc., Cary, NC, USA) and present results as odds ratios (ORs) with 95% CIs, and considered a two-sided *P*-values <0.05 as statistically significant.

Missing data

Approximately 13% of cases at T1–T2, 26% at T2–T3 and 28% at T3–T4, had missing data for one or more variables. For any single variable, the highest proportion of missing data was 14% (Supplementary tables S1–S6). Missing data may lead to bias and substantial loss of precision and power of the estimate of association.[28] As sensitivity analyses, to evaluate the robustness of estimates, we performed multiple imputation to create 10 datasets using the chained regression equations method[28,29] under two assumptions: that values were missing at random (MAR), and that they were missing not at random (MNAR), allowing the possibility that those with missing outcome data were more or less likely to be hazardous drinkers. To model MNAR, we performed a tipping-point analysis where the outcome probability among those with missing data was varied (a shift value) to determine if there was a point at which any significant association became non-significant.[30]

We performed crude and adjusted mixed modelling using the same covariates for each of the 10 imputed datasets and pooled results across imputed sets using Rubin's rules.[31] Under the MNAR assumption, we only present the adjusted modelling if we found a significant association in the complete case analysis.

Results

Table 2 shows for complete case data that, based on their AUDIT-C scores, one-third of responding parents were hazardous drinkers, and that proportion decreased slightly over time. The proportion of parents' partners who were hazardous drinkers (usually men) was nearly twice as high and also decreased slightly over time, while the proportion of adolescents who were hazardous drinkers increased 3-fold from age 14–16 years.

Both the crude and adjusted analyses showed that children at ages 14 and 15, whose parents or parents' partners were hazardous drinkers, were at higher risk of becoming hazardous drinkers at subsequent ages. We did not find a significant association between parents' and children's hazardous drinking in early adolescence (at age 13). The magnitude of the associations changed little after adjusting for hypothesized socio-demographic and family-level confounders (table 3).

Sensitivity analyses

Under a MAR assumption, regression modelling produced similar effect sizes and *P*-values to those obtained from the complete case analysis (table 3). From T2–T3 and T3–T4, under no MNAR assumptions (adolescent hazardous drinking rate 0–100% in the missing participants) did the association between parents' or their partners' hazardous drinking, and adolescent hazardous drinking, become non-significant (Supplementary table S7). Thus, sensitivity analyses confirmed complete case findings.

Discussion

After adjustment for socio-demographic and family factors, hazardous drinking among parents or their partners was associated with adolescent hazardous drinking during mid-adolescence (adjusted ORs between 1.5 and 2). Adjustment for possible confounders attenuated the association only a little and sensitivity analyses confirmed the results were not unduly influenced by assumptions regarding the missing data.

We collected four years of longitudinal data with high retention, from a large cohort consisting of dyads of an adolescent and one of his or her parents, and estimated the associations of interest prospectively to rule out reverse causality. We ascertained parent characteristics from both parent and adolescent reports, reducing the risk of measurement error reflecting participant beliefs about the associations of interest. We developed theory-driven models on the basis that a 'kitchen sink' approach to covariate inclusion does not provide better adjustment for confounding and can introduce bias.[12]

Non-random selection into the cohort constrains generalizability, however, the cohort was broadly representative socio-demographically of the Australian population of similar age. Considering heterogeneity in the cohort with regard to the exposures of interest, the associations tested may reasonably be generalized to populations where similar drinking cultures exist.

Mode of administration (paper-based vs. online) can influence how participants respond to questions.[32] A 2007 meta-analytic review reported that a mixed mode design may reduce bias by providing participants with the opportunity to select their preferred mode.[33] In our study, to minimize information bias (especially that arising from children's reports where parents might be present), we sent separate baseline and follow-up questionnaires, encouraging parents and students to complete them independently of each other, either online or using the paper form and returning it by mail.[15]

Self-report of alcohol use may be biased for the usual reasons that respondents under-report stigmatized behaviour[34] or recall their consumption inaccurately.[35] We sought to reduce these biases by using a simple, validated outcome measure (AUDIT-C), administered confidentially. We asked parents and adolescents to complete questionnaires individually, in private, and to return them in sealed envelopes. The

Table 2 Parents', their partners' and children's drinking rates at each time period; *n* (%)

Dataset comparison	Complete case			Multiple imputation (under MAR assumption)		
	Parent is a hazardous drinker (AUDIT-C ≥ 5)	Parent's partner is a hazardous drinker (AUDIT-C ≥ 5)	Adolescent is a hazardous drinker (AUDIT-C ≥ 3)	Parent is a hazardous drinker (AUDIT-C ≥ 5)	Parent's partner is a hazardous drinker (AUDIT-C ≥ 5)	Adolescent is a hazardous drinker (AUDIT-C ≥ 3)
T1 parent/partner, T2 adolescent	698 (37%)	–[a]	111 (6.2%)	37%	–[a]	6.4%
T2 parent/partner, T3 adolescent	606 (34%)	752 (48%)	218 (13%)	34%	48%	13%
T3 parent/partner, T4 adolescent	485 (28%)	641 (43%)	320 (20%)	28%	43%	20%

a: Data not collected at T1.

Table 3 Prospective associations of parent hazardous drinking and potential confounders with adolescent hazardous drinking

		Adolescent hazardous drinking(C)							
		Complete case				Multiple imputation (under MAR assumption)			
Dataset comparison	Predictor	Crude		Adjusted		Crude		Adjusted	
	Parent hazardous drinking status(P)	Odds ratio (95% CI)	*P*-value	Odds ratio (95% CI)	*P*-value	Odds ratio (95% CI)	*P*-value	Odds ratio (95% CI)	*P*-value
T1/2	Parent a hazardous drinker[a]	1.11 (0.67–1.85)	0.689	1.07 (0.65–1.74)	0.799	1.11 (0.68–1.81)	0.684	1.13 (0.69–1.85)	0.619
T2/3	Parent a hazardous drinker[a]	1.61 (1.16–2.24)	0.005	1.53 (1.05–2.22)	0.026	1.68 (1.21–2.34)	0.002	1.60 (1.13–2.24)	0.007
	Partner a hazardous drinker[b]	2.07 (1.46–2.93)	<0.001	2.02 (1.38–2.96)	<0.001	1.86 (1.39–2.50)	<0.001	1.86 (1.38–2.50)	<0.001
T3/4	Parent a hazardous drinker[a]	1.99 (1.53–2.59)	<0.001	1.87 (1.42–2.46)	<0.001	2.03 (1.54–2.67)	<0.001	2.00 (1.51–2.64)	<0.001
	Partner a hazardous drinker[b]	2.01 (1.52–2.68)	<0.001	1.96 (1.44–2.68)	<0.001	1.99 (1.53–2.60)	<0.001	1.94 (1.48–2.55)	<0.001

(P), Parent report; (C), Child report.

a: Adjusted for parents' socio-demographic characteristics (i.e. education, income, employment and religiosity), and family factors (i.e. household composition, family history of heavy drinking, family relationship and conflict) (Supplementary figure S1).

b: Adjusted for household context, family history of heavy drinking, family relationship and conflict (Supplementary figure S2).

responding parents reported on their partners' alcohol use, and we do not know of evidence bearing on the likely accuracy of such reports.

As in the studies[36–39] identified by Rossow *et al.*,[12] we found that parents' and their partners' hazardous drinking predicted hazardous drinking in their mid-adolescent children. That the association did not remain statistically significant in early adolescence may be in part due to the small number of adolescents who reported drinking hazardously at that age, but the point estimate was consistent with increased risk associated with parent hazardous drinking. This is congruent with the finding that Finnish parents' drinking was positively associated with adolescent hazardous drinking at age 14.[38] Mares *et al.*[37] found that paternal, but not maternal, hazardous drinking predicted adolescent hazardous drinking, which is inconsistent with our findings of increased risk associated with hazardous drinking of both parents (usually mothers) and their partners (usually men).

None of the studies Rossow *et al.*[36–39] identified accounted for potential confounders of the estimated association.[12] We adjusted for a range of potential socio-demographic and family functioning variables in multivariable models, finding no evidence of confounding.

We cannot dismiss the possibility of residual confounding due to incomplete adjustment for genetic risk factors (e.g. ref.[8]) because we only measured family history by parent self-report. However, genetic effects are unlikely to account completely for the strong associations seen here, because they are at least partially matched, given that each parent and their offspring share 50% of their DNA.

Error in the measurement of confounders can bias inferences regarding the influence of parent hazardous drinking on adolescent hazardous drinking. In this study, we assumed the relationship is confounded by variables measured at the same time as parents' drinking status; however, it may be more complicated and involve covariate history leading up to that point.

Various behaviours, e.g. parental alcohol rules, parental supply of alcohol and parental monitoring, may play a role in transmitting alcohol-related behaviours from parent to child. For example, parental alcohol rules may mediate the association.[40] Rossow *et al.*[12] reported that three studies tested hypothetical mediating mechanisms and concluded that the association was partly mediated by either parental monitoring and discipline,[38] and/or by alcohol-specific communication,[37] and not by poor inhibitory control in children.[39]

In recent times, the research on alcohol's 'harm to others' has advanced considerably,[41] (Supplementary file: Additional references) including investigation of harms to children from parental alcohol use. In our study, we found that children aged 14 and 15 whose parent or parent's partner were hazardous drinkers, were at higher risk of being hazardous drinkers at subsequent ages. Thus, within this 'harm to others' framework, strategies to reduce the risk of developing hazardous drinking in children may include parent-targeted programmes and general population strategies that encourage parents to reduce their own alcohol consumption, such as increasing the price of alcohol, and reducing its availability and promotion[42] (Supplementary file: Additional references).

Supplementary data

Supplementary data are available at *EURPUB* online.

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Conflicts of interest: None declared.

Key points

- How parents' drinking influences their children's drinking remains unclear.
- We examined confounding by socio-demographic and family factors.
- Parent hazardous drinking predicted mid-adolescent hazardous drinking.
- Parents should be encouraged to reduce their own alcohol consumption.
- The findings support public policy to encourage parents with hazardous drinking to reduce their consumption.

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