

Association of parental supply of alcohol with adolescent drinking, alcohol-related harms, and alcohol use disorder symptoms: a prospective cohort study

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Summary

Background Some parents supply alcohol to their children, reportedly to reduce harm, yet longitudinal research on risks associated with such supply is compromised by short periods of observation and potential confounding. We aimed to investigate associations between parental supply and supply from other (non-parental) sources, with subsequent drinking outcomes over a 6-year period of adolescence, adjusting for child, parent, family, and peer variables.

Methods We did this prospective cohort study using data from the Australian Parental Supply of Alcohol Longitudinal Study cohort of adolescents. Children in grade 7 (mean age 12 years), and their parents, were recruited between 2010 and 2011 from secondary schools in Sydney, Perth, and Hobart, Australia, and were surveyed annually between 2010 and 2016. We examined the association of exposure to parental supply and other sources of alcohol in 1 year with five outcomes in the subsequent year: binge drinking (more than four standard drinks on a drinking occasion); alcohol-related harms; and symptoms of alcohol abuse (as defined by Diagnostic and Statistical Manual of Mental Disorders, 4th edition [DSM-IV]), alcohol dependence, and alcohol use disorder (as defined by DSM-5). This trial is registered with ClinicalTrials.gov, number NCT02280551.

Findings Between September, 2010, and June, 2011, we recruited 1927 eligible parents and adolescents (mean age 12.9 years [SD 0.52]). Participants were followed up until 2016, during which time binge drinking and experience of alcohol-related harms increased. Adolescents who were supplied alcohol only by parents had higher odds of subsequent binge consumption (odds ratio [OR] 2.58, 95% CI 1.96–3.41; $p < 0.0001$), alcohol-related harm (2.53, 1.99–3.24; $p < 0.0001$), and symptoms of alcohol use disorder (2.51, 1.46–4.29; $p = 0.0008$) than did those reporting no supply. Parental supply of alcohol was not significantly associated with the odds of reporting symptoms of either alcohol abuse or dependence, compared with no supply from any source. Supply from other sources was associated with significant risks of all adverse outcomes, compared with no supply, with an even greater increased risk of adverse outcomes.

Interpretation Providing alcohol to children is associated with alcohol-related harms. There is no evidence to support the view that parental supply protects from adverse drinking outcomes by providing alcohol to their child. Parents should be advised that this practice is associated with risk, both directly and indirectly through increased access to alcohol from other sources.

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Introduction

Alcohol use is an important public health issue associated with increased disease burden, and is of special concern in late adolescence and young adulthood, when alcohol use disorders have their peak age of onset.^{1–3} In many countries (such as the UK), parents are an important provider of alcohol to adolescents before they reach the legal age of purchase.⁴ Parents reportedly provide alcohol to mitigate harm,⁵ but evidence about the associated risks of such parental provision is absent—partly because of the dearth of any prospective studies spanning more than a 2–3-year period, and substantially because of the failure to adequately adjust for confounders in the handful of available cohort studies.⁶

Our previous Australian adolescent cohort study⁷ found that between ages 12 and 15 years, parental supply of alcohol was associated with increased risk of consuming standard drinks, but not of binge drinking.⁷ Although there was no protective effect associated with early parental supply, parentally supplied children did consume fewer alcohol beverages on a typical drinking occasion than did children who obtained their alcohol from other, non-parental sources. Adolescents who sourced their alcohol from non-parental sources (eg, peers, other relatives, themselves) had greater odds both of drinking standard drinks and of binge drinking. No longitudinal research has examined the development

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Research in context

Evidence before this study

We did a systematic review, searching eight electronic databases on Sept 10, 2016 (Medline, MEDLINE In-Process and Other Non-Indexed Citations, EMBASE, PsycINFO, CINAHL, Scopus, Dissertations and Theses, and Cochrane Library), with the search terms “parental provision”, “social hosting”, “parental source of alcohol”, “youth”, “student”, “teenage”, “underage”, “minor”, “risky drinking”, “excessive drinking”, and “binge drinking”. Inclusion criteria were that studies must be peer-reviewed, prospective, longitudinal studies, and analyse parental supply on the basis of reports from adolescent, parent, or both, and were from any publication year. We found seven cohort studies of parental supply of alcohol; the results of these showed that parental supply of alcohol was associated with subsequent risky drinking (pooled estimate odds ratio 2.00, 95% CI 1.72–2.32), but there was substantial risk of confounding bias and publication bias (assessed with the Newcastle–Ottawa scale). In all studies, measurement of exposure was problematic, follow-up times were short, and confounding was poorly controlled. Our cohort study has previously shown that, between age 12–15 years, parental supply of alcohol was associated with increased risk of consuming standard drinks (a standard drink being equal to 10 g of alcohol), but not of binge drinking. Parentally supplied children consumed fewer alcohol beverages on a typical drinking occasion than those who obtained their alcohol from non-parental sources. No study has reported on prospective associations between parental and

other sources of supply of alcohol and subsequent adolescent drinking outcomes, harms, and symptoms of abuse, dependence, or alcohol use disorders, taking into account important potential confounders of any observed association.

Added value of this study

To the best of our knowledge, this is the first study to examine, over a long period of time, prospective associations between both parental supply of alcohol and supply from other sources, and subsequent adolescent drinking outcomes, and to adjust for known covariates. We found a developing pattern of harm associated with parental supply, such that by the sixth follow-up (mean age 17.8 years), parental supply during previous years was associated with binge drinking, alcohol-related harms, and alcohol use disorder symptoms. The findings also showed that parental supply of alcohol not only risks adverse outcomes itself, it also risks increasing supply from other non-parental sources. Non-parental supply was associated with an even greater increased risk of adverse outcomes.

Implications of all the available evidence

Parental provision of alcohol to adolescents is associated with subsequent binge drinking, alcohol-related harm, alcohol use disorder symptoms, and increased access to alcohol from non-parental sources, and these associations persist after controlling for confounding variables. These findings have importance for parents, policy makers, and clinicians, and highlight parents as an important target for prevention.

of alcohol use disorder symptoms associated with parental supply. In view of the associations between supply of alcohol and adverse drinking behaviours in our cohort to date, it is important to examine whether these patterns continue (possibly more moderated drinking associated with parental supply compared with more excessive drinking associated with accessing alcohol from non-parental sources), and to examine risk of alcohol-related harm and alcohol use disorders symptoms.

We aimed to study parental and non-parental supply of alcohol and adjusted associations with five primary outcome variables in adolescents: binge drinking, alcohol-related harms, and the experience of symptoms of alcohol abuse, alcohol dependence, and alcohol use disorder.

Methods

Study design and participants

Between September, 2010, and June, 2011, a cohort of 1927 adolescents, and their parents, were recruited from grade 7 classes in 24 private independent (49%), six Catholic (12%), and 19 government (39%) secondary schools in Sydney, Perth, and Hobart. A detailed description of the recruitment methods, cohort characteristics, and representativeness is provided in the appendix (pp 4–8). The parents of grade 7 adolescents had to provide signed informed consent to be eligible.^{8,9} The cohort was similar to

the Australian population on important demographic measures, parental alcohol use, and child alcohol use.^{9–12} At wave 1 (grade 7 of secondary school), adolescents were a mean age of 12.9 years (SD 0.52), and similar to the Australian population in terms of sex distribution, household composition, racial background, and parental education. Groups of lower socioeconomic status are somewhat under-represented in the cohort; the predominance of students from non-government schools is likely to have biased the cohort towards higher levels of advantage compared with the general population.^{9–12}

Ethics approval was given by UNSW Sydney, and Universities of Tasmania, Newcastle, Queensland, and Curtin University. We obtained signed parental consent for each adolescent participant.

Procedures

Adolescents and parents completed paper or online questionnaires independently of each other to minimise reporting biases; adolescent self-report on alcohol behaviours has been shown to be reliable and valid (appendix pp 9–24, 85–99).^{13,14}

Outcomes

There were five variables, based on adolescent report of: binge drinking; experience of alcohol-related harms; and

See Online for appendix

experience of symptoms of alcohol abuse, alcohol dependence, and alcohol use disorder.

We defined binge drinking as consumption of more than four standard drinks (a standard drink being equal to 10 g of alcohol)¹⁵ on any single occasion in the past year, which was coded into a binary variable (no or yes). This definition is in line with Australian guidelines for adults, which recommend that healthy adults drink no more than four standard drinks on a single occasion to reduce the risk of alcohol-related injury.¹⁶

Adolescents were also asked about the frequency of experiencing a range of alcohol-related harms, using a scale that included 17 alcohol-related harms.¹⁷ A binary variable (no or yes) was coded, indicating whether any of the harms had been experienced. Two additional count outcomes were derived: number of different harms experienced (possible range 0–17), and total number of harms experienced, which was constructed by summing the six frequency response categories for each item (possible range 0–85; appendix pp 9–10).

We assessed symptoms of alcohol abuse from wave 2 using the alcohol abuse items from the Diagnostic Interview Schedule for Children-IV (DISC-IV).¹⁸ A binary variable (no or yes) was coded, indicating whether any of the abuse symptom items had been experienced or not in the past 12 months, corresponding to the Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM-IV) criteria (appendix p 25). We constructed an additional count outcome by summing the number of abuse symptoms experienced (possible range 0–4).

We assessed symptoms of alcohol dependence from wave 5 using the alcohol dependence items from DISC-IV.¹⁸ A binary variable (no or yes) was coded, indicating whether three or more of the dependence symptom items had been experienced in the past 12 months, corresponding to DSM-IV criteria (appendix p 26). We constructed an additional count outcome by summing the number of dependence symptoms experienced (possible range 0–7).

Finally, we assessed symptoms of an alcohol use disorder from wave 5 using the alcohol dependence items from DISC-IV,¹⁸ including an International Classification of Diseases item assessing craving, which corresponded with DSM-5 criteria (appendix p 27). A categorical variable was coded on the basis of number of alcohol use disorder symptoms experienced in the past year (no alcohol use disorder [none or one symptom], mild [2–3 symptoms], moderate [4–5 symptoms], or severe [≥ 6 symptoms]).

Adolescents were asked about who supplied them alcohol (sips or standard drinks) in the past 12 months, including mother, father, other adults, friends, siblings, and self-supply. From this, a dichotomous exposure variable—parental supply—was coded, indicating participants who had received alcohol from parents and those who had not. Adolescent report of parental supply was used because adolescent measures have been shown to be more predictive of adolescent behaviours than have parent measures,¹⁹ and adolescent self-report correlated

well with parental report of supply (intraclass correlation 0.69–0.79; appendix p 29).

A further source of alcohol exposure—other supply—included alcohol supply from non-parental adults, friends, siblings, or self-supply, compared with adolescents reporting no supply from these sources. Parental supply and other supply were not mutually exclusive, and since supply could be derived from both sources, each source was controlled for separately in all analyses.

For the primary analysis, exposures were coded as a four-level variable: no supply of alcohol, parental supply only, other supply only, or both parental and other supply. In other analyses, parental supply and other supply were coded as separate variables. For a secondary planned dose–response analysis, the exposure variables were coded into two measures of the number of years in which supply occurred (0, 1, 2, 3, 4, or 5).

Covariates, which we chose on the basis of the literature on adolescent drinking, and measured at each annual wave, are described elsewhere.⁹ These include parental report of their own alcohol consumption (quantity and frequency), presence of familial alcohol problems, potential home access to alcohol without parental knowledge, parental religiosity, family conflict or positive relations, the presence of older siblings in the home, socioeconomic status of area of residence, parental employment and income, and country of birth. Children reported on the supply of alcohol from parental and from other sources; parental responsiveness, demandingness, or consistency; parental use of alcohol-specific rules; parental monitoring of the child's activities; whether they were living in a one-parent or two-parent household; child sex and age; availability of money to purchase alcohol; child tobacco use; child externalising behaviour; child anxiety and depression symptoms; and their peer's substance use and disapproval of alcohol and tobacco use (appendix pp 13–19).

Statistical analysis

We included data from 6 years (waves 1–6) in this study. We planned primary analyses a priori, and did analyses using random intercept mixed-effects (controlling for within-respondent clustering or correlation over time), to examine the unadjusted and adjusted associations between the exposures and five main outcomes. We also analysed these five outcomes using logistic regression, with results presented as odds ratios (ORs) and CIs. We analysed severity of alcohol use disorder (mild, moderate, severe) and binge drinking (none, 5–10, ≥ 11 drinks) using multinomial logistic regression with results presented as relative risk ratios.

We analysed the four secondary count outcomes (reported number of different harms, total number of harms, number of abuse symptoms, and number of dependence symptoms) using a priori planned negative binomial regression, with results presented as incidence rate ratios (IRRs).

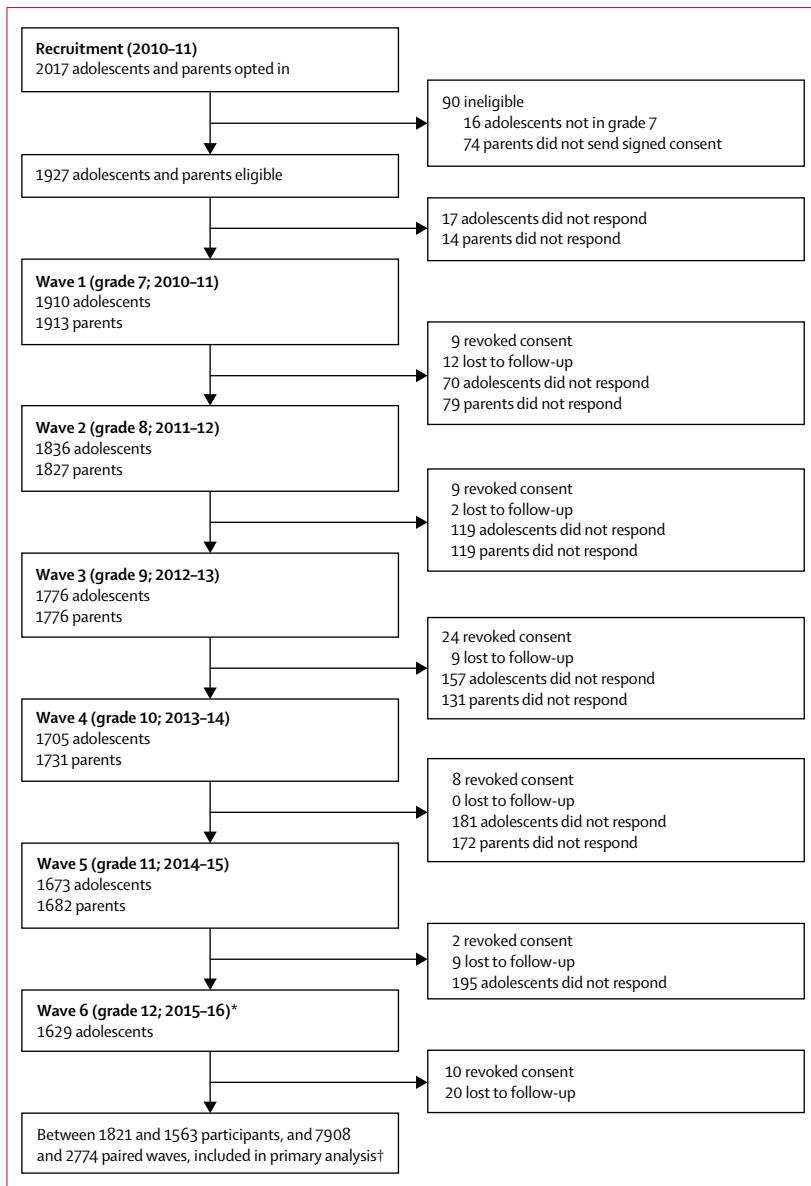


Figure: Study profile

*Parents were not assessed in wave six. †Participants did not have to respond at all waves to be included in the analysis and those not responding at a particular wave were still contacted at subsequent waves unless they actively withdrew from the study or were lost to follow-up because they had no valid contact details; see table 2.

We analysed data from six annual waves, with supply (parental only, other only, and both) compared with no supply from each year, examined against the outcomes from the following year. The analysis thus included five paired time periods (period 1 [waves 1–2], period 2 [waves 2–3], period 3 [waves 3–4], period 4 [waves 4–5], and period 5 [waves 5–6]). Variables were included in adjusted analyses if they showed unadjusted significance at an α of 0.05.

To test for a dose–response relationship, we did planned secondary analyses examining the association between the number of waves of parental or other

supply (between 0 and 5 years of supply) and harms reported in wave 6, using fixed-effect logistic or negative binomial regression, and the same five outcomes as the primary analyses and the four secondary count variables, using multiple imputation to reduce bias due to missing data (appendix pp 33–35). We also did random intercept analyses of the frequency of supply within each wave on the five primary outcomes. We did post-hoc sensitivity analyses of associations with 11 individual symptoms of alcohol abuse, dependence, or use disorders (in light of the rates of endorsement of alcohol use disorder symptoms). Finally, in another post-hoc analysis, we examined the association between parental supply and subsequent other supply using random intercept models. We did all analyses in Stata (version 14.1), using a decision-wise error rate of $p < 0.05$ for planned analyses, reduced to $p < 0.01$ for post-hoc analyses.²⁰

We used data from the Australian Parental Supply of Alcohol Longitudinal Study (APSALS) cohort of adolescents, which is registered with ClinicalTrials.gov (NCT02280551).

Role of the funding source

The funders of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report. RPM, PJC, AA, and MW had access to the raw data. RPM had full access to all the data and had final responsibility for the decision to submit for publication.

Results

The study profile is shown in the figure. The mean age of adolescent participants was 12.9 years (SD 0.52) in wave 1, 13.9 years (0.54) in wave 2, 14.8 years (0.56) in wave 3, 15.8 years (0.55) in wave 4, 16.9 years (0.50) in wave 5, and 17.8 years (0.51) in wave 6. At wave 6, 1494 (92%) of 1629 participants were attending high school. All forms of parental supply of alcohol (ie, adolescents who had parental supply only or both parental and other supply) increased across the six waves of observation, with such supply reported by 291 (15%) of 1910 adolescents in wave 1, 472 (26%) of 1836 in wave 2, 486 (27%) of 1776 in wave 3, 583 (34%) of 1705 in wave 4, 729 (44%) of 1671 in wave 5, and 916 (57%) of 1618 respondents in wave 6 (2 of 1673 cases had missing data at wave 5, and 11 of 1629 cases had missing data on this measure at wave 6; appendix p 36). Parental, familial, child, and peer characteristics associated with parental supply are shown in the appendix (p 38), with information related to the context of supply (special occasion, dinners, parties, etc), and the frequency and typical quantity supplied by parents (p 37).

Binge drinking and experience of alcohol-related harms increased markedly over time (table 1). Whereas only 77 (4%) of 1910 adolescents reported experiencing alcohol-related harm at wave 1, by wave 6 this had increased to 978 (60%) of 1618 (table 1). Endorsement of symptoms of abuse (at least one DISC-IV symptom; appendix p 30) and

dependence (at least three DISC-IV symptoms; appendix p 31) were less common, with 120 (7%) of 1618 reporting alcohol abuse symptoms and 189 (12%) reporting alcohol dependence at wave 6; alcohol use disorder symptoms (at least two DISC-IV symptoms; appendix p 32) were reported by 353 (22%) adolescents at wave 6.

At wave 6, the rates of binge drinking, alcohol-related harm, and symptoms of abuse, dependence, and alcohol use disorder were higher among adolescents who reported both parental and other supply than parental or other supply individually (table 1). Adolescents could get alcohol from parents, other non-parental sources, or both, so these are coded as parental supply only, other supply only, or both sources of supply. Specifically, 632 (81%) of 784 adolescents who received alcohol both from their parents and from others reported that they had binge, 671 (86%) experienced alcohol-related harm, 102 (13%) reported alcohol abuse symptoms, 145 (18%) had at least three dependence symptoms, and 284 (36%) reported at least two alcohol use disorder symptoms (table 1). The most frequently reported alcohol use disorder symptoms in adolescents supplied with alcohol by their parents (n=916) were reported tolerance

(n=310 [34%]), drinking larger amounts or for longer than intended (n=283 [31%]), and spending time getting, consuming, or recovering from alcohol (n=159 [17%]; appendix p 28). The outcome measures were significantly inter-correlated; for example, binge drinking was moderately correlated with harms (0.66) and weakly correlated with abuse symptoms (0.23), and alcohol use disorder symptoms were strongly correlated with dependence symptoms (0.97; appendix p 39).

Adolescents who reported parental supply only had significantly higher odds of binge drinking (OR 2.58, 95% CI 1.96–3.41; $p < 0.0001$) in the subsequent wave, compared with those reporting no supply, a result consistent with higher levels of bingeing (appendix pp 46–48). Adolescents who reported other supply only had even greater odds of binge drinking (5.58, 4.27–7.29; $p < 0.0001$) compared with those reporting no supply. Those who reported both parental and other supply also had increased odds of binge drinking (4.81, 3.75–6.17; $p < 0.0001$), which was higher than for parental supply only, but was lower than would be expected if the effects of receiving parental supply only and other supply only were combined (table 2).

| | Wave 1 | Wave 2 | Wave 3 | Wave 4 | Wave 5 | Wave 6 |
|---|--------------|----------------|----------------|----------------|----------------|----------------|
| Binge drinking (>4 drinks on one occasion) | | | | | | |
| Parental supply only | 3/177 (2%) | 6/241 (2%) | 10/213 (5%) | 14/179 (8%) | 42/186 (23%) | 33/132 (25%) |
| Other supply only | 10/63 (16%) | 36/126 (29%) | 76/193 (39%) | 110/243 (45%) | 190/361 (53%) | 224/361 (62%) |
| Both sources of supply | 12/114 (10%) | 56/231 (24%) | 82/273 (30%) | 194/404 (48%) | 357/543 (66%) | 632/784 (81%) |
| Total | 25/1910 (1%) | 98/1836 (5%) | 168/1776 (9%) | 318/1705 (19%) | 589/1671 (36%) | 889/1618 (55%) |
| Any alcohol-related harms | | | | | | |
| Parental supply only | 16/177 (9%) | 25/241 (10%) | 14/213 (7%) | 22/179 (12%) | 47/186 (25%) | 46/132 (35%) |
| Other supply only | 24/63 (38%) | 70/126 (56%) | 101/193 (52%) | 137/243 (56%) | 253/361 (70%) | 261/361 (72%) |
| Both sources of supply | 37/114 (32%) | 104/231 (45%) | 115/273 (42%) | 237/404 (59%) | 404/543 (74%) | 671/784 (86%) |
| Total | 77/1910 (4%) | 199/1836 (11%) | 230/1776 (13%) | 396/1705 (23%) | 704/1671 (42%) | 978/1618 (60%) |
| Alcohol abuse symptoms (≥ 1)*† | | | | | | |
| Parental supply only | NA | 0 | 0 | 0 | 0 | 0 |
| Other supply only | NA | 10/126 (8%) | 13/193 (7%) | 19/243 (8%) | 18/361 (5%) | 18/361 (5%) |
| Both sources of supply | NA | 13/231 (6%) | 22/273 (8%) | 28/404 (7%) | 54/543 (10%) | 102/784 (13%) |
| Total | NA | 23/1836 (1%) | 35/1776 (2%) | 47/1705 (3%) | 72/1671 (4%) | 120/1618 (7%) |
| Alcohol dependence symptoms (≥ 3)‡ | | | | | | |
| Parental supply only | NA | NA | NA | NA | 0 | 0 |
| Other supply only | NA | NA | NA | NA | 30/361 (8%) | 44/361 (12%) |
| Both sources of supply | NA | NA | NA | NA | 70/543 (13%) | 145/784 (18%) |
| Total | NA | NA | NA | NA | 100/1671 (6%) | 189/1618 (12%) |
| Alcohol use disorder symptoms (≥ 2)‡ | | | | | | |
| Parental supply only | NA | NA | NA | NA | 5/186 (3%) | 2/132 (1%) |
| Other supply only | NA | NA | NA | NA | 55/361 (15%) | 67/361 (19%) |
| Both sources of supply | NA | NA | NA | NA | 132/543 (24%) | 284/784 (36%) |
| Total | NA | NA | NA | NA | 192/1671 (11%) | 353/1618 (22%) |

Data are n/N (%). Figures might differ from figure 1 because of missing data. If a participant was missing data on the parental supply or other supply variable, they were coded as missing because we were unable to confirm whether they also received alcohol from the missing source. NA=not asked. *Results for abuse symptoms do not consider diagnostic criterion B from Diagnostic and Statistical Manual of Mental Disorders, 4th edition—that dependence not be diagnosable—but when diagnostic criterion B is considered, percentages with abuse symptoms in wave 6 are 0.8% for participants who did not receive parental supply (n=3), 4.9% for those who did (n=45), and 3.2% for the total sample (n=52). †Abuse questions were not asked in wave 1. ‡Dependence and alcohol use disorder questions were not asked in waves 1–4.

Table 1: Participants reporting binge drinking, alcohol-related harms, abuse, dependence, or alcohol use disorder symptoms, by source of supply of alcohol, and of the total sample

| | Binge drinking | Any alcohol-related harms | ≥1 abuse symptom | ≥3 dependence symptoms* | ≥2 alcohol use disorder symptoms* |
|----------------------------------|-------------------------------------|-----------------------------------|----------------------------------|----------------------------------|-----------------------------------|
| Current wave supply | | | | | |
| No supply | 1 (ref) | 1 (ref) | 1 (ref) | 1 (ref) | 1 (ref) |
| Parental supply only | 2.58 (1.96–3.41); p<0.0001 | 2.53 (1.99–3.24); p<0.0001 | 0.50 (0.17–1.47); p=0.205 | 1.67 (0.71–3.93); p=0.241 | 2.51 (1.46–4.29); p=0.0008 |
| Other supply only | 5.58 (4.27–7.29); p<0.0001 | 3.46 (2.68–4.47); p<0.0001 | 5.30 (3.14–8.94); p<0.0001 | 3.01 (1.58–5.72); p=0.0008 | 3.81 (2.45–5.91); p<0.0001 |
| Both parental and other supply | 4.81 (3.75–6.17); p<0.0001 | 4.25 (3.38–5.36); p<0.0001 | 5.21 (3.15–8.59); p<0.0001 | 5.04 (2.70–9.42); p<0.0001 | 5.79 (3.78–8.86); p<0.0001 |
| Time period | | | | | |
| 1 (wave 1 to wave 2) | 1 (ref) | 1 (ref) | 1 (ref) | NA | NA |
| 2 (wave 2 to wave 3) | 1.10 (0.78–1.56); p=0.586 | 0.59 (0.45–0.78); p=0.0002 | 0.64 (0.34–1.21); p=0.170 | NA | NA |
| 3 (wave 3 to wave 4) | 2.91 (2.06–4.10); p<0.0001 | 1.41 (1.08–1.84); p=0.011 | 0.59 (0.31–1.13); p=0.111 | NA | NA |
| 4 (wave 4 to wave 5) | 8.16 (5.63–11.81); p<0.0001 | 3.71 (2.78–4.95); p<0.0001 | 0.66 (0.35–1.24); p=0.197 | 1 (ref) | 1 (ref) |
| 5 (wave 5 to wave 6) | 16.93 (11.09–25.86); p<0.0001 | 8.35 (5.92–11.78); p<0.0001 | 0.85 (0.44–1.65); p=0.627 | 1.61 (1.08–2.39); p=0.018 | 1.58 (1.19–2.11); p=0.0015 |
| Cases included (n) | 1821 | 1819 | 1819 | 1563 | 1563 |
| Paired time periods included (n) | 7908 | 7813 | 7745 | 2774 | 2774 |

Data are adjusted odds ratio (95% CI); p value. Results are from random intercept mixed-effects logistic regression models, which we adjusted for other covariates (appendix pp 40–45). Data are included as paired waves, with covariates from one wave and outcome from subsequent wave. NA=not asked. *Dependence and alcohol use disorder symptoms were first assessed in wave 5, so only two time periods (waves 4 and 5) were included for analysis.

Table 2: Associations of source of supply at current wave and time with binge-drinking, harms, and symptoms of abuse, dependence, and alcohol use disorder at the subsequent wave

Similarly, adolescents reporting parental supply only or other supply only had significantly higher odds of experiencing alcohol-related harms than did those reporting no supply (parental supply OR 2.53, 95% CI 1.96–3.34, p<0.0001; other supply 3.46, 2.68–4.47; p<0.0001). Those reporting both parental and other supply also had increased odds of alcohol-related harms (4.25, 3.38–5.36; p<0.0001), however, as with binge drinking, although the odds ratio was higher than parental supply only, it was lower than would be expected if the effects of parental supply only and other supply only were combined (table 2).

Those reporting parental supply only had significantly increased odds of DSM-5 alcohol use disorder compared with no supply (OR 2.51, 95% CI 1.46–4.29; p=0.0008), as did those reporting other supply only (3.81, 2.45–5.91; p<0.0001), and those reporting both parental and other supply (5.79, 3.78–8.86; p<0.0001). Parental supply only was not significantly associated with the odds of reporting symptoms of either DSM-IV outcome (abuse or dependence), compared with no supply from any source.

However, other supply only was associated with much higher odds of both abuse symptoms (5.30, 3.14–8.94; p<0.0001) and dependence symptoms (3.01, 1.58–5.72; p=0.0008) compared with no supply from any source. Adolescents who reported both parental and other supply also had higher odds of alcohol abuse symptoms (5.21, 3.15–8.59; p<0.0001) and alcohol dependence symptoms (5.04, 2.70–9.42; p<0.0001; table 2).

There were significant random effects for all models used to examine the associations between supply and outcomes: binge drinking (intercept SD 1.33, p<0.0001), reporting any harms (1.21, p<0.0001), reporting abuse symptoms (1.07, p<0.0001), reporting dependence symptoms (1.58, p<0.0001), and reporting alcohol use disorder symptoms (0.96, p=0.0009).

Similar to the results for our primary analysis, when we examined the number of harms and symptoms experienced by adolescents, parental supply of alcohol was associated with higher rates of harms (IRR 1.42, 95% CI 1.27–1.58), total number of harms (1.49, 1.30–1.70), and number of dependence symptoms (1.56, 1.31–1.87)—but not the number of abuse symptoms (0.90, 0.66–1.24)—than was no parental supply (appendix pp 63–64). Abuse symptoms were infrequently endorsed.

Analysis of the number of waves of parental supply showed evidence of a dose–response relationship between number of waves of parental supply and binge drinking and report of harms, but not the DSM outcomes (appendix pp 65–82). There was also evidence of a dose–response relationship with supply of alcohol from other sources, particularly for alcohol abuse, with the ORs increasing as the number of waves of other supply increased (appendix pp 65–55). Analysis of frequency of supply within each year showed significant associations between frequency of parental supply and all outcomes except abuse symptoms, which is consistent with the results from our primary analysis (appendix pp 65–82).

Adjusted analysis of the relationship between parental supply and other supply, to explore whether withholding parental supply leads to higher rates of later other supply, showed that parental supply was associated with double the odds of other supply in the subsequent wave (OR 2.05 95% CI 1.69–2.48), compared with no parental supply. This was true both of supply of sips (2.03, 1.66–2.49) and standard drinks (2.67, 1.81–3.95; appendix pp 83–84).

Consistent with the primary analysis, analyses of parental supply and self-supply showed a significant interaction effect, as did analyses of parental and peer supply (appendix pp 51–62). Analyses of potential interaction effects between parental supply and sex or wave were not significant for any outcome, suggesting the effect was consistent by sex and over time.

Discussion

To the best of our knowledge, this is the first study done over a long observation period to examine the prospective

associations between both parental supply of alcohol and supply from other sources, and subsequent adolescent drinking outcomes, harms, and self-reported symptoms of alcohol abuse, dependence, and alcohol use disorder, adjusting for known covariates. In this six-wave longitudinal cohort study, we observed a pattern of adverse alcohol-related outcomes in adolescents aged 12·9 years to 17·8 years, associated with parental supply. Parental supply only was associated with a higher incidence of binge drinking, alcohol-related harm(s), and symptoms of alcohol use disorder, compared with no supply. These results are by contrast with our results assessing alcohol use in adolescents aged 15·9 years, for whom parental supply was associated with consumption of alcohol, but not binge drinking (a previous null result due to insufficient power).⁷ Over time, alcohol consumption has significantly increased, and adolescents are at a higher risk of adverse drinking outcomes, reflecting the cohort having entered a phase of life when binge drinking is known to increase.²¹

Obtaining alcohol from other sources (ie, other supply only) was associated with increased risk of all primary outcomes, including binge consumption, alcohol-related harm(s), and symptoms of abuse, dependence and alcohol use disorder, compared with no supply. Additionally, there was evidence of a dose–response relationship between other supply and the five primary outcomes, which is consistent with earlier age of initiation being associated with later bingeing (appendix pp 65–82).²²

Although other supply was associated with more problems than was parental supply, our study shows that there is no rationale for parents to give alcohol to adolescents younger than the legal purchase age. Parental supply only remains associated with adverse adolescent drinking outcomes, compared with no supply, and a combination of parental and other supply was not associated with lower odds of adverse outcomes than other supply only. Thus, there was no evidence to support the view that parental supply is protective for any of the adolescent drinking-related outcomes.

The child and parent characteristics predicting parental supply have not been well documented, but there are repeated observations that parents supply alcohol to protect their children from heavy drinking,^{5,23} and that lenient parental alcohol rules, and perceptions of peer alcohol use, predict parental supply.²⁴ Qualitative research shows that some parents might see providing alcohol as ensuring the child fits in with peers, and to ensure harm minimisation.²⁵ Although it could be tempting to infer that because other supply of alcohol is associated with higher risk of adverse outcomes, and therefore that parental supply should be preferred, the results of our analyses indicate that parental supply could directly increase risk of harms, while also increasing the likelihood of later supply by others. These results, taken together, reinforce the notion that alcohol consumption leads to harm, and this is true regardless of the source of

that alcohol—be it parents or others. In short, parental supply is not associated with any benefit (ie, reduction in drinking behaviours or harms). These results support the promotion of a precautionary approach to adolescent alcohol supply by parents; to reduce the risk of alcohol-related harm, parents should avoid supplying alcohol to children. Non-supply appears to be the safest option (obvious in some ways), if the parental and societal aim is to protect the health of adolescents and young adults.

Our study has some limitations. Our cohort is not a random sample from the population but a group that agreed to participate, so the results might not be generalisable at a population level. However, our cohort is similar to the Australian population (parents and children) for major demographic and alcohol consumption measures, though populations of low socioeconomic status are under-represented.⁹ The measures of symptoms of abuse, dependence, and alcohol use disorder were self-completed by cohort participants. However, the levels of reported symptoms of abuse and dependence in this cohort are very similar to the levels of alcohol use disorders found in community samples of young adults in Australia²⁶ and other high-income countries.^{27,28} The dependence symptoms of tolerance and use of alcohol in larger amounts or for longer periods than intended had the highest endorsement rates, which is consistent with previous research.²⁹ The current data should not be interpreted to mean that participants have an alcohol use disorder, but rather that there are emergent symptoms that could foreshadow alcohol use problems in the future.

Although we do not generalise the results to other cultures and settings, there remains no evidence from other countries that parental supply reduces risk. However, cultures and settings in which less consumption is normal among young people could show different results,³⁰ and might not show the increased risk seen in this study. Also, we could not account for child educational attainment or problems, as we could not gather nationally consistent measures on the participants. Finally, the analysis does not take into account the absolute amounts or the context of parental supply, but that was not our aim.

The 2016 *Lancet* Commission on adolescent health noted “Given that families and parents remain the most important figures in the lives of most adolescents, the paucity of rigorous research into family influences on adolescent health and wellbeing is a striking knowledge gap.”³ We agree; we found that parental provision of alcohol to their children is associated with subsequent binge drinking, alcohol related-harm(s), and symptoms of alcohol use disorder. There was no evidence of any benefit or protective effect, either directly (ie, no evidence that parental supply is associated with reduced risk of alcohol-related harms), or indirectly (ie, associated with lower risk of accessing alcohol from other sources). Parental supply is associated with increased risk of other supply, not the reverse.

While governments (especially in high-income countries) focus upon prevention through enforcement of legal age of purchase legislation, and school-based education, a major stakeholder with excellent scale and penetration goes largely unnoticed—parents. Parents, policy makers, and clinicians need to be made aware that parental provision of alcohol is associated with risk, not with protection, to reduce the extent of parental supply in high-income countries, and in low-middle-income countries that are increasingly embracing the consumption of alcoholic beverages.³¹

Contributors

RPM, LV, JN, KK, TS, DH, and MW conceived and developed the final methodology for this study. MW, AA, and RPM coordinated recruitment and baseline data collection with RB and NM. AA and MW did all data capture, data entry, data cleaning, and initial data analyses, with contributions from RPM, JN, KK, RB, DH, and PJC. PJC did the statistical analyses with input from RPM, MW, AA, JN, KK, TS, LD, and RB. PJC and RPM prepared the tables and figures, with input from all other authors. RPM led the writing of the paper, and wrote the Introduction. AA, PJC, and RPM wrote the Methods, and PJC wrote the Results section with RPM, while RPM wrote the Discussion with PJC. All authors contributed substantially to the paper. AA and RPM wrote the Research in Context.

Declaration of interests

We declare no competing interests.

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