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Alcohol and parenthood: An integrative analysis of the effects of transition to parenthood in three Australasian cohorts

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ABSTRACT

Aims: To determine the extent to which the transition to parenthood protects against heavy and problematic alcohol consumption in young men and women.
Design: Integrated participant-level data analysis from three population-based prospective Australasian cohort studies.
Setting: General community; participants from the Australian Temperament Study, the Christchurch Health and Development Study, and the Victorian Adolescent Health Cohort Study.
Measurements: Recent binge drinking, alcohol abuse/dependence and number of standard drinks consumed per occasion.
Findings: 4015 participants (2151 females; 54%) were assessed on four occasions between ages 21 and 35. Compared to women with children aged < 12 months, women who had not transitioned to parenthood were more likely to meet the criteria for alcohol abuse/dependence (fully adjusted risk ratio [RR] 3.5; 95% CI 1.5–7.9) and to report recent binge drinking (RR 3.0; 95% CI 2.1–4.3). The proportion of women meeting the criteria for alcohol abuse/dependence and/or binge drinking increased with the age of participants' youngest child, as did the mean number of standard drinks consumed on each occasion (1.8 if the youngest child was < 1 year of age vs. 3.6 for 5+ years of age). Associations between parenthood and male drinking behaviour were considerably weaker.
Conclusions: For most women in their twenties and thirties, parenting a child < 1 year of age was associated with reduced alcohol consumption. However, this protective effect diminished after 12 months with drinking levels close to pre-parenthood levels after five years. There was little change in male drinking with the transition to parenthood.

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1. Introduction

Heavy and problematic alcohol use generally peaks in the third decade of life in high-income countries (White et al., 2006, 2005; Bachman et al., 1997; Chen and Kandel, 1995; Kandel and Logan, 1984; Delker et al., 2016). It is one of the ten leading modifiable risk factors for disease burden across all country income groupings (Vos et al., 2016), as well as bringing additional risks of injury and social problems such as alcohol-related crime (Vergés et al., 2012). For males and females, the quantity and frequency of alcohol consumption increase in the late teenage years and early twenties (Kerr et al., 2009), before decreasing in the late twenties and early thirties (Johnstone et al., 1996; Sher and Gotham, 1999). This reduction often coincides with the transition to first parenthood (Tran et al., 2015a, b), contributing to the long-held belief that parenthood is responsible for this young adult '*maturing out*' in alcohol consumption (Bachman et al., 1997; Bailey et al., 2008). Yet previous research has reported inconsistent findings; some studies have reported reduced consumption in males and females (Bachman et al., 1997; Staff et al., 2010; Hajema and Knibbe, 1998; Labouvie, 1996; Shore, 1997), while others have found reductions in only young women in the period immediately before (Bailey et al., 2008) and after (Jaffee et al., 2001; Woodward et al., 2006; Christie-Mizell and Peralta, 2009) childbirth.

Other processes may also explain young adult shifts in alcohol use. Neurodevelopmental maturation over this period - with accompanying reductions in risk-taking behaviours and sensation-seeking - is one possibility (Steinberg et al., 2008; Casey, 2015; Tseng and O'Donnell, 2007). Alternatively, changes in drinking may follow other young adult transitions that are incompatible with heavy alcohol use, including marriage and entering the labour market (Vergés et al., 2012; Winick, 1962; Yamaguchi and Kandel, 1985; Leonard and Eiden, 2007; Bachman et al., 2002; Merline et al., 2004). Furthermore, recent changes in the timing of young adult role transitions - including parenthood (Patton et al., 2018) - may affect the extent to which heavy and problematic drinking becomes entrenched (Gilbert et al., 1999). The effects of parenthood on drinking patterns may also be reduced by the recent trend of having fewer children and with greater spacing between births (ABS. Births, 2016). These shifts in social development across young adulthood raise uncertainty about the relevance of earlier findings from studies in settings of higher and early fertility (Hajema and Knibbe, 1998; Labouvie, 1996).

There is potential for myriad serious adverse consequences for the offspring of people with problematic alcohol use patterns (Velleman and Templeton, 2007), resulting from both biological and social influences. These include the effects of alcohol on infants via antenatal exposure or breastfeeding (Nykjaer et al., 2014), insecure attachment and maternal bond (Eiden et al., 2009), neglectful parenting (Dube et al., 2001), and modelling unsafe alcohol use which is replicated by offspring at a later age (Dube et al., 2001). In light of this, it is important to examine the effects of parenthood on heavy and problematic alcohol use in young adults. To do so, we used data from three longitudinal Australasian cohort studies which measured alcohol use from adolescence into young adulthood: the Australian Temperament Study (ATP) (Edwards et al., 2013), the Christchurch Health and Development Study (CHDS) (Fergusson and Horwood, 2001) and the Victorian Adolescent Health Cohort Study (VAHCS) (Patton et al., 2007). We have, for the first time, explored the links between parenting stage (defined by the current age of the participant's youngest child) and alcohol, hypothesising that participants with younger children would report a greater reduction in heavy and problematic alcohol use compared to participants with no children and those with older children.

2. Method

2.1. Design and Participants

We integrated data from three prospective cohort studies in the Cannabis Cohorts Research Consortium (Silins et al., 2014, 2015; Horwood et al., 2012, 2010; Hutchinson et al., 2015). The ATP (Edwards et al., 2013) commenced in 1983 as a representative sample of 2443 infants (aged 4–8 months) and their parents across Victoria, Australia. The CHDS (Fergusson and Horwood, 2001) is a birth cohort study of 1265 children born in the urban region of Christchurch, New Zealand in 1977. The VAHCS (Patton et al., 2007) is based on a representative sample of 1943 mid-secondary adolescents resident in Victoria, Australia, who were born in 1976–1978. Further details about the cohorts are provided in Supplementary Table 1a.

2.2. Participant Age and Temporal Alignment

The ages of VAHCS and CHDS participants were similar during the adult waves. ATP participants were born four years later and were slightly younger than VAHCS and CHDS participants at their three adult assessments. Henceforth Waves 1–4 refer to the four adult assessments at the ages of approximately 21, 24, 30 and 35 years, respectively. The ATP did not contribute Wave 1 data.

2.3. Age of Youngest Child

Each study collected data on the age of the participant's youngest child at the time of each assessment (categorised as: not a parent, youngest child aged < 1 year, 1–4 years or 5+ years).

2.4. Alcohol Use

Alcohol abuse/dependence at each adult wave was assessed using the alcohol and substance abuse modules of the Composite International Diagnostic Interview (CIDI) (Robins et al., 1988) for all participants (CHDS), and for those who had consumed more than 11 standard drinks in the past 12 months (VAHCS). Alcohol abuse/dependence was not assessed in the ATP. Past week binge drinking (all cohorts) and the total dose of alcohol consumed on the last drinking occasion (VAHCS and CHDS) were also measured (see Supplementary Box 1 for further detail).

2.5. Background Covariates

Each cohort identified adolescent and family covariates associated with being a parent and alcohol consumption. This included adolescent problem drinking for all cohorts. Further detail is provided in Supplementary Table 1b.

2.6. Time-Varying Covariates

Adult factors measured by all cohorts were identified that a) varied across waves; b) were influential on both parenting and alcohol outcomes; and c) reflected the transition to adulthood. These were: living with a parent, being in a serious relationship, and having a major depressive or anxiety disorder. Further detail about the measurement of depression and anxiety is provided in Supplementary Table 1c.

2.7. Statistical Analysis

We summarised proportions (parenting, alcohol use/dependence, binge drinking) and means (number of drinks) by cohort, wave and participant sex. Crude exposure-outcome associations were examined by summarising alcohol use outcomes by age of youngest child for each cohort and participant sex. Sex-specific adjusted associations between parenting and alcohol use were obtained by fitting multivariable regression models for each outcome to the data, pooled across cohorts. These models included the age of a participant's youngest child, participant sex and their interaction as predictors, and were initially adjusted for wave (a proxy for participant age) and cohort (Model 1).

They were then additionally adjusted for background confounders (Model 2) and further for time-varying confounders (Model 3). Background confounders differed across cohorts, so adjustment for these was performed via including the estimated propensity score as predictor in the regression (Silins et al., 2014; Rosenbaum and Rubin, 1983) (see Supplementary Table 1b).

Each outcome model was fitted with generalised estimating equations, using an exchangeable correlation structure and robust standard errors to account for clustering due to repeated measures. For binary outcomes, log-link Poisson models were used to obtain risk ratio (RR) estimates, while number of standard drinks was modelled using a log-link negative binomial model to obtain incidence rate ratio (IRR) estimates. Evidence for an exposure-sex interaction was assessed using the Wald test (two-sided). Bayes factors were calculated to assess the strength of evidence where no associations between parenting stage and alcohol use outcomes were found (Beard et al., 2016), using the online Bayes Factor Calculator developed by (Dienes (2014)) and adapted in Javascript by (Singh (2018)).

The primary analyses were based on pooled samples of 2767 participants (alcohol abuse/dependence and number of drinks) and 4015 participants (binge drinking) with data on at least one background covariate and at least one wave of data on both exposure and outcome. We used multiple imputation to minimise potential bias due to missing data (see Supplementary Box 2 for further information). In supplementary analyses, we tested for between-cohort heterogeneity and changing effect of exposure by age of participant (wave) via Wald tests for three way interactions between exposure, sex and cohort and exposure, sex and participant age/ wave in separate, fully adjusted models in the multiply imputed datasets.

In sensitivity analyses, we compared our primary results using imputed data to those obtained using observed data. In the observed data, we also investigated the potential additional effect of spacing between children via further adjustment for having more than one child aged less than 3 years. All analyses were conducted using Stata v. 14·2 (StataCorp, 2013).

2.8. Ethics Approval

Ethics approvals were obtained from The Royal Children's Hospital Human Research Ethics Committee (REC) in Melbourne (VAHCS), the Australian Institute of Family Studies Human REC (ATP), and the Southern Regional Health and Disability REC in Christchurch (CHDS). Ethics approval to harmonise the data from these cohorts was granted by the University of New South Wales Human REC (HC11365). The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

3. Results

3.1. Parenting Status and Alcohol Outcomes

In each of the three cohorts and at each wave, a higher proportion of females reported having transitioned to parenthood (Table 1). Although most participants had become parents by age 35, a substantial minority (40.4% of females and 49.1% of males) had not. Alcohol use declined after the age of 25 years, observed for all alcohol outcomes and across most time points and cohorts. A higher proportion of males than females met the diagnostic criteria for alcohol abuse/dependence at each time point across cohorts, and more males also reported binge drinking. The mean number of standard drinks consumed during each drinking session was higher for males than females across cohorts and at all waves.

3.2. Alcohol Consumption as a Function of the Youngest Child's Age

In most categories, a higher proportion of males and females without children met the criteria for alcohol abuse/dependence and binge drinking than participants with a child (Table 2; for results by cohort, see Supplementary Tables 2a and b). Crude rates of alcohol abuse/dependence in this sample were higher in both males and females with older youngest children than in those with a child aged < 1 year.

The pooled estimate for alcohol abuse/dependence in women who were not parents was 11.1% (95%CI: 10.1–12.1), whilst for women who were parents estimates ranged from 2.6% (95%CI: 1.2–4.9) to 6.4% (95%CI: 4.3–9.1). A similar pattern was observed for binge drinking in females who were parents, but not in males who were parents (whose rates of weekly binge drinking did not appear to increase with child age). The pooled estimate for binge drinking in women who were not parents was 25.0% (95%CI: 23.8–26.1), whilst for women who were parents estimates ranged from 7.0% (95%CI: 4.9–9.5) to 15.5% (95%CI: 12.5–18.9). The mean number of standard drinks consumed per session was positively associated with age of the youngest child for males and females, with the mean number consumed by participants whose youngest child was aged 5+ years exactly matching the mean for non-parents in males (7.3) and almost matching the mean in females (3.6 vs 3.8) in this sample.

3.3. Risk Associations (Adjusted)

Table 3 shows the estimated associations between parenting and reported alcohol-related behaviours, adjusted first for cohort and age, then for background confounders, and then for time-varying confounders reflecting adult transitions. Preliminary analyses provided little indication of employment status being predictive of drinking behaviours in this sample, and this was not included in these models. For women, in the model adjusted for cohort and age (Model 1), there was consistent evidence across all outcomes of an association with age of youngest child. Women without children were consistently at highest risk of adverse alcohol consumption; those with a youngest child aged 5+ were at the next highest risk, and those with a youngest child aged 1–4 ha d only a moderate increase in risk compared to those with a child aged < 1 year. Preliminary analyses indicated that adolescent problem drinking was associated with a small increase in the likelihood of transitioning to parenthood by the age of 35, but adjustment for this and other background covariates (Model 2) made little difference to estimates. Additional adjustment for time-varying covariates (Model 3) had a modest impact on effect sizes: women with no children had more than a threefold higher risk of alcohol abuse/dependence (RR: 3.5; 95%CI 1.5–7.9) and of binge drinking (RR: 3.0; 95%CI 2.1–4.3), and they reported consuming twice as many drinks per occasion as women with a child aged < 1 year (IRR: 2.0; 95%CI 1.6–2.6). The effect sizes for males were much weaker and provided little indication of association after covariate adjustment. There was some evidence that the transition to becoming a parent was not associated with any change in male binge drinking (Bayes factor: 0.06) or number of drinks consumed (Bayes factor: 0.19) (Supplementary Table 3).

Fig. 1 shows a consistent pattern in which the adjusted rate/mean of each outcome was highest for women who were not parents, lowest for those whose youngest child aged < 1 year, and increasing thereafter with age of youngest child. For males, there was no predictable pattern of alcohol outcomes by parenting stage.

3.4. Alcohol Consumption and Youngest Child's Age Across Cohorts and Waves

The pattern of results was similar across cohorts (Supplementary Tables 2a, 2b and 4). Tests of between-cohort heterogeneity (Supplementary Table 4) showed an apparent difference in the effect

Table 1
Rates of parenting and alcohol outcomes in three cohorts, by participant gender and wave: imputed data, all participants with at least one background covariate and at least one wave of exposure and outcome.

	Females								Males							
	21 years		24 years		30 years		35 years		21 years		24 years		30 years		35 years	
	%	(n*/N)	%	(n*/N)	%	(n*/N)	%	(n*/N)	%	(n*/N)	%	(n*/N)	%	(n*/N)	%	(n*/N)
Parent Y/N																
VAHCS	4	(34/925)	10	(95/925)	28	(259/925)	65	(603/925)	2	(16/817)	7	(55/817)	20	(162/817)	52	(424/817)
CHDS	14	(76/525)	26	(139/525)	45	(234/525)	69	(360/525)	2	(11/500)	16	(78/500)	32	(162/500)	60	(299/500)
ATP	.	.	10	(73/701)	25	(174/701)	46	(319/701)	.	.	7	(39/547)	16	(89/547)	41	(225/547)
Combined data	8	(110/1450)	14	(307/2151)	31	(667/2151)	60	(1283/2151)	2	(28/1317)	9	(172/1864)	22	(413/1864)	51	(948/1864)
Alcohol abuse/dependence																
VAHCS	13	(125/925)	13	(117/925)	9	(80/925)	5	(49/925)	23	(186/817)	27	(224/817)	19	(153/817)	15	(119/817)
CHDS	11	(58/525)	10	(52/525)	5	(25/525)	4	(22/525)	26	(130/500)	17	(85/500)	14	(69/500)	15	(73/500)
ATP -data not collected	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
Combined data	13	(182/1450)	12	(169/1450)	7	(105/1450)	5	(71/1450)	24	(317/1317)	24	(310/1317)	17	(222/1317)	15	(193/1317)
Binge drinking																
VAHCS	31	(285/925)	32	(298/925)	22	(203/925)	20	(189/925)	55	(448/817)	58	(476/817)	57	(466/817)	51	(418/817)
CHDS	18	(94/525)	15	(80/525)	13	(68/525)	9	(48/525)	40	(200/500)	25	(127/500)	28	(138/500)	19	(97/500)
ATP	.	.	22	(155/701)	18	(126/701)	15	(104/701)	.	.	39	(213/547)	39	(212/547)	36	(198/547)
Combined data	26	(379/1450)	25	(533/2151)	18	(397/2151)	16	(341/2151)	49	(648/1317)	44	(816/1864)	44	(816/1864)	38	(713/1864)

Number of drinks	mean	(SD)	mean	(SD)	mean	(SD)	mean	(SD)	mean	(SD)	mean	(SD)	mean	(SD)	mean	(SD)
VAHCS	3.6	(5.4)	3.5	(5.2)	2.5	(5.2)	1.9	(3.1)	7.6	(8.6)	7.9	(8.6)	6.5	(8.0)	5.4	(6.0)
CHDS	5.4	(6.1)	4.9	(5.5)	4.1	(4.2)	3.1	(3.4)	10.4	(10.1)	7.3	(8.4)	6.3	(7.0)	5	(6.1)
ATP -data not collected	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
Combined data	4.3	(5.7)	4	(5.3)	3.1	(5.0)	2.4	(3.3)	8.7	(9.3)	7.7	(8.6)	6.4	(7.6)	5.2	(6.0)

Abuse/ dependence and number of drinks: Total participants = 2767, total observations = 11,068. Binge drinking: Total participants = 4015, total observations = 14,812.

* All n's estimated from MI proportions.

Table 2
Rates of alcohol outcomes by age of youngest child in combined cohort data, by participant gender and wave: imputed data, all participants with at least one background covariate and at least one wave of exposure and outcome.

	Females								Males							
	Age of youngest child								Age of youngest child							
	Not a parent		< 1 year		1 - 4 years		5+ years		Not a parent		< 1 year		1 - 4 years		5+ years	
	%	(n*/N)	%	(n/N)	%	(n/N)	%	(n/N)	%	(n/N)	%	(n/N)	%	(n/N)	%	(n/N)
Alcohol abuse/dependence																
21 years	13	(175/1340)	5	(2/31)	8	(6/76)	1	(0/2)	24	(312/1290)	14	(2/11)	19	(3/16)	.	.
24 years	13	(155/1217)	3	(2/58)	7	(9/140)	9	(3/36)	24	(279/1184)	16	(6/38)	26	(21/84)	28	(3/12)
30 years	9	(81/955)	2	(3/138)	5	(13/247)	7	(8/109)	17	(172/993)	10	(9/92)	17	(28/172)	21	(13/61)
35 years	7	(32/487)	2	(3/119)	3	(19/555)	6	(17/288)	17	(99/595)	8	(8/97)	14	(62/459)	14	(23/166)
Pooled	**11**	**(444/3999)**	**3**	**(9/347)**	**5**	**(47/1019)**	**6**	**(28/436)**	**21**	**(862/4061)**	**11**	**(25/238)**	**16**	**(115/730)**	**16**	**(38/239)**
Binge drinking																
21 years	28	(368/1340)	6	(2/31)	11	(9/77)	0	(0/2)	50	(640/1289)	29	(4/12)	33	(5/16)	.	.
24 years	27	(494/1844)	11	(10/88)	11	(20/172)	20	(10/47)	45	(754/1692)	37	(17/46)	37	(39/105)	32	(7/21)
30 years	23	(333/1484)	4	(8/192)	11	(37/347)	15	(19/129)	45	(651/1451)	43	(50/116)	38	(85/226)	43	(30/70)
35 years	22	(186/868)	8	(16/192)	12	(87/746)	15	(52/345)	42	(383/916)	39	(64/164)	33	(191/587)	38	(75/197)
Pooled	**25**	**(1382/5536)**	**7**	**(35/503)**	**11**	**(152/1342)**	**15**	**(81/523)**	**45**	**(2427/5349)**	**40**	**(135/337)**	**34**	**(320/935)**	**39**	**(112/288)**

Number of drinks	mean	(SD)	mean	(SD)	mean	(SD)	mean	(SD)	mean	(SD)	mean	(SD)	mean	(SD)	mean	(SD)
21 years	4.2	(5.7)	4	(6.8)	4.7	(5.8)	1.3	(2.7)	8.7	(9.3)	8.4	(8.3)	6.1	(8.4)	.	.
24 years	3.9	(5.2)	2.5	(3.7)	4.3	(6.2)	7	(6.4)	7.6	(8.3)	8.9	(12.0)	8.3	(10.2)	9	(8.2)
30 years	3.4	(5.2)	1.3	(3.0)	2	(3.5)	4.8	(6.2)	6.4	(7.7)	5.6	(6.0)	6.5	(7.9)	8.4	(8.2)
35 years	2.8	(3.3)	1.3	(2.2)	2.0	(2.9)	2.7	(3.9)	5.4	(5.9)	4.5	(4.4)	4.7	(5.2)	6.8	(8.7)
Pooled	**3.8**	**(5.2)**	**1.8**	**(3.5)**	**2.6**	**(4.0)**	**3.6**	**(4.9)**	**7.3**	**(8.3)**	**5.8**	**(7.1)**	**5.6**	**(6.8)**	**7.3**	**(8.5)**

Abuse/ dependence and number of drinks: Total participants = 2767, total observations = 11,068. Binge drinking: Total participants = 4015, total observations = 14,812.

* All n's estimated from MI proportions.

Table 3
Associations between age of youngest child and alcohol outcomes by participant gender, in progressively adjusted models. Data pooled over three cohorts and repeated assessments from age 21-35. Models using generalised estimating equations with robust SEs.

	Females							Males							Gender difference
	Age of youngest child							Age of youngest child							
	Not a parent		< 1 year	1 - 4 years		5+ years		Not a parent		< 1 year	1 - 4 years		5+ years		
MODEL 1, adjusted for cohort and participant age	**RR**	**(95% CI)**	**RR**	**RR**	**(95% CI)**	**RR**	**(95% CI)**	***P1***	**RR**	**(95% CI)**	**RR**	**RR**	**(95% CI)**	**RR**	**(95% CI)**	***P2***	***P3***
Alcohol abuse/dependence	3.8	(1.6, 8.8)	1	2.0	(0.8, 5.0)	2.8	(1.1, 7.2)	< 0.001	1.6	(1.1, 2.3)	1	1.5	(1.0, 2.3)	1.6	(1.0, 2.6)	0.102	0.009
Binge drinking	3.2	(2.2, 4.5)	1	1.7	(1.1, 2.5)	2.4	(1.6, 3.6)	< 0.001	1.1	(1.0, 1.3)	1	0.9	(0.8, 1.1)	1.1	(0.9, 1.3)	0.003	< 0.001
	IRR	**(95% CI)**	**IRR**	**IRR**	**(95% CI)**	**IRR**	**(95% CI)**	***P1***	**IRR**	**(95% CI)**	**IRR**	**IRR**	**(95% CI)**	**IRR**	**(95% CI)**	***P2***	***P3***
Number of drinks	2.0	(1.6, 2.6)	1	1.5	(1.2, 1.9)	2.1	(1.7, 2.8)	< 0.001	1.2	(1.0, 1.4)	1	1.0	(0.9, 1.2)	1.3	(1.1, 1.6)	0.006	< 0.001
MODEL 2, also adjusted for background covs*	**RR**	**(95% CI)**	**RR**	**RR**	**(95% CI)**	**RR**	**(95% CI)**	***P1***	**RR**	**(95% CI)**	**RR**	**RR**	**(95% CI)**	**RR**	**(95% CI)**	***P2***	***P3***
Alcohol abuse/dependence	4.2	(1.8, 9.5)	1	2.0	(0.8, 4.8)	2.5	(1.0, 6.3)	< 0.001	1.6	(1.1, 2.4)	1	1.5	(1.0, 2.2)	1.5	(1.0, 2.4)	0.082	0.002
Binge drinking	3.3	(2.3, 4.7)	1	1.7	(1.1, 2.4)	2.3	(1.6, 3.4)	< 0.001	1.1	(1.0, 1.3)	1	0.9	(0.8, 1.1)	1.1	(0.9, 1.3)	0.002	< 0.001
	IRR	**(95% CI)**	**IRR**	**IRR**	**(95% CI)**	**IRR**	**(95% CI)**	***P1***	**IRR**	**(95% CI)**	**IRR**	**IRR**	**(95% CI)**	**IRR**	**(95% CI)**	***P2***	
Number of drinks	2.2	(1.7, 2.7)	1	1.5	(1.2, 1.9)	2.0	(1.6, 2.6)	< 0.001	1.2	(1.0, 1.4)	1	1.0	(0.9, 1.2)	1.3	(1.0, 1.5)	0.007	< 0.001
MODEL 3, also adjusted for time-varying covs**	**RR**	**(95% CI)**	**RR**	**RR**	**(95% CI)**	**RR**	**(95% CI)**	***P1***	**RR**	**(95% CI)**	**RR**	**RR**	**(95% CI)**	**RR**	**(95% CI)**	***P2***	***P3***
Alcohol abuse/dependence	3.5	(1.5, 7.9)	1	1.9	(0.8, 4.5)	2.1	(0.8, 5.2)	< 0.001	1.4	(0.9, 2.0)	1	1.5	(1.0, 2.2)	1.4	(0.9, 2.2)	0.382	0.001
Binge drinking	3.0	(2.1, 4.3)	1	1.6	(1.1, 2.4)	2.1	(1.4, 3.2)	< 0.001	1.0	(0.9, 1.2)	1	0.9	(0.8, 1.1)	1.0	(0.8, 1.2)	0.191	< 0.001
	IRR	**(95% CI)**	**IRR**	**IRR**	**(95% CI)**	**IRR**	**(95% CI)**	***P1***	**IRR**	**(95% CI)**	**IRR**	**IRR**	**(95% CI)**	**IRR**	**(95% CI)**	***P2***	
Number of drinks	2.0	(1.6, 2.6)	1	1.5	(1.2, 1.9)	1.9	(1.5, 2.5)	< 0.001	1.1	(0.9, 1.3)	1	1.0	(0.8, 1.2)	1.2	(1.0, 1.5)	0.107	< 0.001

P^1: p value for effect of child age (all categories compared to baseline < 1 year) in females.
P^2: p value for effect of child age (all categories compared to baseline < 1 year) in males.
P^3: p value for interaction between effect of child age and gender.
All models adjusted for cohort and age. Model 2 also adjusted for background confounders, model 3 additionally adjusted for time-varying covariates: imputed data, all participants with at least one background covariate and at least one wave of exposure and outcome. Abuse/ dependence and number of drinks: Total participants = 2767, total observations = 11,068. Binge drinking: Total participants = 4015, total observations = 14,812.

* Adol and background covariate score: propensity score based on predicted likelihood of being a parent at wave.

** Time-varying covariates: have a serious partner, live with parents, have anxiety and/ or depression.

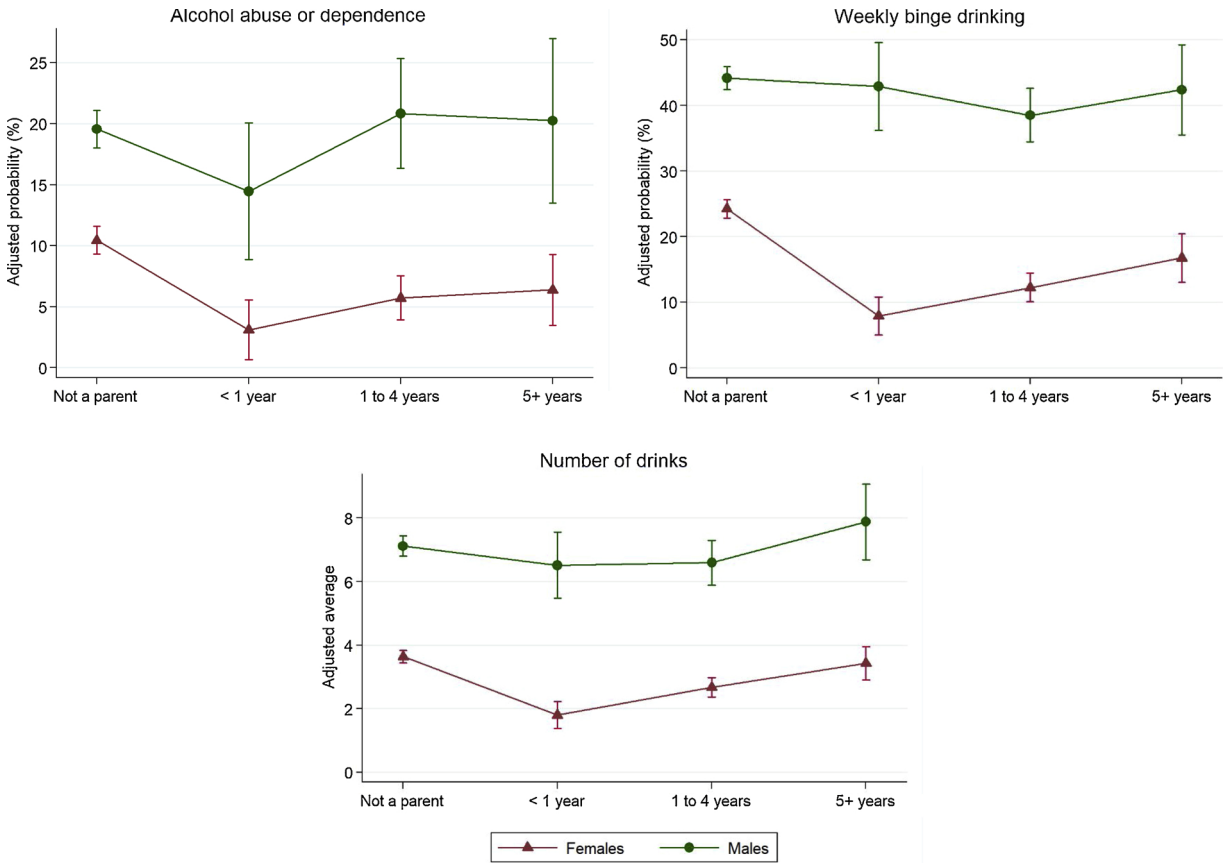

Fig. 1. Adjusted probabilities of alcohol abuse or dependence and binge drinking (with 95% CIs) and adjusted average number of drinks (with SEs) from fully adjusted models, pooled over three cohorts and repeated assessments from age 21–35, by participant gender: imputed data, all participants with background covariates and at least one wave of exposure and outcome. Abuse/ dependence and number of drinks: Total participants = 2767, total observations = 11,068. Binge drinking: Total participants = 4015, total observations = 14,812.

sizes by cohort in binge drinking in women, resulting from the low rates of binge drinking in CHDS mothers with children aged < 1 year, but evidence for an interaction by cohort was weak ($p = 0.331$). There was evidence ($p < 0.001$) of a difference between VAHCS and CHDS women for number of drinks, but the difference in effect sizes was minimal.

The pattern of results was also similar by age of participant (allowing for interaction with sex) (Supplementary Table 5). There was some evidence of difference in the effects of child age across age groups of women ($p = 0.027$, $p = 0.085$ and $p = 0.132$ for tests of interaction in models for alcohol abuse, binge drinking and average drinks, respectively), but there was no discernible pattern in the change in effects across female age (i.e., the effects did not increase or decrease as women grew older). There was no evidence of difference in effects of child age across male age for any alcohol outcome ($p > 0.4$).

3.5. Sensitivity Analysis – Complete Case Data

Supplementary Table 6 shows that fully adjusted associations between age of youngest child and drinking behaviours in observed data followed slightly larger effect sizes but similar patterns to those in the imputed datasets. Also in observed data, Supplementary Table 7 demonstrates little evidence of an additional effect from a concentration of child spacing (all $p > 0.3$), with little to no additional increase in risk or rate of outcomes in those with more than one child aged < 3 years, and little change to estimate sizes for the effect of age of youngest child.

4. Discussion

There were marked and consistent differences in patterns of alcohol consumption between women with children and those without. Fewer females with young children met the criteria for alcohol use disorder or binge drinking than those without children. For mothers, an increase in age of the youngest child brought an increase in the risks of alcohol abuse/dependence and binge drinking. The number of drinks consumed also increased with age of youngest child and, by the time the youngest reached 5 years of age, was close to pre-parenting levels. The effect sizes for women were large for all three measures of alcohol consumption in the short-term, and decreased in magnitude over time. In contrast, there was no evidence that parenting was associated with changes in male drinking.

Across cohorts, the transition to parenthood was associated with reduced alcohol consumption for young mothers but, for most, this was relatively short-lived and did not equate to a persisting 'maturing out' (Vergés et al., 2012; Winick, 1962; Yamaguchi and Kandel, 1985). There are several possible explanations for the observed associations. First, it is possible that participants who were parents had additional protective factors, including an overall healthier lifestyle and a social circle involving lower levels of alcohol consumption. Secondly, persisting alcohol consumption during pregnancy adversely affects the growth and development of the offspring (Patton et al., 2018; Vassoler et al., 2014; Muggli et al., 2017). As such, it is likely that young mothers' decision to protect their child/ren during the pregnancy and breastfeeding periods (Nykjaer et al., 2014; Tay et al., 2017), in addition to the responsibilities and demands associated with this stage of parenting, mean that many women - who have traditionally been the

primary caregiver - do not consume alcohol as often, nor at such high levels, as they did prior to having children. The frequency of drinking in social situations (e.g., at a bar with friends) also typically decreases in the short-term following the birth of a child (Paradis et al., 2011). Although we did not measure the context in which alcohol was consumed, this may have contributed to our findings. As children get older, the need for breastfeeding decreases (Tay et al., 2017) and this may contribute to the increase in risky alcohol consumption we observed. No equivalent social mechanism exists for males, likely contributing to the absence of change we observed.

There are clear public health implications arising from our findings. For most women in our study, the reductions in alcohol consumption associated with parenting a child < 1 year of age almost disappeared after the first year of parenthood. The transition to parenthood is now occurring later (Kippen, 2004) and, in many high-income countries, a growing number of women do not ever make this transition (ABS. Births, 2016). If men and women are entering pregnancy and parenthood with more entrenched patterns of heavy alcohol consumption, it is more likely that these patterns will persist beyond these transitions. Additionally, the duration of time between pregnancies – previously a brief period that exerted a protective effect on women's alcohol consumption – has lengthened in recent years (ABS. Births, 2016) and this period subsequently now functions as less of a protective factor. Most reductions in alcohol use were relatively short-lived; future public health policy and research efforts may seek to identify candidate interventions to extend this window of reduced drinking from pregnancy and childbirth beyond the 12 months we observed and through to the next pregnancy. A timely health sector response would likely benefit both women (to extend the duration of the protective effect on their alcohol consumption beyond the first 12 months following childbirth) and men (to reduce their alcohol use during this period). Alongside the transition to parenthood, a number of other factors likely exert an influence on alcohol use. In addition to getting married, ending full-time education, entering the labour market and purchasing a first property, social norms within one's peer group may play an important contributing role in determining alcohol use (Borsari and Carey, 2001; Leonard and Mudar, 2003; Andrews et al., 2002). Delays in the transition to parenthood and fewer births suggest that we can no longer rely on these processes to bring about a maturing out of heavy and problematic alcohol use. As such, a different - and more assertive - approach to prevention of alcohol use disorders in the young adult years is required.

4.1. Strengths and Limitations

Our study has several strengths. First, by integrating participant-level data from three prospective cohort studies, we were able to control for a broader range of covariates (using propensity score modelling) than would be possible using traditional study-level meta-analytic methods. Second, by assessing the age of participants' youngest child across cohorts and time, we were able to examine the temporal impact of becoming a parent on alcohol use for both males and females. The repeated measures analysis enabled us to assess the effects of child age across the parental lifespan, and to determine whether the effects of parental stage on problematic alcohol consumption differed according to parental age. Finally, our findings were consistent across cohorts despite data being collected by different research teams, using different measures, with distinct populations, and with different timing (and consequently different prevalence) of exposures.

Our study also has limitations. First, data were obtained by self-report and may have been subject to a social desirability bias, the extent of which can vary with age (Brener et al., 2003). Second, our datasets were sampled from countries characterised by permissive and heavy-drinking cultures. Similarities in the cultural and social context and epidemiology of alcohol use between these and other high-income countries such as the USA (UNICEF, 2013) suggests that findings are likely to be generalisable to those settings. However, generalisability to low- and middle-income countries, where fertility rates and the demography of parenthood differ, remains to be established. Finally, our data did not permit analysis of the reasons why women gradually increased their alcohol consumption over the first few years following childbirth. It is possible that some women may drink alcohol as a coping mechanism in response to the demands of parenthood, or that they only ceased using alcohol during pregnancy out of necessity to protect their unborn child and, with this need removed, there are fewer perceived reasons to reduce their alcohol consumption. Such reasons should be explored in future studies.

4.2. Conclusions

The transition to parenthood no longer has a sustained impact on parental alcohol consumption in contemporary Australasia, a region that had made a demographic transition to low fertility and late parenthood. No changes were observed in male drinking during the perinatal period or beyond, and reductions in women's drinking were the most pronounced during the first 12 months post-partum. A more assertive approach to prevention and reducing alcohol consumption is now needed during these years. Given the numerous adverse outcomes associated with having a parent with an alcohol use disorder (West and Prinz, 1987; Eiden et al., 1999; Lynskey et al., 1994; Christoffersen and Soothill, 2003), investment in interventions in adults aged 25–35 years will likely yield a double dividend for parents and the next generation alike.

Conflicts of Interest

No conflict declared.

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Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.drugalcdep.2019.02.004.

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