BMC Public Health

RESEARCH Open Access

Early and risky adolescent alcohol use independently predict alcohol, tobacco, cannabis and other drug use in early adulthood in Ireland: a longitudinal analysis of a nationally representative cohort

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Abstract

Background Early and risky adolescent alcohol use have each been associated with adult alcohol consumption. However, it remains unclear whether these behaviours independently predict later-life substance use when considered jointly, and research examining links with substances other than alcohol is limited. This study addresses these gaps by examining longitudinal associations between age at first alcohol and risky adolescent alcohol use, with alcohol, tobacco, cannabis and other drug use in early adulthood, and aims to identify critical periods for public health interventions.

Methods Growing Up in Ireland is a nationally representative cohort (recruited aged 9 [Wave 1], born 1997–1998). Survey-weighted logistic regression examined whether age at first alcoholic drink and risky alcohol use at age 17 (Alcohol Use Disorders Identification Test scores) independently predict high-risk alcohol (AUDIT > 15), tobacco, cannabis and other drug use at age 20. Models were adjusted for age, sex, academic ability, personality, psychological factors, socioeconomic status, familial, peer and neighbourhood substance use.

Results The study included 4554 participants (49.8% female). Early alcohol use was common, with 27% reporting use aged 14 or younger. By age 20, 14% reported high-risk alcohol, 38% tobacco, 24% cannabis and 28% other drug use. Older age at first alcohol was associated with dose-response reductions in the odds of high-risk alcohol, tobacco, cannabis and other drug use at age 20, relative to those initiating alcohol at 14 or younger. Adolescents with high-risk alcohol use had double the odds of tobacco (adjusted odds ratio (aOR) 2.1, 95% confidence intervals (CI) 1.3–3.30) and other drug use (aOR 2.5, 95% CI 1.6–4.1) and an 11-fold increase in the odds of continued high-risk alcohol use (aOR 11.5, 95% CI 7.0–18.6) at age 20, relative to adolescents with low-risk alcohol use.

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Conclusions Age at first alcohol and risky adolescent alcohol use independently predict substance use in early adulthood when considered jointly in extensively adjusted models. These findings highlight the continued urgent need for public health interventions that address factors associated with early alcohol use and support adolescents who use alcohol in a high-risk manner given their elevated risk of progression to more serious substance use as adults.

Keywords Age at first alcoholic drink, Alcohol use disorders identification test, Tobacco, Cannabis, Drugs, Adolescent, Young adult

Introduction

Worldwide, 2.6 million deaths were attributed to alcohol consumption in 2019 [1]. Alcohol use is a causal factor in more than 200 diseases, including alcohol dependence, liver disease, cardiovascular disease, and cancer [1, 2]. Younger age groups are disproportionately affected, with 13% of alcohol-attributable deaths occurring among those aged 20–39 years old [1]. Even low levels of alcohol consumption among young people are associated with health risks [3]. In Ireland, alcohol use results in an estimated three to four deaths per day, ranking it as the eighth leading cause of death nationally [4].

Despite existing population-level policies that seek to reduce alcohol-related harms among young people, such as the minimum legal purchase age of 18 in Ireland, alcohol use often begins in early-mid adolescence, with levels and frequency of use typically peaking in early adulthood [5]. Delaying the onset of alcohol use among young people is thought important to reduce subsequent risk of problematic alcohol use, based on literature reporting correlations between age at first alcoholic drink (AFD) and alcohol-related outcomes later in life [1, 5, 6]. However, a causal association between AFD and later-life adverse outcomes remains the subject of intense debate [6, 7]. Some argue that earlier alcohol use results in increased alcohol consumption [8], for which there is consistent evidence of a link to adult alcohol problems [9]. Others suggest that early alcohol use merely serves as a marker of risk for higher risk substance use, meaning that those who have underlying vulnerability or risk factors for alcohol and other drug problems, such as externalising behaviours, may also be likely to start drinking alcohol early [5]. It is possible that both mechanisms—causal effects of early alcohol use and its role as a marker of risk—may be at play.

A number of studies reporting associations between AFD and later substance use face methodological challenges [6, 7]. These include: cross-sectional data, inconsistent measures (varying between age at first sip, full standard drink, intoxication and regular drinking), selection bias, retrospective reports which may suffer from recall bias, and inadequate control for potentially confounding factors such as personality, externalising behaviours, familial, peer and neighbourhood substance use [6, 7]. Longitudinal studies using prospectively collected data are thought most suitable for examination of the relationship between AFD and substance use outcomes, but a 2014 review highlighted a shortage of such studies [6].

We identified twelve longitudinal studies that examined the impact of AFD on alcohol or other drug use in late adolescence or early adulthood in general population samples. Among these, five demonstrated an association between AFD and subsequent alcohol or other drug-related outcomes that was robust to adjustment for relevant confounders [8, 10–12], two report that the association was attenuated when confounders were adjusted for [13, 14], and five did not control for important confounders, such as externalising behaviours and familial drug-use [15–19]. Only three of the studies examined outcomes involving drugs other than alcohol [12, 14, 17]. Behrendt et al. reported younger alcohol onset was associated with cannabis use in a German cohort [12], whereas Newton-Howes et al. did not observe a significant association with substance use disorders among a New Zealand cohort [14]. Lastly, Moss et al. reported a significant association between early alcohol and ever use of illicit drugs, but not for ever use of cocaine or daily cannabis use in a US cohort [17]. However, this analysis did not adjust for externalising symptoms [17].

Although the literature on AFD has expanded in recent years, existing evidence is conflicting, and there has been limited investigation of the association between AFD and subsequent use of drugs other than alcohol. Additionally, only one study has assessed whether both early and excessive alcohol use independently predicted alcohol-related problems when considered jointly [10]. Recent studies argue that further investigation of the impact of alcohol use behaviours across the life course using longitudinal data is required to support evidence-informed prevention practice and policy that aim to reduce alcohol-related harms [6, 10].

This study addresses these gaps by examining whether AFD and risky alcohol use at age 17, measured by scores on the Alcohol Use Disorder Identification Test (AUDIT), independently predict high-risk alcohol, tobacco, cannabis and other drug use at 20 years old, when considered jointly, and adjusted for risk factors which may confound the association [20]. We hypothesised that earlier AFD and elevated AUDIT scores would independently predict a higher likelihood of high-risk alcohol, tobacco, cannabis and other drug use at 20 years old, despite adjustment for covariates.

Methods

Study design

This is a secondary analysis of Growing Up in Ireland (GUI), a nationally representative cohort of young people in Ireland. GUI used two-stage sampling, with the primary school system as the initial sampling frame. Wave 1 included 8568 children aged 9 in 2007/2008. Wave 2 was conducted in 2011/2012 when participants were aged 13 ($N = 7525$, response rate 88%). Wave 3 followed in 2015/2016 at age 17 ($N = 6216$, response rate 73%), and Wave 4 took place in 2018/2019 at age 20 ($N = 5190$, response rate 61%). In accordance with statistical disclosure control rules for GUI, participant counts under 30 are not presented in this paper. The strengthening the reporting of observational studies in epidemiology (STROBE) reporting guidelines were followed [21].

Participants

Participants who completed Waves 1–4 were eligible for inclusion. Those who had responded yes to a control question on fictitious drug use or were missing observations for the outcomes or school ID were excluded. In total, 175 participants were excluded, resulting in a study population of 4554.

Procedures

Exposures

Age at first alcoholic drink (AFD) was defined as the first time the young person had an alcoholic drink (more than a few sips) and was constructed from four self-reported variables collected at Waves 2, 3 and 4. Notably, this variable was not collected at Wave 1 of GUI. To minimise recall bias, responses from Wave 2– when participants were aged 13– were prioritised, followed by those from Waves 3 and 4 [6]. The merging process followed these steps: Participants who reported alcohol use at Wave 2 were categorised as ≤ 14 (years old). Wave 3 responses were then incorporated into the categories of ≤ 14, 15, 16, 17, 18 unless a participant had already been classified as ≤ 14 based on their response at Wave 2. Responses from Wave 4 were used only for participants who had not indicated age at first alcoholic drink at either Wave 2 or 3. Ultimately, AFD was categorised as aged ≤ 14, 15, 16, 17, ≥ 18 and no alcohol use. Due to slight changes in the question and response options between Waves 2 and 3 of GUI (Methods A1), it was necessary to group those with the earliest AFD into the ≤ 14 category. Consistency of responses between waves was assessed using percentage correct and intraclass correlation coefficient.

Alcohol Use Disorder Identification Test (AUDIT) scores, a widely used alcohol screening measure developed by the World Health Organization, were self-reported at age 17 (Wave 3), and standard cut-points were applied: "0–7" (low-risk), "8–15" (increasing risk) and ">15" (high-risk) [22].

Outcomes

The four outcomes of interest, all self-reported at age 20 (Wave 4), were high-risk alcohol use (AUDIT > 15), current tobacco use (occasional or daily), current cannabis use (occasional or weekly), and past-year other drug use, which included cocaine, ecstasy, ketamine, amphetamines, poppers, LSD, magic mushrooms, crack, or the misuse of prescription drugs. Each outcome was dichotomised, distinguishing between those who reported use at age 20, and those who did not.

Covariates

Individual, family, social and environmental covariates considered potential confounders were chosen a priori, including age at outcome [5], sex (male, female) [23, 24], and personality (measured using parent-reported scores on the validated Ten-Item Personality Inventory (TIPI) at Wave 2 [25] across the "Big-Five" personality domains: Extraversion, Conscientiousness, Openness to Experiences, Agreeableness and Emotional Stability) [26]. Externalising behaviours were represented by parent-reported scores on the Conduct and Hyperactivity subscales of the well-established Strengths and Difficulties Questionnaire (SDQ) at Waves 1–3 [13, 24, 27, 28]. Childhood academic ability was measured using the Drumcondra reading test which was administered at Wave 1 [29–31]. Family variables that were parent-reported included parental education (Wave 2) [32], social class (Wave 2) [33], household structure (Wave 2) [24], parental tobacco use (Waves 1–3) [24, 34], and parental monitoring (Wave 2) [35]. Familial alcohol use disorder (AUD) or drug-use was parent-reported at Waves 1–2 and youth-reported at Wave 3 [23, 24, 30, 36, 37]. Social influences were youth-reported at Wave 3 and included peer alcohol, tobacco and cannabis use [23, 30, 38–40]. Lastly, environmental influences [41, 42], measured using neighbourhood substance use, was parent-reported at Wave 2.

Further details on variables are available in Methods A1.

Statistical analysis

R version 4.4.0 was used.

Summary statistics and missing observations were examined. Under the missing at random assumption, multiple imputation by chained equations was used to impute incomplete exposures and create ten complete data frames [43].

Bivariable and multivariable logistic regression models were fitted using generalised estimating equations

(GEE) with an exchangeable correlation structure and a cluster vector of school ID, to account for participant clustering within schools [44]. In the bivariable analysis, separate models were run for AFD and AUDIT with each outcome, resulting in a total of eight bivariable models. In the multivariable analysis, AFD and AUDIT were included in the same models for each outcome, leading to four multivariable models. Regression models were fit to each imputed dataset and effect estimates were pooled. Overall variance was calculated, taking into account variance within and between datasets, in accordance with Rubin's rules [43]. To ensure results could be considered nationally representative, analyses were re-weighted. Survey weights were provided by GUI and involve the structural adjustment of the sample to the population using Census of Population characteristics, including sex, socioeconomic status, household structure and Drumcondra test scores, to account for interwave attrition ensuring that the results can be considered nationally representative, and adjustment of the standard errors to account for the two-stage sampling design [45]. Regression analyses were conducted in both multiply imputed and complete case data. Multicollinearity and model explanatory power were assessed with variance inflation factors and Tjur's R square, respectively.

Further detail is available in Methods A2–A5.

Results

Descriptive statistics

The study population included 4554 participants (49% female, mean age at outcome 20). Over a quarter had their first alcoholic drink aged 14 or younger. At age 17, 6% had AUDIT scores exceeding 15, which is considered high-risk. In early adulthood, levels of substance use were high– 14% reported alcohol use that was classified as high-risk, 38% reported current tobacco use (23% occasionally, 15% daily), 24% current cannabis use and 28% had used other drugs in the past-year. (Table 1)

Missingness within variables was low. Only two variables exceeded 1.5% missingness: AUDIT score at 17 years old (7%) and Drumcondra test (2%) (Table 1). When characteristics of missing and non-missing observations were compared in these variables, there were significant differences, suggesting a missing-at-random mechanism. (Table A1)

Cross-tabulation

When AFD and AUDIT scores at age 17 were cross-tabulated with the four outcomes, a clear pattern emerged: older AFD was associated with a lower proportion of high-risk alcohol, tobacco, cannabis or other drug use at 20. Higher AUDIT scores at age 17 were associated with higher proportions of high-risk alcohol, tobacco, cannabis and other drug use at 20. (Table 2)

Bivariable regression analysis

Age at first alcoholic drink

A strong association was observed between AFD and substance use at age 20. As AFD was delayed, effect estimates declined in a monotonic fashion, indicating reduced odds for each substance use outcome at age 20, relative to those who had their first alcoholic drink aged 14 or younger. (Table 3)

AUDIT scores at 17 years old

There was strong evidence supporting an association between AUDIT scores at age 17 and each of the outcomes at 20. The largest effect estimates were demonstrated for the relationship between AUDIT scores at 17 and 20 years old. Those whose alcohol use had been classified as at increasing risk at age 17 had a four-fold increase (OR 4.1, 95% CI 3.0–5.4) in the odds of high-risk alcohol use at age 20, while those whose alcohol use was already classified as high-risk when aged 17 had a 14-fold increase (OR 13.8, 95% 8.8–21.6) in the odds of continued high-risk alcohol use at age 20, relative to adolescents who had been classified as low-risk. Across the four outcomes considered, effect estimates increased in a dose-response fashion with higher AUDIT scores at 17. (Table 3)

Multivariable regression analyses

In multivariable analyses, which included both exposures and covariates as detailed in Table 4, older AFD remained associated with reduced probability of high-risk alcohol use at age 20 although effect sizes were attenuated. Elevated AUDIT scores at age 17 remained strongly associated with high-risk alcohol use at 20. As AFD was delayed, odds ratios for tobacco use at 20 sequentially declined, while elevated AUDIT scores at 17 were associated with increased odds for tobacco use. Similarly, older AFD was associated with sequential reductions in the odds of cannabis use aged 20. While elevated AUDIT scores at 17 were associated with elevated effect estimates for cannabis use at 20, this association was not statistically significant (aOR 1.31, 95% CI 0.85–2.00). There was a strong association between AFD and other drug use at 20, with odds ratios decreasing sequentially with older AFD. Elevated AUDIT scores at 17 were strongly associated with other drug use, with scores > 15 linked to more than a two-fold increased odds (aOR 2.5, 95% CI 1.6–4.1). (Table 4)

Additional analyses

Consistency of AFD measures: between Wave 2 and 3 measures there was 78% agreement, Wave 2 and 4 measures 80% and between Wave 3 and 4 measures, 55% and the intraclass correlation coefficient was 0.7 (95% CI 0.7–0.8) (Table A2). *Complete case analyses*: Direction and

Table 1 Descriptive statistics and missing observations within the study population ($N=4554$)

	Unweighted	Weighted	Missing (*N* (%)
Exposures			
Age at first alcoholic drink N (%)			0 (0.00)
≤ 14	1065 (23.39)	1232 (27.05)	
15	810 (17.79)	763 (16.74)	
16	1362 (29.91)	1241 (27.26)	
17	874 (19.19)	878 (19.29)	
≥ 18	326 (7.16)	305 (6.69)	
No alcohol use	117 (2.57)	135 (2.96)	
AUDIT score at 17 years old N (%)			302 (6.63)
Low-risk (0–7)	2675 (62.91)	2650 (62.33)	
Increasing risk (8–15)	1369 (32.20)	1368 (32.18)	
High-risk (> 15)	208 (4.90)	234 (5.49)	
Outcomes			
AUDIT score at 20 years old N (%)			0 (0.00)
Low/increasing risk (≤ 15)	3871 (85.00)	3941 (86.53)	
High-risk (> 15)	683 (15.00)	613 (13.47)	
Tobacco use at 20 years old N (%): yes	1736 (38.12)	1744 (38.29)	0 (0.00)
Cannabis use at 20 years old N (%): yes	1233 (27.08)	1109 (24.35)	0 (0.00)
Other drug use at 20 years old N (%): yes	1266 (27.80)	1270 (27.90)	0 (0.00)
Covariates			0 (0.00)
Age at outcome mean (SD)	20.10 (0.29)	20.07 (0.29)	
Sex N (%): female	2362 (51.87)	2270 (49.84)	0 (0.00)
Drumcondra reading test median (IQR)	77.80 (63.90–88.90)	72.50 (52.50–85.00)	84 (1.85)
TIPI Extraversion median (IQR)	3.50 (2.50–5.50)	3.50 (2.50–5.50)	< 0.66%*
TIPI Agreeableness median (IQR)	5.50 (3.50–7.00)	5.00 (3.50–7.00)	< 0.66%*
TIPI Emotional Stability median (IQR**)**	4.50 (3.00–6.50)	4.00 (3.00–6.50)	< 0.66%*
TIPI Openness to experiences median (IQR)	4.50 (3.00–6.50)	5.00 (3.00–6.50)	< 0.66%*
TIPI Conscientiousness median (IQR)	4.50 (3.00–6.50)	4.00 (2.50–7.00)	< 0.66%*
Parental monitoring median (IQR)	42.0 (39.0–44.0)	42 (38–44)	< 0.66%*
SDQ Conduct N (%)			0 (0.00)
Normal	3563 (78.24)	3344 (73.44)	
Borderline	534 (11.73)	602 (13.22)	
Abnormal	457 (10.04)	608 (13.35)	
SDQ Hyperactivity N (%)			0 (0.00)
Normal	3598 (79.01)	3341 (73.36)	
Borderline	353 (7.75)	423 (9.29)	
Abnormal	603 (13.24)	790 (17.35)	
Household social class N (%)			0 (0.00)
Professional	824 (18.09)	466 (10.23)	
Managerial & technical	1890 (41.50)	1485 (32.60)	
Non-manual	831 (18.25)	887 (19.47)	
Manual	805 (17.68)	1230 (27.02)	
Other	204 (4.48)	486 (10.68)	
Parental education N (%)			0 (0.00)
3rd level degree	2082 (45.72)	1295 (28.44)	
3rd level non-degree	1137 (24.97)	1038 (22.80)	
High secondary &/TechVoc	1077 (23.65)	1551 (34.05)	
Low secondary or less	258 (5.67)	670 (14.71)	
Household structure N (%)			0 (0.00)
Two-parent	4060 (89.15)	3662 (80.42)	
One-parent	494 (10.85)	892 (19.58)	
Familial AUD/drug-use N (%): yes	455 (9.99)	586 (12.88)	0 (0.00)
Parental smoking N (%): yes	1967 (43.19)	2271 (49.86)	0 (0.00)

Table 1 (continued)

	Unweighted	Weighted	Missing (*N* (%)
Peer drunkenness N (%): yes	4321 (95.53)	4285 (94.74)	31 (0.68)
Peer tobacco use N (%): yes	3635 (80.40)	3632 (80.34)	33 (0.72)
Peer cannabis use N (%): yes	2632 (58.32)	2571 (56.97)	41 (0.90)
Neighbourhood substance use N (%): common	9%*	12%*	< 0.66%*

N = count, SD = standard deviation, IQR = Interquartile Range, AUDIT = Alcohol Use Disorders Identification Test, TIPI = Ten-Item Personality Inventory, SDQ = Strengths and Difficulties Questionnaire, TechVoc = technical and vocational, AUD = alcohol use disorder

*Exact numbers suppressed due to cell counts < 30

Table 2 Age at first alcoholic drink and AUDIT scores at 17 years old cross-tabulated with AUDIT scores, tobacco, cannabis and other drug use at 20 years old

	AUDIT		***Tobacco***		***Cannabis***		***Other drugs***	
	≤ 15	**> 15**	**No**	**Yes**	**No**	**Yes**	**No**	**Yes**
	N (%) *							
Age at first alcoholic drink								
≤ 14	970 (78.73)	262 (21.27)	536 (43.52)	696 (56.48)	799 (64.85)	433 (35.15)	614 (49.80)	618 (50.20)
15	625 (81.92)	138 (18.08)	363 (47.59)	400 (52.41)	506 (66.34)	257 (33.66)	479 (62.84)	283 (37.16)
16	1105 (89.07)	136 (10.94)	818 (65.88)	423 (34.12)	950 (76.57)	291 (23.43)	980 (78.94)	261 (21.06)
≥ 17/no alcohol**	1240 (94.10)	78 (5.90)	1093 (82.93)	225 (17.07)	1190 (90.28)	128 (9.72)	1211 (91.86)	107 (8.14)
AUDIT score at 17 years old* **								
Low-risk	2481 (93.60)	170 (6.40)	1877 (70.81)	773 (29.19)	2158 (81.44)	492 (18.56)	2145 (80.94)	505 (19.06)
Increasing risk	1061 (77.57)	307 (22.43)	599 (43.76)	770 (56.24)	885 (64.65)	484 (35.35)	778 (56.87)	590 (43.13)
High-risk	119 (51.04)	114 (48.96)	85 (36.54)	148 (63.46)	133 (57.05)	100 (42.95)	89 (37.89)	145 (62.11)

AUDIT = Alcohol Use Disorders Identification Test, N = count

*weighted cell counts and row percentages are based on observed data therefore the sample size is 4554 for cross-tabulations between age at first alcoholic drink and outcomes, but 4252 for cross-tabulations with AUDIT score at 17 years old due to missing observations (*N* = 302)

**due to Central Statistics Office Statistical Disclosure Control rules on minimum cell counts, cross-tabulations that resulted in cells containing < 30 study participants were collapsed

*** AUDIT scores were categorised as "0–7" (low-risk), "8–15" (increasing risk) and ">15" (high-risk)

Table 3 Age at first alcoholic drink, AUDIT score at 17 years old and high-risk alcohol, tobacco, cannabis and other drug use at 20 years old examined using survey-weighted bivariable GEE logistic regression models in multiply imputed data (*N* = 4554)

	High-risk alcohol		***Tobacco***		***Cannabis***		***Other drugs***	
	OR (95% CI)	**p**	**OR (95% CI)**	**p**	**OR (95% CI)**	**p**	**OR (95% CI)**	**p**
Age at first alcoholic drink								
≤ 14	Ref		Ref		Ref		Ref	
15	0.81 (0.61–1.09)	0.169	0.86 (0.65–1.13)	0.28	0.93 (0.73–1.19)	0.556	0.59 (0.45–0.76)	< 0.001
16	0.44 (0.33–0.60)	< 0.001	0.40 (0.32–0.50)	< 0.001	0.56 (0.44–0.71)	< 0.001	0.26 (0.20–0.35)	< 0.001
17	0.32 (0.22–0.46)	< 0.001	0.23 (0.17–0.31)	< 0.001	0.25 (0.19–0.34)	< 0.001	0.11 (0.08–0.15)	< 0.001
≥ 18/no alcohol*	0.07 (0.02–0.22)	< 0.001	0.04 (0.02–0.10)	< 0.001	0.10 (0.05–0.21)	< 0.001	0.05 (0.02–0.11)	< 0.001
AUDIT score at 17 years old**								
Low-risk	Ref		Ref		Ref		Ref	
Increasing risk	4.06 (3.04–5.43)	< 0.001	3.19 (2.63–3.86)	< 0.001	2.44 (1.97–3.02)	< 0.001	3.34 (2.70–4.11)	< 0.001
High-risk	13.81 (8.84–21.56)	< 0.001	4.40 (2.86–6.77)	< 0.001	3.47 (2.35–5.12)	< 0.001	7.18 (4.73–10.89)	< 0.001

AUDIT = Alcohol Use Disorders Identification Test, GEE = generalised estimating equations, N = count, OR = odds ratio, CI = confidence intervals, p = p-value

*Categories collapsed to achieve stable model estimates

**AUDIT scores were categorised as "0–7" (low-risk), "8–15" (increasing risk) and ">15" (high-risk)

strength of associations were consistent with main analyses (Table A3). *Covariates*: all associations from this analysis are fully reported in Table A4. The key results were as follows. Male sex was associated with a two-fold increase in the odds of cannabis (aOR 2.1, 95% CI 1.7–2.6) and a 40% increase for other drug use (aOR 1.4, 95% CI 1.2–1.8). Elevated externalising scores were associated with a 50% increase in the odds of tobacco use (aOR 1.5, 95% CI 1.1–2.0) and high-risk alcohol use (aOR 1.5, 95% CI 1.1–2.2), respectively. Lower parental monitoring was associated with a 30–40% increase in the odds of cannabis (aOR 1.3, 95% CI 1.0–1.7) and tobacco use (aOR 1.4, 95% CI 1.1–1.8), respectively. Lastly, those with friends who used cannabis at 17 had a four- and three-fold increase in

Table 4 Age at first alcoholic drink, AUDIT score at 17 years old and high-risk alcohol, tobacco, cannabis and other drug use at 20 years old examined using survey-weighted multivariable GEE regression models in multiply imputed data (*N* = 4554)

	*High-risk alcohol**		*Tobacco***		*Cannabis****		*Other drugs****	
	aOR (95% CI)	**p**	**aOR (95% CI)**	**p**	**aOR (95% CI)**	**p**	**aOR (95% CI)**	**p**
Age at first alcoholic drink								
≤ 14	Ref		Ref		Ref		Ref	
15	0.88 (0.66–1.18)	0.395	0.92 (0.69–1.23)	0.573	0.95 (0.72–1.24)	0.688	0.61 (0.46–0.82)	0.001
16	0.68 (0.49–0.95)	0.024	0.52 (0.40–0.67)	< 0.001	0.75 (0.58–0.97)	0.027	0.37 (0.28–0.50)	< 0.001
17	0.70 (0.46–1.05)	0.087	0.35 (0.26–0.48)	< 0.001	0.45 (0.32–0.64)	< 0.001	0.21 (0.14–0.30)	< 0.001
≥ 18/no alcohol****	0.19 (0.06–0.55)	0.002	0.08 (0.04–0.15)	< 0.001	0.19 (0.08–0.42)	< 0.001	0.11 (0.05–0.24)	< 0.001
AUDIT score at 17 years old *****								
Low-risk	Ref		Ref		Ref		Ref	
Increasing risk	3.08 (2.25–4.22)	< 0.001	1.94 (1.57–2.39)	< 0.001	1.28 (1.02–1.61)	0.033	1.63 (1.27–2.08)	< 0.001
High-risk	11.45 (7.04–18.63)	< 0.001	2.07 (1.28–3.33)	0.003	1.31 (0.85–2.00)	0.218	2.53 (1.58–4.06)	< 0.001
Tjur's R2******	0.139		0.156		0.191		0.23	
VIF *******	All < 4.25		All < 2.3		All < 3.5		All < 3.35	

AUDIT = Alcohol Use Disorders Identification Test categorised as "0–7" (low-risk), "8–15" (increasing risk) and ">15" (high-risk), GEE = generalised estimating equations, aOR = adjusted odds ratio, CI = confidence intervals, p = p-value, R2 = R square, VIF = variance inflation factor

All models were adjusted for age at outcome, sex, Drumcondra test, extraversion, agreeableness, emotional stability, openness, conscientiousness, SDQ conduct, SDQ hyperactivity, parental monitoring, household social class, household structure, family AUD/drug-use & neighbourhood substance use

*The model examining high-risk alcohol use was additionally adjusted for parental education and peer drunkenness

**The model examining tobacco use was not adjusted for parental education, peer or parental tobacco use due to issues with multicollinearity

***The models examining cannabis and other drug use were additionally adjusted for parental education and peer cannabis use

**** Categories ≥ 18 (7% of participants) and no alcohol use (3%) were collapsed due to small numbers to achieve stable model estimates

***** AUDIT scores were categorised as "0–7" (low-risk), "8–15" (increasing risk) and ">15" (high-risk)

****** Tjur's R2 and VIF estimated for analyses from each of ten imputed data frames and averaged

the odds of cannabis (aOR 3.9, 95% CI 3.0–5.0) and other drug use (aOR 3.0, 95% 2.3–3.8), respectively.

Discussion

Key results

This study used prospective data to examine the relationship between AFD, AUDIT scores at age 17 and high-risk alcohol, tobacco, cannabis and other drug use at age 20, in a large, nationally representative and contemporary cohort. We found that early alcohol use was common, with over a quarter consuming their first alcoholic drink aged 14 or younger. Levels of substance use at 20 years old were also high; 14% reported alcohol use that was classified as high-risk, 38% used tobacco (occasional or daily), 24% cannabis and 28% other drugs.

Older age at first alcohol was associated with reduced probability for each of the four distinct substance use outcomes in early adulthood. Effect estimates decreased substantially and sequentially with increased AFD, suggesting a gradient of risk across adolescence. The strength of the association between AFD and substance use outcomes at age 20 was reduced but remained substantial when adjusted for the individual, family, and neighbourhood covariates. However, the association with high-risk alcohol use at 20 was much reduced when considered alongside AUDIT scores at 17. This is unsurprising as the AUDIT scores were repeated measures, and it was perhaps striking that any association nonetheless remained evident. In our additional files, we include results from a multivariable model without AUDIT scores at 17 which shows a strong and sequential association between AFD and high-risk alcohol use at 20 (Table A4). Overall, our study indicates there is a relationship between AFD and subsequent high-risk alcohol use. Having a higher AUDIT score at age 17 was associated with elevated odds for each substance use outcome, even in multivariable analysis. However, limited statistical support and a small effect size for the outcome of cannabis (Table 4) suggest a weaker relationship between risky adolescent alcohol use and subsequent cannabis use in early adulthood, than the other substances we examined.

As mentioned earlier, existing evidence among population-based cohorts presents conflicting findings on the relationship between AFD and later substance use. Among studies that adjusted for relevant covariates, our results align with five [8, 10–12, 46], but contrast with two [13, 14]. Possible reasons for this divergence could include differences in outcomes examined, lack of statistical power and cultural differences. For instance, Newton-Howes et al. focused solely on substance use disorders (SUDs) diagnosed between ages 15 and 35 in a New Zealand cohort, and found no association after controlling for covariates [14]. It is possible that risk for SUDS, compared to general drug use in early adulthood, is more strongly influenced by "common liability" risk factors– a concept that suggests interacting genetic, environmental and behavioural influences contribute to addiction vulnerability, than by early alcohol use [14, 20]. Rossow et al. examined the outcomes of heavy episodic drinking and AUDIT scores > 8 at age 26–27 in a Norwegian cohort and found no association with early alcohol use, apart from among those with conduct problems [13]. Our sample was much larger which may have afforded us power to detect significant effects. However, culture may also play a significant role. In Ireland, alcohol is widely available and heavy consumption is common at celebrations, sporting events and various other occasions. This cultural norm may contribute to both the levels of early and risky patterns of alcohol use and the associations with substance use in early adulthood that we describe. The strong predictive power of AFD for substance use in early adulthood including dose-response relationships, even after extensive covariate adjustment, suggests a possible causal relationship.

However, these analyses alone cannot establish causality, as there remains the possibility that the associations we observe represent a persistent "common liability" not fully accounted for by our adjustments [20]. Notably, twin studies attempting to disentangle causality have reported conflicting results. One Australian twin study suggested that AFD may have a causal effect on subsequent drunkenness and the onset of regular drinking which in turn affect later-life alcohol problems [47]. Another Australian twin study, found that AFD does not predict later alcohol problems whereas early onset of regular drinking (defined as drinking at least once a month for six of more months) did [48]. A potential explanation for this apparent discordance with our results, could be that in the Irish context, AFD may correspond to the early onset of regular drinking for many, whereas this may not be the case in Australia. However, we lack sufficient data to investigate this further. Additionally, a Norwegian twin study from Norway concluded that the link between early alcohol initiation and later-life AUD is not causal but instead reflects shared genetic risk factors [49]. These varied findings highlight the complexity of disentangling causal pathways.

Implications for policy, practice and future research

Even without robust causal inference, our findings show strong associations between substance use in early adulthood, and early and risky adolescent alcohol use, even in models adjusted for other known risk factors. This suggests that addressing factors associated with early and risky alcohol use during adolescence may reduce substance use and associated harms in early adulthood. While it has been widely reported that alcohol use among adolescents is declining internationally [50], it is notable that 44% still reported first alcohol use at age 15 or younger in this cohort. Furthermore, by age 17, 6% of the study population were already engaging in high-risk alcohol consumption, highlighting that there remains substantial scope for improvement. As young people are at particular risk of alcohol-related harms even with low levels of use [3], this should continue to be an important priority for society. Population-wide initiatives including minimum unit pricing, restricting alcohol advertising and addition of warning labels to alcohol products– key components of the Irish Public Health (Alcohol) Act– may afford some improvement [51]. However, lack of enforcement for measures introduced to date may limit impact [52]. Furthermore, although evaluating this trend was beyond the scope of this study, we note with concern the rapid expansion of marketing for zero-alcohol products in recent years, including the resurgence of alcohol brand name sponsorship at leading international sports fixtures. Given the established causal link between alcohol marketing and youth alcohol use [53], consideration should be given to whether marketing for zero-alcohol products– bearing identical brand names to alcoholic products– warrant similar regulation as alcoholic products.

Strengths

This study has a number of strengths. First, the sample is large and once reweighted is considered nationally representative. Second, the longitudinal design allowed examination of temporality. Third, multiple imputation was employed to manage missing data, avoiding reductions in power and introduction of bias through systematic dropout– both issues commonly associated with listwise deletion. We also repeated analyses in complete case data to check consistency. Fifthly, we adjusted models for a comprehensive set of potential confounders.

Limitations

However, there are also limitations. First, the analysis could be viewed as exploratory as the protocol was not pre-registered, although covariates were selected based on existing literature and availability. Second, the AFD measure was constructed using variables measured at Waves 2–4 in order to harness the earliest reports of first alcohol use. Unfortunately, question and response options changed slightly between Wave 2 and 3 (Methods A1), making it necessary to collapse those with the earliest AFD into a category of ≤ 14, preventing us from examining effects at earlier ages in more detail. Third, like most cohorts, GUI has experienced differential interwave attrition, reweighting was used to account for this. Fourth, we did not examine e-cigarette use as an outcome in this study, which represents a research gap that future studies could address. Fifth, while the results are nationally representative they may not be generalisable to other contexts. Lastly, residual confounding remains possible given the observational study design.

Conclusion

This study used prospective, nationally representative data from Ireland to examine the relationship between age at first alcoholic drink, risky alcohol use in adolescence and subsequent high-risk alcohol, tobacco, cannabis and other drug use at 20. We show strong associations and dose-response effects even in extensively adjusted analyses. Our findings suggest that prevention programmes that delay and reduce alcohol use among adolescents may yield positive effects for alcohol and other drug use in early adulthood. Additionally, they highlight the need for targeted interventions for individuals aged 20, given the high levels of substance use detected at this age.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12889-025-22262-w.

Supplementary Material 1

Acknowledgements
Growing Up in Ireland (GUI) is funded by the Government of Ireland. GUI is managed as a partnership between the Department of Children, Equality, Disability, Integration and Youth (DCEDIY) and the Central Statistics Office (CSO). The CSO are responsible for the survey element of GUI. Results in this report are based on analyses of data from Research Microdata Files provided by the CSO. Neither the CSO nor DCEDIY take any responsibility for the views expressed or the outputs generated from these analyses.

Author contributions
NDM: Funding acquisition, Conceptualization, Supervision, Writing– review and editing. MMB: Conceptualization, Formal Analysis, Writing– original draft. DM: Methodology, Writing– review and editing. AD: Methodology, Writing– review and editing. SRM: Methodology, Writing– review and editing. BG: Methodology, Writing– review and editing. MC: Funding acquisition, Writing– review and editing. LZ: Funding acquisition, Writing– review and editing. BPS: Funding acquisition, Writing– review and editing. EN: Funding acquisition. JHI: Funding acquisition, Writing– review and editing. CM: Funding acquisition, Methodology, Writing– review and editing. CW: Methodology, Writing– review and editing.

Funding
This study was funded by the Health Research Board of Ireland Tender Reference 200344. The Health Research Board (HRB) is a state agency under the Department of Health which supports and funds health and social care research and provides evidence to inform policy and practise. HRB researchers contributed to study design and provided feedback on the draft manuscript.

Data availability
This research has been conducted using anonymised Growing Up in Ireland (GUI) Researcher Microdata Files accessed via the Central Statistics Office (CSO) of Ireland. We do not have permission to share this data but requests to access the data used can be made directly to the CSO. https://www.growingup.gov.ie/information-for-researchers/.

Declarations

Ethics approval and consent to participate
The authors received approval from the Central Statistics Office of Ireland to use the RMF Cohort '98 dataset. The GUI Study received ethical approval from the Research Ethics Committee within the Health Research Board of Ireland (for Wave 1) and the Department of Children, Equality, Disability, Integration and Youth (for Waves 2–4). All participants gave informed consent in the GUI Study. This study is in accordance with the ethical standards as per the Declaration of Helsinki (1964) and subsequent amendments. The present analyses did not require additional ethical approval.

Consent for publication
Not applicable.

Competing interests
BS has received payment for court reports on civil cases involving drug and/or alcohol intoxication and is Vice Chair (unpaid role) of Alcohol Action Ireland, a charity involved in advocacy on alcohol policy. All other authors declare no competing interests.

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Received: 24 January 2025 / Accepted: 10 March 2025
Published online: 24 March 2025

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