



Reciprocal relationships between adolescent mental health difficulties and alcohol consumption

Janet Kiri¹ · James Hall² · Samuele Cortese^{1,3,4,5,6,7} · Valerie Brandt^{1,8}

Received: 18 July 2024 / Accepted: 9 January 2025
© The Author(s) 2025

Abstract

The directionality of the relationship between adolescent alcohol consumption and mental health difficulties remains poorly understood. This study investigates the longitudinal relationship between alcohol use frequency, internalizing and externalizing symptoms from the ages of 11 to 17. We conducted a random-intercept cross-lagged panel model across three timepoints (ages: 11yrs, 14yrs, 17yrs; 50.4% female) in the Millennium Cohort Study ($N=10,647$). Survey weights were used to account for attrition. At each timepoint, past month alcohol use frequency was self-reported, parents and cohort members reported internalizing/externalizing symptoms using the Strengths and Difficulties Questionnaire. We controlled for alcohol expectancies, sex, and four cumulative risk indices (perinatal risk, early childhood adverse parenting, longitudinal parent-level risk occurrence, and persistent household socioeconomic deprivation). More frequent past month alcohol use at age 11 predicted increased internalizing symptoms at age 14 ($\beta=0.06$; $p=.01$). More frequent past month alcohol use at age 14 predicted increased externalizing symptoms at age 17 ($\beta=0.11$; $p<.001$). Increased internalizing symptoms consistently predicted reduced alcohol use at the next timepoint throughout the study period (11 years: $\beta=-0.04$; $p=.03$; 14 years: $\beta=-0.09$; $p<.001$). Increased externalizing symptoms at age 11 predicted increased alcohol consumption at age 14 ($\beta=0.06$; $p=.004$). Frequent adolescent alcohol consumption represents a significant risk for subsequent mental health difficulties. Externalizing symptoms and alcohol use frequency appear to exacerbate one another. Internalizing symptoms may reduce the risk of frequent alcohol consumption. Incorporating routine alcohol screening into adolescent mental health treatment settings could reduce the risk of comorbid externalizing and alcohol use disorders.

Keywords Developmental psychopathology · Alcohol consumption · Mental health difficulties · Random-intercept cross lagged panel model

Introduction

Adolescents are particularly prone to engaging in risky behaviours [1]. For instance, in the UK around 50% of adolescents report experimenting with alcohol, drugs, or

tobacco by the age of 14 [2]. Adolescence also comprises a key developmental period for the emergence of several psychiatric disorders [3], with nearly half of individuals worldwide reporting the onset of symptoms before age 18 [4].

✉ Janet Kiri
jk3g18@soton.ac.uk

¹ School of Psychology, Centre for Innovation in Mental Health, University of Southampton, University Road, Southampton SO17 1BJ, UK

² Southampton Education School, University of Southampton, Southampton, UK

³ Clinical and Experimental Sciences (CNS and Psychiatry), Faculty of Medicine, University of Southampton, Southampton, UK

⁴ Solent NHS Trust, Southampton, UK

⁵ Hassenfeld Children's Hospital at NYU Langone, New York University Child Study Center, New York City, NY, USA

⁶ Division of Psychiatry and Applied Psychology, School of Medicine, University of Nottingham, Nottingham, UK

⁷ DiMePre-J-Department of Precision and Regenerative Medicine-Jonic Area, University of Bari "Aldo Moro", Bari, Italy

⁸ Clinic of Psychiatry, Social Psychiatry and Psychotherapy, Hannover Medical School, Hanover, Germany

Adolescent alcohol consumption and mental health difficulties are intimately related [5]. Frequent adolescent alcohol consumption represents a risk factor for the development of Alcohol Use Disorders (AUDs) and various psychiatric disorders in adulthood [6–8]. Similarly, adolescent mental health difficulties have been found to reflect robust risk factors for a range of psychiatric disorders and AUDs in adulthood [9], suggesting that mental health difficulties and frequent alcohol consumption during adolescence may place individuals at a heightened risk of developing comorbid AUDs and psychiatric disorders later in development [10]. Comorbidity between AUDs and psychiatric disorders, compared to either in isolation, has been linked to more severe symptomatology and functional impairment [11].

However, it remains unclear how alcohol use and mental health difficulties influence each other before adulthood [10]. The few studies that have explicitly investigated the reciprocal relationship between alcohol consumption and mental health difficulties in adolescence have produced mixed findings [12–14]. Furthermore, previous research investigating the temporal sequencing of co-occurring AUDs and psychiatric disorders has often relied on retrospective reports collected after the diagnosis of one or both conditions [10, 15]. Several risk factors, such as prenatal alcohol exposure, negative parenting styles, poor parental mental health, and socioeconomic deprivation, have been implicated in the development of psychiatric disorders and AUDs [16–18]. However, prior longitudinal studies examining the relationship between mental health difficulties and alcohol consumption have either controlled for a limited number of risk factors collected at a single timepoint [14] or have investigated risk factors from a single domain [12]. Clarifying the nature of the relationship between mental health difficulties and alcohol consumption would better inform preventative efforts that could be implemented starting in early adolescence.

Moreover, prospective longitudinal investigations using statistical approaches that separate the stable trait-like differences across individuals (between-person associations) from an individual's fluctuations in alcohol consumption and reported mental health difficulties over time (within-person associations), prior to the emergence of AUDs, may provide insight into the developmental pathways to comorbid AUDs and psychiatric disorders in adulthood. Whilst employing approaches that disassociate within-person from between-person effects does not provide an absolute indication of causality, it facilitates a better understanding of the temporal predominance between mental health difficulties and alcohol consumption during adolescence [19, 20].

The current study examined whether there is a reciprocal relationship between mental health difficulties and

alcohol consumption from the ages of 11 to 17, dissociating within-person from between-person associations, and controlling for shared risk factors at the perinatal, parent, and household level [21]. Using a random-intercept cross-lagged panel model (RI-CLPM), we aimed to clarify the temporal sequencing and directionality of the relationship between mental health difficulties and monthly alcohol use frequency. We hypothesised that there would be significant reciprocal relationships, whereby increases in reported mental health difficulties would precede increases in alcohol consumption (and vice versa) across the study period.

The current study used data from the British Millennium Cohort Study (MCS) which follows a sample of around $N=19,500$ children (and their families) since their birth in 2000–2001. Detailed data collection, sampling and stratification procedures have been described elsewhere [22]. There were seven waves of data collection: at 9 months (T1), 3 years (T2), 5 years (T3), 7 years (T4), 11 years (T5), 14 years (T6), and 17 years (T7). Information was collected on a range of topics including mental health, finances, and parent-child relationships. Parents provided written informed consent at each timepoint for the participation of them and their child and for the data to be made available for secondary data analysis through the UK Data Archive: <https://www.data-archive.ac.uk/>. Ethical approval for this secondary data analysis was granted by the University of Southampton ethics committee (ERGO: 79894.A1).

Methods

Participants

The final analytical sample comprised $N=10,647$ participants (50.4% female; 79.2% Caucasian). Inclusion/exclusion criteria are described in detail in Supplementary Figure S1. Participants who did not complete any alcohol or mental health measures or lacked survey weights at T7 were excluded from the analytical sample. In line with previous investigations of substance use in the MCS [23], participants who reported use of the fake drug “Semeron” at T7, were excluded.

Primary measures

Alcohol use frequency was self-reported for the previous 30 days at each timepoint on the scale (0) “Never”, (1) “1–2 times”, (2) “3–5 times”, (3) “6–9 times”, (4) “10–19 times”, (5) “20–39 times”, and (6) “40 or more times”. Due to the low volume of responses in some categories, responses were condensed into three categories: (0) “Never”, (1) “1–2 times per month”, and (2) “more than 3 times a month”

[24]. Higher scores reflect more frequent monthly alcohol consumption.

Internalizing and externalizing symptoms were assessed by parent- and self-report, using the Strengths and Difficulties Questionnaire (SDQ) at each timepoint. The parent-report was used at ages 11 and 14, whereas the self-report was used at age 17 (see Supplementary Table S1 for further details). The SDQ has demonstrated clinical utility for predicting psychiatric disorders in normative samples [25]. For our purposes, internalizing symptoms were measured using the emotional problems subscale (range: 0–10), externalizing symptoms were measured with the hyperactivity/inattention and conduct problems subscales (range: 0–20). Higher scores indicate a greater number of symptoms.

Covariates

To evaluate the influence of multiple risks that have been implicated in the development of AUDs and mental health difficulties, salient risk factors collected throughout the cohort member's childhood were divided by ecological level (child, parent and household). The combined risk at each level was assessed via cumulative risk indices (CRIs) that accounted for the developmental timing of risk exposure (for detailed variable information see Supplementary Tables S1 and S2). Individual risks were dichotomized to reflect whether the cohort member reported (0) "no risk exposure" or (1) "risk exposure", with CRI scores reflecting the number of risks encountered. CRIs involving risks collected across multiple timepoints were computed for participants with data on over 50% of respective indicators across multiple waves, while CRIs compiled of variables assessed at a single timepoint were computed for those with data on over 25% of respective indicators [26]. CRIs included the following: perinatal CRI, early childhood (EC) adverse parenting, longitudinal parent-level risk occurrence, and persistent household socioeconomic deprivation (SED). Additional details for the variables included in each CRI, alongside the computation of each CRI, are available in the Supplementary Material (see Supplementary Tables S1–S2). Self-reported alcohol expectancies at age 11, which have been implicated in the development of problematic alcohol use behaviours during adolescence and early adulthood [27], and the participant's parent-reported biological sex were also controlled for (see Supplementary Table S1 for further details).

Table 1 presents an overview of the characteristics and differences between participants with at least one missing value on mental health difficulties or alcohol use, compared to participants with complete data across the three timepoints. There were significant differences between the complete and missing samples on all variables - except for

monthly alcohol use at age 11 and internalizing symptoms at age 17 - with small to modest effect sizes (Cramer's $V \leq 0.16$, Cohen's $d \leq 0.41$). A full correlation matrix is presented in Supplementary Table S3.

Statistical analysis

The analysis was conducted the "lavaan" package in the R environment [28].

A random-intercept cross-lagged panel model (RI-CLPM) [19] was employed to explore the dynamic relationship between monthly alcohol use, internalizing, and externalizing symptoms across T5–T7, controlling for sex, alcohol expectancies and salient cumulative risk factors. RI-CLPM demonstrates significant advantages over the traditional cross-lagged panel models used in previous research [12–14], through including random intercepts which enables the delineation of between-person from within-person variances [19]. Additional details regarding the RI-CLPM are available in the Supplementary Materials (see Supplement 1).

A trivariate (monthly alcohol use, internalizing and externalizing symptoms) RI-CLPM was conducted to adjust for the high co-occurrence between internalizing and externalizing symptoms. This approach inherently accounts for the shared variance between monthly alcohol consumption, internalizing, and externalizing symptoms at both the between-person and within-person level, enabling an exploration of the dynamic associations between these constructs throughout adolescence. Figure 1 present a conceptual diagram of the unconditional trivariate model [29]. The following predefined thresholds were used to assess model fit; Comparative Fit Index (CFI; good fit ≥ 0.95), Tucker-Lewis Index (TLI; acceptable fit ≥ 0.90 , good fit ≥ 0.95), Standardized Root Mean Square Residual (SRMR; good fit ≤ 0.08) and Root Mean Square Error of Approximation (RMSEA; good fit < 0.05) [30].

Contemporary bivariate correlations among alcohol use, externalizing, and internalizing symptoms were estimated. Attrition and the clustered sampling design of the MCS sample were addressed with survey weights. Full Information Maximum Likelihood handled missing data, and due to skewed variables, Maximum Likelihood Estimation with Robust Standard Errors (MLR) was employed.

Conceptual Diagram of the Trivariate Random-Intercept Cross-Lagged Panel Model. T5: timepoint five (same pattern for subsequent timepoints). Cross-lagged and autoregressive paths (solid black lines), contemporaneous correlations at each timepoint (dashed black lines), between-person associations between the random-intercepts (dashed grey lines) are shown. All other paths are represented with solid grey lines for ease of interpretation.

Table 1 Characteristics and differences between sample with complete data on mental health difficulties and monthly alcohol use ($n=7172$) and sample with at least one missing value ($n=3475$)

	Complete data ($n=7172$)		Missing data ($n=3475$)		Chi-square (df)	P value	Effect size
	N (%)	Mean (SD)	N (%)	Mean (SD)			
Sex					48.23 (1)	<0.001	0.07 ^b
Male	3403 (34.6%)	-	1476 (15.0%)	-	-	-	-
Female	3769 (38.3%)	-	1192 (12.1%)	-	-	-	-
Ethnicity					242.07 (5)	<0.001	0.16 ^b
White	5901 (61.6%)	-	1695 (17.7%)	-	-	-	-
Mixed	323 (3.4%)	-	128 (1.3%)	-	-	-	-
Black	193 (2.0%)	-	118 (1.2%)	-	-	-	-
Indian	169 (1.8%)	-	103 (1.1%)	-	-	-	-
Pakistan	421 (4.4%)	-	307 (3.2%)	-	-	-	-
Other	125 (1.3%)	-	103 (1.1%)	-	-	-	-
Monthly alcohol use							
Age 11					1.02 (2)	0.60	0.01 ^b
Never	6958 (71.6%)	-	2464 (25.3%)	-	-	-	-
1–2 times	182 (1.9%)	-	69 (0.7%)	-	-	-	-
3 or more times	32 (0.3%)	-	15 (0.2%)	-	-	-	-
Age 14					16.62 (2)	<0.001	0.04 ^b
Never	5611 (60.9%)	-	1681 (18.2%)	-	-	-	-
1–2 times	1143 (12.4%)	-	257 (2.8%)	-	-	-	-
3 or more times	418 (4.5%)	-	104 (1.1%)	-	-	-	-
Age 17					167.13 (2)	<0.001	0.13 ^b
Never	2553 (26.4%)	-	1249 (12.9%)	-	-	-	-
1–2 times	2310 (23.9%)	-	697 (7.2%)	-	-	-	-
3 or more times	2309 (23.9%)	-	562 (5.8%)	-	-	-	-
Internalizing symptoms							
Age 11	7172 (75.1%)	1.73 (1.90)	2384 (24.9%)	2.04 (2.13)	6.25 (3727) ^a	<0.001	0.16 ^c
Age 14	7172 (75.1%)	1.92 (2.08)	2379 (24.9%)	2.32 (2.24)	7.77 (3833) ^a	<0.001	0.19 ^c
Age 17	7172 (72.9%)	3.51 (2.45)	2665 (27.1%)	3.43 (2.46)	1.45 (9835) ^a	0.15	0.03 ^c
Externalizing symptoms							
Age 11	7172 (76.8%)	3.98 (3.29)	2161 (23.2%)	5.09 (3.84)	12.19 (3173) ^a	<0.001	0.33 ^c
Age 14	7172 (75.2%)	3.92 (3.31)	2370 (24.8%)	5.32 (3.86)	15.88 (3586) ^a	<0.001	0.41 ^c
Age 17	7172 (72.9%)	5.54 (3.27)	2664 (27.1%)	5.81 (3.35)	3.57 (9834) ^a	<0.001	0.08 ^c

^aIndependent Samples t-test. ^bCramer's V. ^cCohen's d

Results

Longitudinal relationship between monthly alcohol use, internalizing and externalizing symptoms

The RI-CLPM model exhibited good fit ($\chi^2(31)=306.91$, $P<.001$; RMSEA=0.045 [0.04–0.05]; CFI=0.97; TLI=0.90; SRMR=0.04).

Increased internalizing symptoms at age 11 predicted reduced alcohol use at the next timepoint ($\beta=-0.04$; $SE=0.02$; 95% CI, -0.08 to -0.004; $P=.03$), as did increased symptoms at age 14 ($\beta=-0.09$; $SE=0.02$; 95% CI, -0.12 to -0.05; $P<.001$). Elevated internalizing symptoms at age 11 were associated with increased externalizing symptoms at age 14 ($\beta=0.04$; $SE=0.02$; 95% CI, 0.003–0.08; $P=.046$). The same path between ages 14–17 was not significant (Fig. 2).

Elevated externalizing symptoms at age 11 were associated with increased monthly alcohol use ($\beta=0.06$; $SE=0.02$; 95% CI, 0.02–0.10; $P=.004$) and internalizing symptoms ($\beta=0.12$; $SE=0.02$; 95% CI, 0.07–0.16; $P<.001$) at age 14. The same paths between ages 14–17 were not significant (Fig. 2).

The Cross-Lagged and Autoregressive Paths Between Alcohol Use and Internalizing/Externalizing Symptoms. This figure shows the paths ($*p<.05$, $**p<.001$), between monthly alcohol use frequency and internalizing/externalizing symptoms (presented in black) and between internalizing and externalizing symptoms (presented in grey). Solid lines represent significant paths. Dashed lines represent non-significant paths. Standardized estimates with 95% confidence intervals are presented for significant paths only.

Elevated alcohol use frequency at age 11 predicted increased internalizing symptoms at age 14 ($\beta=0.06$;

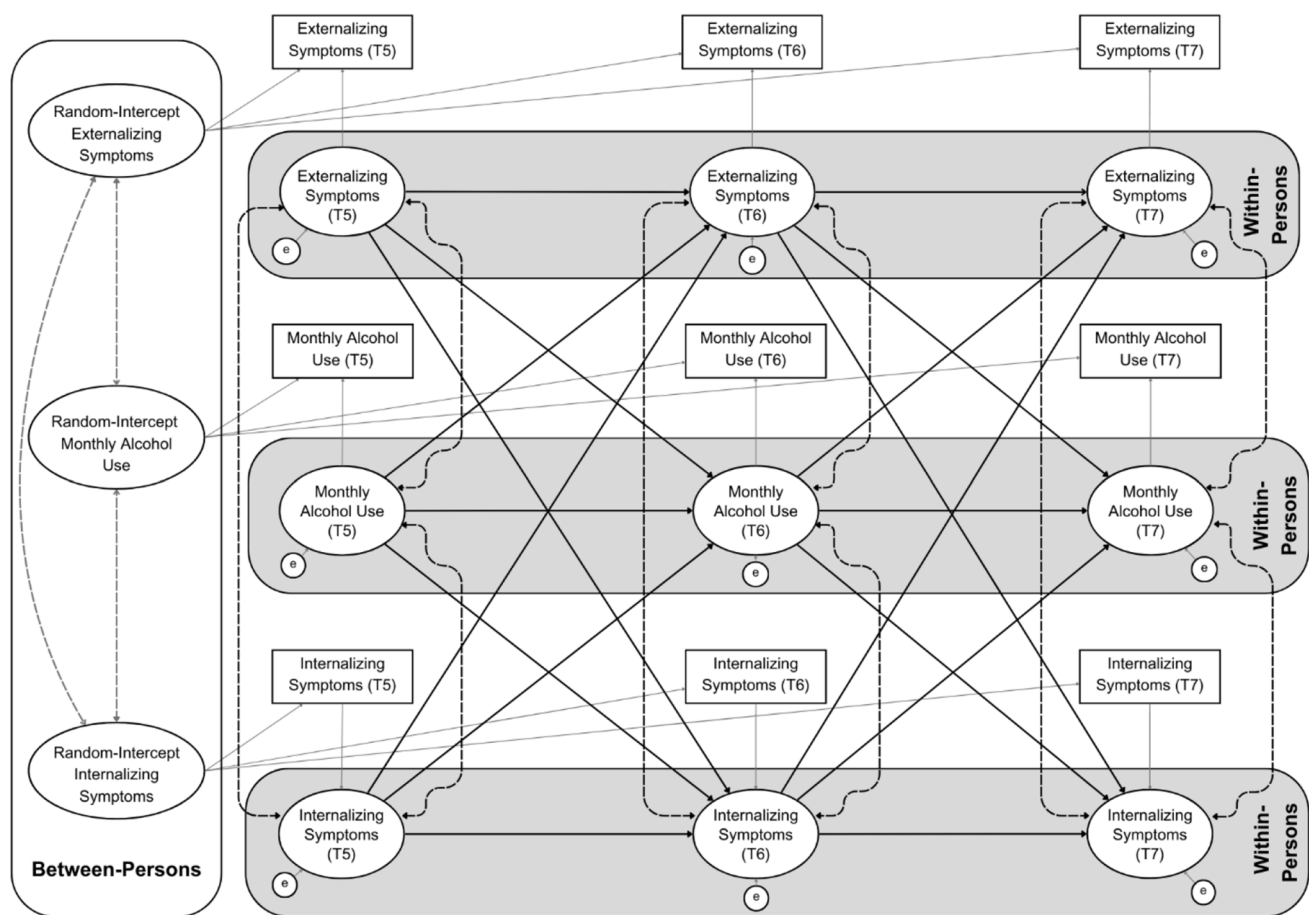


Figure. 1 Conceptual diagram of the trivariate random-intercept cross-lagged panel model. T5: timepoint five (same pattern for subsequent timepoints). Cross-lagged and autoregressive paths (solid black lines), contemporaneous correlations at each timepoint (dashed black lines),

between-person associations between the random-intercepts (dashed grey lines) are shown. All other paths are represented with solid grey lines for ease of interpretation

$SE=0.02$; 95% CI, 0.01–0.11; $P=.01$), and increased monthly alcohol use at age 14 predicted increased externalizing symptoms at age 17 ($\beta=0.11$; $SE=0.02$; 95% CI, 0.08–0.15; $P<.001$). Monthly alcohol use at age 11 was not significantly associated with any changes in externalizing symptoms at age 14, nor was alcohol use at age 14 significantly associated with changes in internalizing symptoms at age 17 (Fig. 2).

Within-person contemporaneous correlations and associations between random intercepts, reflecting between-person trait-like differences, are reported in Table 2. The trivariate RI-CLPM showed positive significant autoregressive paths for internalizing and externalizing symptoms across ages 11 to 17, with diminishing carry-over effects over time. Interestingly, the monthly alcohol use autoregressive path was significant from ages 14 to age 17, but not between ages 11 and 14, indicating that stability in monthly alcohol use occurs in late, but not in early adolescence (Table 2).

Influence of perinatal, parent and household-level CRIs

Table 3 and Supplementary Figures S2–S8 present all results of the conditional RI-CLPM model.

Perinatal CRI and early childhood adverse parenting showed no significant association with monthly alcohol use at any timepoint but did predict increased externalizing symptoms across all ages (Table 3, Supplementary Figures S2 and S3). Additionally, perinatal CRI predicted increased internalizing symptoms at age 17, whilst early childhood adverse parenting predicted increased internalizing symptoms at ages 11 and 14. Longitudinal parent-level risk occurrence was associated with elevated monthly alcohol use at ages 14 and 17, and increased internalizing and externalizing symptoms across all ages (Table 3, Supplementary Figure S4). Persistent household socioeconomic deprivation was associated with reduced monthly alcohol use at ages 14 and 17 (Table 3, Supplementary Figure S5). It showed

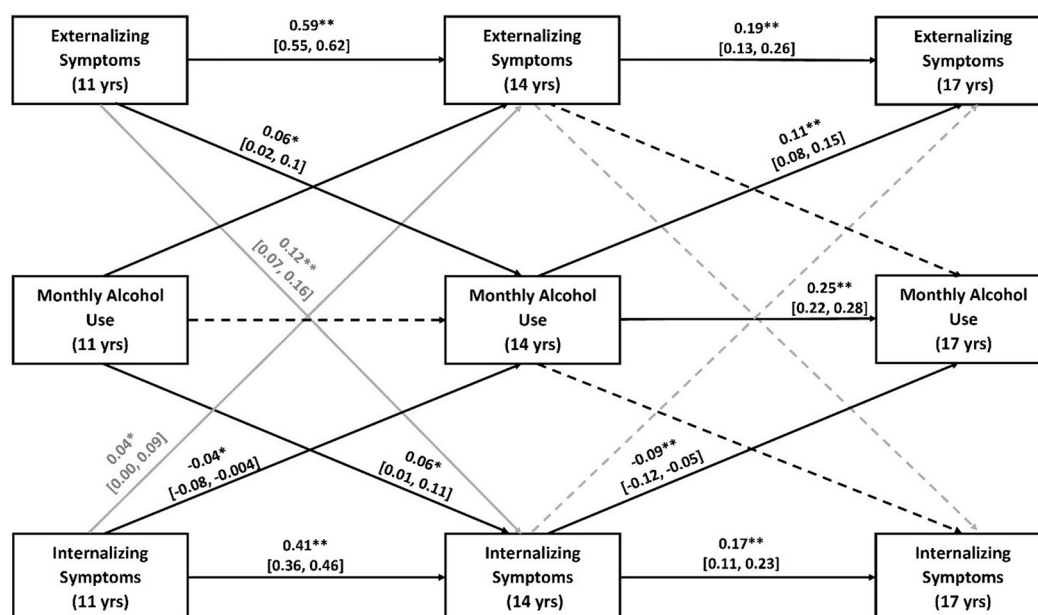


Figure. 2 The cross-lagged and autoregressive paths between alcohol Use and internalizing/externalizing symptoms. This figure shows the paths (* $p < .05$, ** $p < .001$), between monthly alcohol use frequency and internalizing/externalizing symptoms (presented in black) and

between internalizing and externalizing symptoms (presented in grey). Solid lines represent significant paths. Dashed lines represent non-significant paths. Standardized estimates with 95% confidence intervals are presented for significant paths only

Table 2 The within-person contemporaneous correlations and between-person associations between Random intercepts

	<i>r</i>	SE	95% CI	
			Lower	Upper
Within-person contemporaneous correlations				
<i>Age 11</i>				
Monthly alcohol use~internalizing symptoms	0.11*	0.04	0.04	0.18
Monthly alcohol use~externalizing symptoms	0.06*	0.03	0.01	0.12
Internalizing symptoms~externalizing symptoms	0.38**	0.03	0.32	0.43
<i>Age 14</i>				
Monthly alcohol use~internalizing symptoms	0.01	0.02	-0.03	0.05
Monthly alcohol use~externalizing symptoms	0.09**	0.02	0.05	0.13
Internalizing symptoms~externalizing symptoms	0.30**	0.02	0.26	0.33
<i>Age 17</i>				
Monthly alcohol use~internalizing symptoms	-0.03	0.02	-0.06	0.001
Monthly alcohol use~externalizing symptoms	0.13**	0.02	0.10	0.16
Internalizing symptoms~externalizing symptoms	0.36**	0.02	0.33	0.40
Between-person associations				
Monthly alcohol use~internalizing symptoms	-0.28*	0.09	-0.45	-0.10
Monthly alcohol use~externalizing symptoms	-0.01	0.07	-0.15	0.13
Internalizing symptoms~externalizing symptoms	0.24	0.14	-0.03	0.51

* $p < .05$. ** $p < .001$

a positive association with internalizing symptoms at ages 11 and 14, and with externalizing symptoms across all ages.

Influence of sex and adolescent alcohol expectancies

Boys reported significantly more frequent monthly alcohol use than girls at age 11 (Table 3, Supplementary Figure S6).

They also reported lower levels of internalizing, and higher levels of externalizing symptoms, across all ages. Positive alcohol expectancies were associated with elevated monthly alcohol use across all ages (Table 3, Supplementary Figure S7). Negative alcohol expectancies were associated with reduced monthly alcohol use at age 11 (Table 3, Supplementary Figure S8).

Table 3 The direct effects of sex, CRIs and alcohol expectancies on monthly alcohol use, internalizing and externalizing symptoms

	Monthly alcohol use				Internalizing symptoms				Externalizing symptoms			
	β	SE	95% CI		β	SE	95% CI		β	SE	95% CI	
			Lower	Upper			Lower	Upper			Lower	Upper
Age 11												
Male	0.04*	0.01	0.01	0.06	-0.07**	0.02	-0.10	-0.04	0.13**	0.02	-0.10	0.16
Perinatal CRI	0.01	0.02	-0.02	0.04	0.02	0.02	-0.01	0.05	0.05*	0.02	0.02	0.08
Longitudinal parent-pevel risk occurrence	0.02	0.02	-0.02	0.05	0.11**	0.02	0.08	0.14	0.15**	0.02	0.12	0.18
Persistent household SED	0.01	0.02	-0.02	0.04	0.08**	0.02	0.05	0.12	0.12**	0.01	0.10	0.15
Early childhood adverse parenting	-0.02	0.02	-0.05	0.01	0.14**	0.02	0.10	0.17	0.25**	0.02	0.22	0.28
Positive alcohol expectancies	0.11**	0.02	0.08	0.15	0.04*	0.02	0.01	0.07	0.02	0.02	-0.01	0.05
Negative alcohol expectancies	-0.06**	0.01	-0.09	-0.03	-0.01	0.01	-0.04	0.01	-0.05**	0.01	-0.08	-0.02
Age 14												
Male	-0.02	0.02	-0.05	0.010	-0.16**	0.02	-0.19	-0.13	0.12**	0.02	0.09	0.15
Perinatal CRI	0.01	0.02	-0.02	0.04	0.02	0.02	-0.01	0.05	0.06**	0.01	0.03	0.09
Longitudinal parent-level risk occurrence	0.09**	0.02	0.05	0.12	0.11**	0.02	0.08	0.14	0.14**	0.01	0.12	0.17
Persistent household SED	-0.05**	0.01	-0.08	-0.03	0.10**	0.02	0.07	0.13	0.13**	0.01	0.11	0.16
Early childhood adverse parenting	0.01	0.02	-0.03	0.04	0.11**	0.02	0.08	0.14	0.20**	0.02	0.17	0.23
Positive alcohol expectancies	0.09**	0.01	0.07	0.12	-0.002	0.02	-0.03	0.03	-0.002	0.02	-0.03	0.03
Negative alcohol expectancies	-0.02	0.01	-0.05	0.01	-0.03	0.01	-0.06	0.00	-0.04*	0.01	-0.07	-0.02
Age 17												
Male	0.02	0.01	-0.01	0.04	-0.34**	0.01	-0.37	-0.31	0.09**	0.02	0.06	0.13
Perinatal CRI	0.02	0.02	-0.01	0.05	0.03*	0.01	0.00	0.06	0.06**	0.02	0.03	0.09
Longitudinal parent-level risk occurrence	0.06**	0.02	0.03	0.10	0.06**	0.02	0.03	0.09	0.10**	0.01	0.07	0.13
Persistent household SED	-0.18**	0.01	-0.20	-0.15	0.02	0.01	0.00	0.05	0.05**	0.01	0.02	0.07
Early childhood adverse parenting	-0.03	0.02	-0.06	0.00	0.01	0.01	-0.02	0.03	0.09**	0.01	0.06	0.11
Positive alcohol expectancies	0.09**	0.02	0.05	0.12	0.03*	0.01	0.00	0.06	0.07**	0.02	0.03	0.10
Negative alcohol expectancies	0.01	0.02	-0.03	0.04	0.01	0.01	-0.02	0.04	0.01	0.02	-0.02	0.04

* $p < .05$. ** $p < .001$

Discussion

To our knowledge, the current study is the first to uncover a reciprocal relationship between adolescent mental health difficulties and frequent alcohol consumption between 11 and 17 years. We found a significant reciprocal association between more externalizing symptoms and more frequent monthly alcohol use from the ages of 11 to 17, providing novel evidence of links between alcohol use and externalizing disorders already during adolescence.

More specifically, we found that increased externalizing symptoms in early adolescence (age 11) predicted increased alcohol use at ages 14–17, which in turn predicted elevated externalizing symptoms at age 17. These results are consistent with, and expand upon previous findings showing that externalizing symptoms represent a risk factor for increased alcohol use in adulthood [9]. Hence, the findings lend support to the externalizing pathway to comorbid AUDs and externalizing disorders, which suggests that the behavioural disinhibition often associated with externalizing symptoms increases adolescents' propensity for engaging in deviant behaviour, like underage drinking [31]. Furthermore, while little research has explored the possible underlying mechanisms of alcohol as a risk factor for externalizing symptoms, available studies have shown that adolescents carrying a polymorphism of the aldehyde dehydrogenase 2 (ALDH2) gene commonly associated with reduced alcohol consumption [32], were also less likely to report aggressive behaviour or attentional deficits during adolescence [33]. Therefore, our findings lend further support to theoretical models positing that the potentiated neurotoxic effects of alcohol on the developing adolescent brain might elicit neuroadaptations in regions implicated in the pathogenesis of mental health difficulties [34]. Overall, our results suggest that externalizing symptomatology and alcohol consumption serve to maintain and/or exacerbate one another throughout adolescence.

Our results also showed a reciprocal relationship between internalizing symptoms and alcohol use from the ages of 11 to 17. While increased monthly alcohol use during early adolescence (11–14yrs) predicted more internalizing symptoms at age 14, more internalizing symptoms predicted reduced monthly alcohol consumption across adolescence. This is in line with previous research linking adolescent alcohol consumption, even at subclinical levels, to an increased risk for developing depressive symptoms in adulthood [8]. Our results expand upon previous research in the field by showing that the link between alcohol consumption and internalizing symptomatology already exists in adolescence. Conversely, the finding that more internalizing symptoms consistently predicted a reduced likelihood of engaging in frequent alcohol consumption contradicted

our expectations. Previous studies show mixed results on the relationship between internalizing disorders and alcohol consumption [12, 14], and this may be due to the observed relationship between higher internalizing and higher externalizing symptoms [29]. It is possible that when externalizing symptoms are controlled for, internalizing symptoms are related to reduced alcohol consumption. In support of this, Nurnberger and colleagues [35], found that adolescent externalizing disorders predicted an earlier onset of AUD in early adulthood. However, regarding internalizing disorders, this association was only significant in the presence of a co-occurring externalizing disorder. As adolescent drinking often occurs in social contexts with peers [36], it is possible that the elevated levels of social withdrawal associated with internalizing symptoms [37], may inadvertently reduce social opportunities for frequent alcohol consumption. It is plausible that the motivation to drink to cope with negative emotionality, hypothesized to underlie the increased risk of AUD resulting from internalizing symptoms, only develops in adulthood, rather than during the initiation/escalation of alcohol use during adolescence [34]. Thus, disparities in previous research findings may in part be due to the influence of developmental timing on the temporal relationship between internalizing symptoms and alcohol use. In support of this, research suggests that the protective influence of internalizing symptoms diminishes with age [38].

In terms of the risk factors we controlled for, we found that exposure to more parental risk factors, such as parental alcohol or drug consumption, domestic violence, or poor parental mental health before 11 years was significantly associated with higher levels of adolescent alcohol use and mental health difficulties, consistent with existing literature [39, 40]. Interestingly, adolescents from higher socioeconomic backgrounds were more likely to report frequent alcohol use, consistent with research conducted in this age group in other British samples [41].

Also, and in line with previous findings (see Smit et al. for a review) [27], positive alcohol expectancies, such as the belief in enhanced confidence and sociability, during early adolescence, predicted increased alcohol use across all ages. In contrast, negative expectancies, such as the belief that drinking hinders schoolwork, only predicted reduced alcohol use at age 11. Overall, the findings underscore the crucial role of positive alcohol expectancies as a modifiable risk factor for the initiation/escalation of underage drinking throughout adolescence. Additionally, in accordance with the literature, boys reported more frequent monthly alcohol use at age 11 [42]. Boys also reported higher levels of externalizing, and lower levels of internalizing symptoms across all ages [43], compared to girls.

Limitations

The current study relied on a single-item measure of alcohol use frequency. This has been found to be effective method of screening for problematic adolescent alcohol consumption [44]. However, while previous research shows that frequent adolescent alcohol consumption reflects a risk factor for subsequent AUDs and psychiatric disorders in adulthood [6–8], the relationship between adolescent alcohol use and mental health difficulties may differ depending on the dimension of adolescent drinking behaviour that was measured [45]. Thus, future research should explore other dimensions, such as the frequency of heavy episodic drinking, for a more nuanced understanding of the temporal relationship between various facets of adolescent drinking behaviour and mental health difficulties.

Implications

The current findings emphasize the significance of adolescent alcohol use as a risk factor for subsequent mental health difficulties, indicating that early screening in adolescence followed by preventative interventions against underage drinking also may ameliorate the risk of future mental health difficulties. Screening for externalizing disorders in childhood and early adolescence may enable the early identification of adolescents at a higher risk of engaging in frequent underage drinking. Targeted interventions to address externalizing symptomatology prior to alcohol initiation may also diminish the risk of underage drinking. Initial evidence in the field of attention-deficit/hyperactivity disorder (ADHD) research may inform such strategies. Indeed, stimulant medications for children with ADHD have been found to at reduce both externalizing symptomatology and the risk of future substance use [46].

Additionally, evidence from this study may inform future strategies aimed at preventing the development of comorbid AUDs and externalizing disorders. The interconnected nature of externalizing symptoms and alcohol use during adolescence point to the need for a unified approach. Alcohol screening and brief intervention (SBI) has been shown as a cost-effective intervention with demonstrated efficacy for reducing adolescent alcohol consumption [47]. Therefore, incorporating SBI into adolescent mental health treatment settings could facilitate the early identification and referral of adolescents with high levels of externalizing symptoms and problematic alcohol consumption to substance abuse treatment services. This approach may help to reduce the risk of future comorbid AUDs and externalizing disorders in early adulthood.

Conclusions

Our findings revealed that frequent adolescent alcohol use posed a risk for both higher externalizing and internalizing symptoms, while higher internalizing symptoms were associated with less frequent alcohol use across adolescence. Additionally, the study extends the existing evidence implicating externalizing symptoms as a risk factor for frequent alcohol consumption in adolescence by uncovering the existence of a reciprocal relationship between externalizing symptoms and alcohol use frequency. Overall, our findings provide a strong rationale for additional research assessing the implementation of routine screening, followed by an appropriate evidence-based intervention to reduce alcohol consumption, for adolescents presenting to mental health services as an effective way to prevent AUDs and psychiatric conditions in adulthood.

Supplementary Information The online version contains supplementary material available at <https://doi.org/10.1007/s00787-025-02644-6>.

Author contributions Author Contributions J.K., J.H. and V.B. contributed to the conceptualization of the research design. J.K. contributed to the formal analysis, interpretation and writing of the original draft. J.K., J.H., S.C. and V.B. contributed to the methodology. J.H., S.C. and V.B. supervised the work. J.K., J.H., S.C. and V.B. contributed to reviewing and editing the final manuscript.

Funding No funding was received towards this work.

Data availability The Millennium Cohort Study dataset can be accessed via the UK Data Service. The R script and output for the RI-CLPM and the SPSS syntax needed to compute the CRI variables are available on Pure.

Declarations

Ethical approval Ethical approval for this secondary data analysis was granted by the University of Southampton ethics committee (ERGO: 79894.A1).

Consent to participate Parents provided written informed consent at each timepoint for the participation of them and their child and for the data to be made available for secondary data analysis through the UK Data Archive: <https://www.data-archive.ac.uk/>.

Competing interests The authors declare no competing interests.

Open Access This article is licensed under a Creative Commons Attribution 4.0 International License, which permits use, sharing, adaptation, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if changes were made. The images or other third party material in this article are included in the article's Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons licence and your intended use is not permitted by statutory regulation or exceeds the permitted

use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit <http://creativecommons.org/licenses/by/4.0/>.

References

- Steinberg L (2008) A Social Neuroscience Perspective on adolescent risk-taking. *Dev Rev* 28:78–106
- Fitzsimons E, Jackman J, Kyprianides A, Villadsen A (2018) Determinants of risky behaviours in adolescence: evidence from the UK Centre for Longitudinal studies. In: Centre for Longitudinal Studies
- Gilmore KJ, Meersand P (2014) Normal child and adolescent development: a psychodynamic primer. American Psychiatric Publishing, Inc
- Solmi M, Radua J, Olivola M, Croce E, Soardo L, Salazar de Pablo G, Il Shin J, Kirkbride JB, Jones P, Kim JH, Kim JY, Carvalho AF, Seeman MV, Correll CU, Fusar-Poli P (2022) Age at onset of mental disorders worldwide: large-scale meta-analysis of 192 epidemiological studies. *Mol Psychiatry* 27:281–295
- Puddephatt JA, Irizar P, Jones A, Gage SH, Goodwin L (2022) Associations of common mental disorder with alcohol use in the adult general population: a systematic review and meta-analysis. *Addiction* 117:1543–1572
- Bonomo YA, Bowes G, Coffey C, Carlin JB, Patton GC (2004) Teenage drinking and the onset of alcohol dependence: a cohort study over seven years. *Addiction* 99:1520–1528
- Maldonado-Molina MM, Reingle JM, Jennings WG (2011) Does Alcohol Use Predict violent behaviors? The relationship between Alcohol Use and Violence in a nationally Representative Longitudinal Sample. *Youth Violence Juvenile Justice* 9:99–111
- Pedrelli P, Shapero B, Archibald A, Dale C (2016) Alcohol use and depression during adolescence and young adulthood: a summary and interpretation of mixed findings. *Curr Addict Rep* 3:91–97
- Meque I, Dachew BA, Maravilla JC, Salom C, Alati R (2019) Externalizing and internalizing symptoms in childhood and adolescence and the risk of alcohol use disorders in young adulthood: a meta-analysis of longitudinal studies. *Aust N Z J Psychiatry* 53:965–975
- Castillo-Carniglia A, Keyes KM, Hasin DS, Cerdá M (2019) Psychiatric comorbidities in alcohol use disorder. *Lancet Psychiatry* 6:1068–1080
- Gadermann AM, Alonso J, Vilagut G, Zaslavsky AM, Kessler RC (2012) Comorbidity and disease burden in the National Comorbidity Survey replication (NCS-R). *Depress Anxiety* 29:797–806
- Jun HJ, Sacco P, Bright CL, Camlin EA (2015) Relations among internalizing and externalizing symptoms and drinking frequency during adolescence. *Subst Use Misuse* 50:1814–1825
- Murray AL, Eisner M, Ohsuth I, Ribeaud D (2017) No evidence that Substance Use causes ADHD symptoms in adolescence. *J Drug Issues* 47:405–410
- Schleider JL, Ye F, Wang F, Hipwell AE, Chung T, Sartor CE (2019) Longitudinal reciprocal associations between anxiety, Depression, and Alcohol Use in adolescent girls. *Alcoholism: Clin Experimental Res* 43:98–107
- Brière FN, Rohde P, Seeley JR, Klein D, Lewinsohn PM (2014) Comorbidity between major depression and alcohol use disorder from adolescence to adulthood. *Compr Psychiatry* 55:526–533
- Chassin L, Colder CR, Hussong A, Sher KJ (2016) Substance use and substance use disorders. Developmental psychopathology: Maladaptation and psychopathology, vol 3, 3rd edn. John Wiley & Sons, Inc., Hoboken, NJ, US, pp 833–897
- Lynch SJ, Sunderland M, Newton NC, Chapman C (2021) A systematic review of transdiagnostic risk and protective factors for general and specific psychopathology in young people. *Clin Psychol Rev* 87:102036
- Moggi F (2005) Etiological theories on the relationship of Mental disorders and Substance Use disorders. In: Rössler W, Stohler R (eds) Dual diagnosis: the evolving conceptual Framework. S.Karger AG, p 0
- Hamaker EL, Kuiper RM, Grasman RP (2015) A critique of the cross-lagged panel model. *Psychol Methods* 20:102–116
- Littlefield AK, King KM, Acuff SF, Foster KT, Murphy JG, Witkiewitz K (2022) Limitations of cross-lagged panel models in addiction research and alternative models: an empirical example using project MATCH. *Psychol Addict Behav* 36:271–283
- Arango C, Dragioti E, Solmi M, Cortese S, Domschke K, Murray RM, Jones PB, Uher R, Carvalho AF, Reichenberg A, Shin JI, Andreassen OA, Correll CU, Fusar-Poli P (2021) Risk and protective factors for mental disorders beyond genetics: an evidence-based atlas. *World Psychiatry* 20:417–436
- Joshi H, Fitzsimons E (2016) The Millennium Cohort Study: the making of a multi-purpose resource for Social Science and policy. *Longitud Life Course Stud* 7:409–430
- Reyes BD, Hargreaves DS, Creese H (2021) Early-life maternal attachment and risky health behaviours in adolescence: findings from the United Kingdom Millennium Cohort Study. *BMC Public Health*: 1–11
- Purba AK, Henderson M, Baxter A, Katikireddi SV, Pearce A (2023) The relationship between time spent on social media and adolescent alcohol use: a longitudinal analysis of the UK Millennium cohort study. *European Journal of Public Health:Advance online publication*
- Goodman R (2001) Psychometric properties of the strengths and difficulties questionnaire. *J Am Acad Child Adolesc Psychiatry* 40:1337–1345
- Hogye SI, Lucassen N, Jansen PW, Schuurmans IK, Keizer R (2022) Cumulative risk and internalizing and externalizing problems in early childhood: compensatory and buffering roles of family functioning and family regularity. *Adversity Resil Sci* 3:149–167
- Smit K, Voogt C, Hiemstra M, Kleinjan M, Otten R, Kuntsche E (2018) Development of alcohol expectancies and early alcohol use in children and adolescents: a systematic review. *Clin Psychol Rev* 60:136–146
- Rossee Y (2012) Lavaan: an R Package for Structural equation modeling. *J Stat Softw* 48:1–36
- Achenbach TM, Ivanova MY, Rescorla LA, Turner LV, Althoff RR (2016) Internalizing/Externalizing problems: review and recommendations for clinical and Research Applications. *J Am Acad Child Adolesc Psychiatry* 55:647–656
- Hooper D, Coughlan J, Mullen MR (2008) Structural equation modelling: guidelines for determining model fit. *Electron J Bus Res Methods* 6:53–60
- Zucker RA, Heitzeg MM, Nigg JT (2011) Parsing the Undercontrol/Disinhibition Pathway to Substance Use disorders: a Multi-level Developmental Problem. *Child Dev Perspect* 5:248–255
- Dasgupta A (2015) Genetic markers of Alcohol Use Disorder. Alcohol and its biomarkers: clinical aspects and laboratory determination. Elsevier, pp 245–288
- Chao M, Li X, McGue M (2017) The Causal Role of Alcohol Use in Adolescent Externalizing and Internalizing Problems: A Mendelian Randomization Study. *Alcoholism, Clinical and Experimental Research* 41:1953–1960
- Koob GF, Volkow ND (2016) Neurobiology of addiction: a neurocircuitry analysis. *Lancet Psychiatry* 3:760–773
- Nurnberger JI Jr., Yang Z, Zang Y, Acion L, Bierut L, Bucholz K, Chan G, Dick DM, Edenberg HJ, Kramer J, Kuperman S, Rice

- JP, Schuckit M (2019) Development of Alcohol Use Disorder as a function of Age, Severity, and Comorbidity with Externalizing and Internalizing disorders in a Young Adult Cohort. *J Psychiatr Brain Sci* 4
36. Terry-McElrath YM, Stern SA, Patrick ME (2017) Do alcohol use reasons and contexts differentiate adolescent high-intensity drinking? Data from US high school seniors, 2005–2016. *Psychol Addict Behav* 31:775–785
 37. Achenbach TM, Edelbrock CS (1978) The classification of child psychopathology: a review and analysis of empirical efforts. *Psychol Bull* 85:1275–1301
 38. Colder CR, Shyhalia K, Frndak S, Read JP, Lengua LJ, Hawk LW, Wiczorek WF (2017) The Prospective Association Between Internalizing Symptoms and Adolescent Alcohol Involvement and the Moderating Role of Age and Externalizing Symptoms. *Alcoholism, Clinical and Experimental Research* 41:2185–2196
 39. Andreas JB, Ask Torvik F, Ystrom E, Skurtveit S, Handal M, Martinez P, Laslett A-M, Lund IO (2022) Parental risk constellations and future alcohol use disorder (AUD) in offspring: a combined HUNT survey and health registries study. *Psychol Addict Behav* 36:375–386
 40. Kuppens S, Moore SC, Gross V, Lowthian E, Siddaway AP (2020) The Enduring effects of parental alcohol, Tobacco, and Drug Use on Child Well-being: a Multilevel Meta-Analysis. *Dev Psychopathol* 32:765–778
 41. Kendler KS, Gardner CO, Hickman M, Heron J, Macleod J, Lewis G, Dick DM (2014) Socioeconomic status and alcohol-related behaviors in mid- to late adolescence in the Avon Longitudinal Study of parents and children. *J Stud Alcohol Drug* 75:541–545
 42. Maggs JL, Staff J, Patrick ME, Wray-Lake L, Schulenberg JE (2015) Alcohol use at the cusp of adolescence: a prospective national birth cohort study of prevalence and risk factors. *J Adolesc Health* 56:639–645
 43. Rescorla L, Ivanova MY, Achenbach TM, Begovac I, Chahed M, Drugli MB, Emerich DR, Fung DSS, Haider M, Hansson K, Hewitt N, Jaimes S, Larsson B, Maggolini A, Marković J, Mitrović D, Moreira P, Oliveira JT, Olsson M, Ooi YP, Petot D, Pisa C, Pomalima R, da Rocha MM, Rudan V, Sekulić S, Shahini M, de Silveas M, Szivovicza EF, Valverde L, Vera J, Villa LA, Viola MC, Woo L, Zhang BSC EY (2012) International Epidemiology of Child and adolescent psychopathology II: integration and applications of dimensional findings from 44 societies. *J Am Acad Child Adolesc Psychiatry* 51:1273–1283e1278
 44. Toner P, Böhnke JR, Andersen P, McCambridge J (2019) Alcohol screening and assessment measures for young people: a systematic review and meta-analysis of validation studies. *Drug Alcohol Depend* 202:39–49
 45. Mason WA, Kosterman R, Haggerty KP, Hawkins JD, Redmond C, Spoth RL, Shin C (2008) Dimensions of adolescent alcohol involvement as predictors of young-adult major depression. *J Stud Alcohol Drugs* 69:275–285
 46. Groenman AP, Oosterlaan J, Rommelse NN, Franke B, Greven CU, Hoekstra PJ, Hartman CA, Luman M, Roeyers H, Oades RD, Sergeant JA, Buitelaar JK, Faraone SV (2013) Stimulant treatment for attention-deficit hyperactivity disorder and risk of developing substance use disorder. *Br J Psychiatry* 203:112–119
 47. Tanner-Smith EE, Lipsey MW (2015) Brief alcohol interventions for adolescents and young adults: a systematic review and meta-analysis. *J Subst Abuse Treat* 51:1–18