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The relationships between use of alcohol, tobacco and coffee in adolescence and mood disorders in adulthood

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Abstract

Introduction: Alcohol, tobacco and coffee are commonly used substances and use in adolescence has previously been linked to mood disorders. However, few large prospective studies have investigated adolescent use in relation to mental health outcomes in adulthood. The main aim of this study was to examine the prospective associations between alcohol use, cigarette smoking and coffee consumption at age 16 and subsequent mood disorders up to 33 years of age.

Methods: Data from The Northern Finland Birth Cohort 1986 Study were used and a total of 7660 participants (49.9% male) were included. Associations between alcohol use, cigarette smoking and coffee consumption at age 16 and later diagnoses of major depression and bipolar disorder were examined using multinomial logistic regression analyses.

Results: Mean number of cigarettes/day (OR, 1.23 [95% CI 1.01–1.50]) and mean volume of alcohol consumption (OR, 1.22 [95% CI 1.01–1.47]), but not frequency of excessive drinking, in adolescence were associated with increased

risk for subsequent bipolar disorder after adjustment for sex, parental psychiatric disorders, family structure, illicit substance use, and emotional and behavioral problems at age 16. An association between cigarette smoking and major depression attenuated to statistically non-significant when adjusted for emotional and behavioral problems. No associations were observed between adolescent coffee consumption and subsequent mood disorders.

Conclusions: This is the first study to report an association of adolescent cigarette smoking and subsequent bipolar disorder diagnosis providing grounds for further research and pointing to a place for preventive measures among adolescents.

KEYWORDS

adolescence, alcohol, bipolar disorder, coffee, major depression, mood disorder, tobacco

1 | INTRODUCTION

Alcohol, tobacco, and coffee are the most commonly used substances in the general population, and consumption is often initiated during adolescence.[1,2] Previous research has indicated a link between use of these substances and mood disorders, though alcohol and tobacco have been subject to more investigations than coffee.[3–9] Epidemiological prospective associations have been mostly studied for major depression (MD),[3,7] while bipolar disorder (BP), which is characterized by manic or hypomanic episodes and is often chronic, is far less studied.[6] Furthermore, there are only a handful of studies that have utilized prospectively collected general population data from adolescence to adulthood.[6,7]

Importantly, due to ongoing brain development, adolescents may be particularly sensitive to the detrimental effects of substance use.[10,11] Longitudinal studies indicate that adolescent alcohol use and cigarette smoking may cause adverse neurobiological alterations[10–12] while the effect of coffee is unclear.[13,14] The relationships between substance use and mood disorders are complex and in epidemiological studies they may be affected by latent factors or confounding.

Both frequency and quantity of adolescent alcohol use has been shown to associate with subsequent MD.[3] Previous prospective studies have reported associations between higher levels of adolescent alcohol use,[15] frequency of drinking[16] bingeing,[17–19] and alcohol use problems.[20] Many of the previous findings are, however, not consistent, and concurrent psychopathology and other types of substance use often confounds the relationship.[3,18,21] Although alcohol use disorder (AUD) is associated with a four times greater risk of BP,[22] there is limited evidence to support that alcohol use precipitates BP.[6] However, previous studies have either been small, included selected populations or used only symptom-level information to determine possible BP.[6]

Both smoking status and heaviness of smoking, have been shown to associate with increased risk of subsequent MD,[4,23] and this is presumably evident also for

Significant Outcomes

- Cigarette smoking and alcohol consumption in adolescence were associated with increased risk of subsequent bipolar disorder after adjustment for background variables including adolescent emotional and behavioral problems
- Associations between cigarette smoking and alcohol consumption in adolescence and subsequent major depression attenuated to nonsignificant after adjusting for adolescent emotional and behavioral problems.
- No association between adolescent coffee consumption and later affective disorders were found.

Limitations

- Lack of information about mental health symptoms before age 16 may cause risk of undetected reversed causality between substance use and mood disorder.
- Using self-reported information may have caused underestimation of substance use.
- A relatively low number of patients with bipolar disorder gave limited power for identifying associations for consumption frequencies measured by categorical variables.

adolescents.[24] While the association between smoking and depression has been widely studied, current knowledge is scarce for BP.[8] Smoking is two to three times more common in BP compared to the general population,[25] and although smoking initiation usually precedes BP, there seems to be a complex bidirectional causality.[26–28]

It has been postulated that coffee drinking is associated with decreased risk of MD, especially among those with high caffeine intake.[5,29] However, previous evidence is based on mostly cross-sectional studies with adult populations alone.[5,29] It has been speculated that caffeine may have an impact on clinical symptoms in BP,[30] but no prospective studies have been conducted on the matter.

In this study, we aim to examine the prospective associations between alcohol use, cigarette smoking and coffee consumption at age 15/16 and subsequent register-based diagnoses of MD and BP by the age of 32/33. A range of relevant covariates was included, such as parental lifetime psychiatric diagnoses as well as illicit drug use and psychopathology of the participants at baseline.

2 | MATERIALS AND METHODS

2.1 | Participants

The Northern Finland Birth Cohort 1986 Study (NFBC1986) is an ongoing follow-up study including all children with expected date of birth between July 1st 1985 and June 30th 1986, comprising 99% ($n = 9432$) of all children born alive in the target period from the two northernmost provinces in Finland.[31]

A two-phased follow-up study commenced when participants were aged 15/16, in year 2001/2002. First, participants and their parents were sent self-report questionnaires ($n = 9215$) regarding health and wellbeing, including questions about emotional and behavioral problems (Youth Self Report), coffee consumption and cigarette smoking ($n = 7344$). Thereafter, all participants were invited to a field study where they reported volume of alcohol consumption, frequency of excessive drinking and illicit substance use ($n = 6799$) using questionnaires.

We excluded persons who had intellectual disability (ICD-10: F70-F79) or had been diagnosed with mood disorder (ICD-10: F30.x, F31.x, F32.x, F33.x, F34.0, F34.1, F34.8, F34.9, F38.x, F39, F41.2, F53.0 or ICPC-2: P73, P76), MD ($n = 73$) or BP ($n = 2$), before the age of 16. A total of 7660 persons were included in our analysis (49.9% male) (Figure 1). Three outlier observations (≥1890 g/week) in the alcohol consumption variable and three outliers (≥88 cups/day) in the coffee consumption variable were removed from the analyses.

Informed consent was obtained from all participants and their parents. The 15/16-year follow-up study was approved by the Ethics committee of the Northern Ostrobothnia Hospital District in Finland (June 17, 1999). More information is available from the NFBC1986 webpage at http://www.oulu.fi/nfbc/node/40696.

2.2 | Outcome variables

Diagnoses of mood disorders (MD or BP) were obtained from two national health care registers by the National Institute for Health and Welfare until the end of 2018, that is, by the age of 32/33. The Care Register for Health Care contains International Classification of Diseases, tenth revision (ICD-10)[32] diagnosis data about patients discharged from inpatient care (1972–2018), and from 1998 onwards also on specialized outpatient care. The Register of Primary Health Care Visits includes all outpatient primary health care visits in Finland between the years 2011 and 2018. Mood disorder cases were identified using The International Classification of Primary Care 2 (ICPC-2) codes[33] and ICD-10 diagnoses. MD included ICD-10 diagnosis codes F32.x, F33.x, F34.1, F34.8, F34.9, F38.x, F39, F41.2, and F53.0, and the ICPC-2 code P76 (depressive disorder: 103 cases). The diagnostic code F53.0 (postpartum depression) was included in the analysis as the distinction is unclear and it is closely associated with previous MD episodes.[34] BP included ICD-10 diagnosis codes F30.x, F31.x, and F34.0 and the ICPC-2 code P73 (affective psychosis: 1 case). Of those with BP, 80% also had MD. A three-class mood disorder outcome variable was defined as (1) no mood disorder, (2) MD without BP, and (3) BP.

2.3 | Exposure variables

To collect information about *mean volume of alcohol consumption* at age 15/16, the participants were asked: "How often have you drunk [beverage] during the past 12 months?" and "How much [beverage] did you usually drink in a day when you drank it?" The included beverages were beer/cider/long drinks, light wine, wine, and spirits; the responses were converted into total grams ethanol per day. To obtain *frequency of excessive drinking* the participants were shown a visual depiction of a standard drink (equaling 12 g pure alcohol) and asked how many times during the past 30 days they had had six/four drinks or more, for boys/girls. The response options were (1) never; (2) once; (3) twice; (4) 3–5; (5) 6–9 or (6) >10 times. Based on the distribution the data were pooled into a three-class variable: (1) never, (2) once or twice, and (3) three times or more.

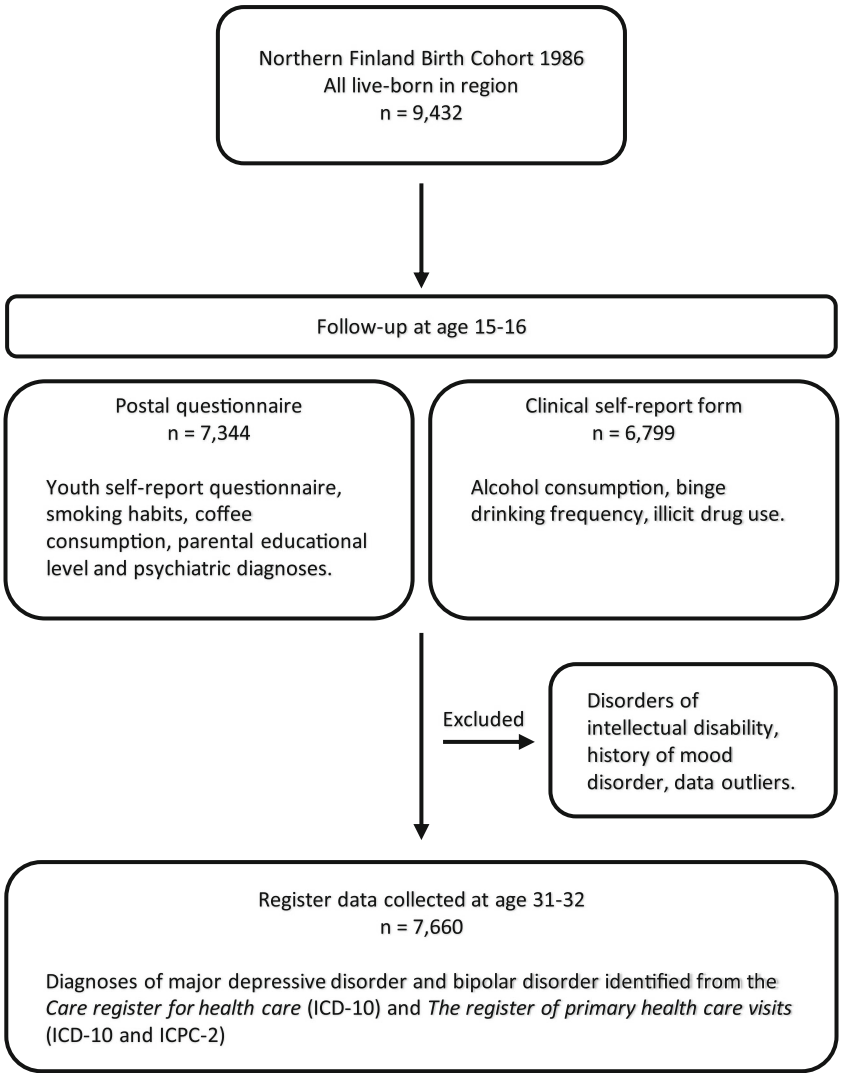

FIGURE 1 Flow chart of the data collection

Information about *cigarette smoking* was obtained by asking: "Have you ever smoked?" If yes, participants were asked "Do you smoke now?" with response options (1) not at all, (2) occasionally, (3) 1 day per week, (4) 2–4 days per week, (5) 5–6 days per week, and (6) 7 days a week. These two questions were combined and dichotomized to *daily smoking (no/yes)*. The question "How much do you smoke now?" was asked separately for filter and other cigarettes and summed up as a continuous variable of *number of cigarettes/day*.

Coffee consumption was determined by asking "How many cups of coffee do you drink in a day?" This was asked separately for filtered and brewed coffee and summed up as a continuous variable.

2.4 | Confounding variables

Parental educational level was defined by the highest education level achieved by either parent when participants were 15/16 years old. This variable was categorized into (1) <12 years (without a secondary schooling degree), and (2) ≥12 years (vocational or secondary upper-level schooling). The classification of the *family structure* included (1) families with both parents living with the participant all the time, and (2) all other family types. Lifetime parental psychiatric disorder (ICD-10 diagnosis codes F00-F69, F80-F99 from approximately 1960–2001) were obtained from three nationwide registers: (1) Register of Health Care during the years 1972 to 2018 (includes inpatient care and visits to specialized outpatient health care since 1998); (2) Disability pensions of the Finnish Centre for Pensions (1965–2016); and (3) Register of Primary Health Care Visits (2011–2018). The variable was operationalized as *parental psychiatric disorder (no/yes)*. These two variables were constructed to adjust for potential factors related to genetic or environmental heredity.

Data on lifetime illicit substance use at age 15/16 were collected with several questions regarding use of cannabis, prescription drugs, inhalants and other illicit

substances, and combined into a *Illicit substance use (no/yes)* variable.

The *Youth Self Report (YSR)* was administered at age 15/16 and covers adolescents' emotional or behavioral problems the past 6 months. As the YSR possibly taps into premorbid or subdiagnostic mental disorder it serves as an important confounding variable. The form includes 29 items on externalizing, and 30 items on internalizing problems (from total of 118) and responses are scored with statements being (0) not true; (1) somewhat/sometimes true or (2) very true. YSR subscales with >3 missing values were excluded while subscales with ≤3 missing were replaced by the mean value of the particular subscale for that person.[35,36] Items concerning substance use was removed from the YSR total score as illicit substance use was included separately.

2.5 | Statistical methods

Kruskal–Wallis and Chi-square-tests were used for studying the associations of background variables and mood disorders. Multinomial logistic regression analyses with odds ratios (OR) and 95% confidence intervals (CI) were used to examine associations between exposure and outcome variables.

The continuous exposure variables (grams/alcohol, cups/coffee and cigarettes/day) were standardized with standard deviation (SD), that is, change in OR reflects steps of one SD. Predefined models with adjustment for the following covariates were utilized: Model 1: sex; Model 2: also family structure and parental psychiatric disorder; Model 3: also illicit substance use; Model 4: also

TABLE 1 Background variables and consumption of alcohol, cigarettes and coffee in adolescence by mood disorder diagnosis in adulthood

		No mood disorder $n = 6696$ (87.4%)	Major depression $n = 879$ (11.5%)	Bipolar disorder $n = 85$ (1.1%)	Test of difference p Value
Female	*n* (%)	3215 (48.0)	562 (63.9)	63 (74.1)	<0.001[a]
Cohabiting parents	*n* (%)	4483 (78.7)	508 (68.6)	49 (68.1)	<0.001[a]
Parent ≥12 years of education	*n* (%)	2187 (37.7)	282 (37.7)	30 (42.9)	0.671[a]
Parent psychiatric diagnosis	*n* (%)	2400 (35.8)	432 (49.1)	48 (56.6)	<0.001[a]
Lifetime illicit substance use, age 16	*n* (%)	702 (12.2)	164 (22.9)	24 (32.4)	<0.001[a]
Youth Self-Report total score	Median (IQR)	23.0 (14.0–34.0)	31.3 (20.0–46.0)	33.0 (24.0–46.0)	<0.001[b]
Alcohol consumption					
Grams/day	Median (IQR)	0.5 (0.0–2.9)	0.8 (0.0–4.6)	2.4 (0.0–8.7)	<0.001[b]
Frequency of excessive drinking[c]					
None	*n* (%)	3522 (61.2)	405 (56.6)	36 (48.0)	<0.001[a]
1–2 times	*n* (%)	1726 (30.0)	215 (30.0)	26 (34.7)	
≥3 times	*n* (%)	506 (8.8)	96 (13.4)	13 (17.3)	
Cigarette smoking					
Smoking habit					
Not at all	*n* (%)	4000 (66.7)	466 (58.8)	33 (41.8)	<0.001[a]
1–6 days a week	*n* (%)	1258 (21.0)	177 (22.3)	21 (26.6)	
Every day	*n* (%)	742 (12.4)	150 (19.9)	25 (31.6)	
Cigarettes/day	Median (IQR)	0.0 (0.0–0.0)	0.0 (0.0–0.2)	0.0 (0.0–0.6)	<0.001[b]
Cigarettes/day	Mean (SD)	1.6 (4.1)	2.5 (5.0)	3.9 (6.4)	
Coffee consumption					
≥1 cup of coffee/day	*n* (%)	1177 (39.9)	231 (41.6)	27 (45.0)	0.415[a]
Cups of coffee/day	Median (IQR)	0.0 (0.0–0.2)	0.0 (0.0–0.2)	0.0 (0.0–0.3)	0.850[b]
Cups of coffee/day	Mean (SD)	1.8 (4.4)	1.7 (2.9)	1.9 (3.0)	

Abbreviations: IQR, Interquartile range (25th and 75th percentile); SD, standard deviation.

[a]Chi Square test.

[b]Kruskal–Wallis test.

[c]Last 30 days.

TABLE 2 Multinomial logistic regression of effect of alcohol, cigarette and coffee consumption in adolescence on mood disorder in adulthood

		Major depression		Bipolar disorder	
	n	**OR (95% CI)**	***p* Value**	**OR (95% CI)**	***p* Value**
Alcohol					
Grams/day[a]					
Model 1	6562	1.19 (1.10–1.29)	<0.001	1.41 (1.23–1.62)	<0.001
Model 2	5607	1.16 (1.07–1.27)	0.001	1.37 (1.17–1.59)	<0.001
Model 3	5594	1.08 (0.99–1.19)	0.100	1.28 (1.08–1.51)	0.004
Model 4	5349	1.06 (0.96–1.17)	0.244	1.22 (1.01–1.47)	0.036
Frequency of excessive drinking[b]					
Model 1	6545				
1–2 times		1.07 (0.90–1.28)	0.440	1.44 (0.87–2.40)	0.157
≥3 times		1.66 (1.30–2.12)	<0.001	2.54 (1.34–4.84)	0.004
Model 2	5592				
1–2 times		1.05 (0.86–1.27)	0.646	1.33 (0.77–2.29)	0.309
≥3 times		1.64 (1.26–2.14)	<0.001	1.62 (0.74–3.58)	0.229
Model 3	5578				
1–2 times		0.93 (0.76–1.13)	0.457	1.11 (0.63–1.97)	0.721
≥3 times		1.24 (0.93–1.65)	0.144	1.08 (0.46–2.52)	0.867
Model 4	5334				
1–2 times		0.89 (0.73–1.10)	0.272	1.05 (0.59–1.86)	0.877
≥3 times		1.09 (0.81–1.48)	0.557	0.85 (0.35–2.09)	0.729
Cigarettes					
Daily smoking (no/yes)[c]					
Model 1	6872	1.63 (1.34–1.98)	<0.001	3.19 (1.97–5.16)	<0.001
Model 2	6016	1.44 (1.15–1.79)	0.001	2.49 (1.43–4.33)	0.001
Model 3	5362	1.20 (0.93–1.55)	0.155	2.09 (1.12–3.91)	0.021
Model 4	5283	1.07 (0.82–1.39)	0.612	1.83 (0.98–3.45)	0.059
Cigarettes/day[a]					
Model 1	6718	1.21 (1.13–1.29)	<0.001	1.43 (1.25–1.65)	<0.001
Model 2	5882	1.18 (1.10–1.26)	<0.001	1.34 (1.14–1.59)	0.001
Model 3	5246	1.11 (1.02–1.20)	0.021	1.27 (1.05–1.54)	0.012
Model 4	5164	1.07 (0.98–1.17)	0.157	1.23 (1.01–1.50)	0.039
Coffee					
Cups of coffee/day[a]					
Model 1	6854	1.01 (0.92–1.10)	0.924	1.09 (0.88–1.34)	0.437
Model 2	5995	0.98 (0.88–1.09)	0.728	0.98 (0.70–1.38)	0.915
Model 3	5344	0.97 (0.85–1.10)	0.605	0.90 (0.56–1.45)	0.670
Model 4	5267	0.95 (0.83–1.09)	0.483	0.87 (0.52–1.46)	0.607

Note: OR = Odds ratio; 95% CI = 95% confidence interval of OR. Reference group: No mood disorder. *Model 1*: Adjustment for sex; *Model 2*: Additional adjustment for parental psychiatric disorder and family structure; *Model 3*: Additional adjustment for illicit substance use; *Model 4*: Additional adjustment for YSR.

[a]For continuous variables change in OR reflects steps of one standard deviation.

[b]Reference group: no excessive drinking last 30 days.

[c]Reference group: no daily smoking.

YSR total score. Results were considered statistically significant at $p < 0.05$.

In the current study, there were fewer participants in the follow-up at age 15/16 among habitants of urban areas (80% vs. 85%, $p < 0.001$), males (78% vs. 84%, $p < 0.001$), and among participants with maternal (77% vs. 82%, $p < 0.001$) or paternal (79% vs. 81%, $p < 0.008$) history of psychiatric disorders. Therefore, to address potential attrition bias we used inverse probability weighting for sex, parental psychiatric disorder and urbanicity.[37] The associations were still significant and of similar magnitude in the weighted analyses as in the unweighted analyses (Tables S1 and S2).

To evaluate our findings from the regression analyses against potential confounding factors, we calculated *E*-values (Table S3). An *E*-value with the lower bound of the CI represents the minimum strength of association between unmeasured confounding factors and outcome that would be required to explain away the association between exposure and outcome variables presented in a regression analysis. An *E*-value that is relatively large in comparison to the OR indicates that the association is unlikely to be explained by unmeasured confounding factors. To control for effect of smoking on alcohol consumption and vice versa, additional regression models were added (Table S4) as sensitivity analyses. Linear regression and multicollinearity diagnostics with variance inflation factor (VIF) scores were used to detect correlation between multiple covariates. VIF >5 was used as an indicator of multicollinearity.

Statistical analyses were performed using SPSS version 25 and R version 3.6.0 (sensitivity analysis).

3 | RESULTS

Of the total sample of 7660 individuals, 879 (11.5%) were diagnosed with MD and 85 (1.1%) with BP by the age of 33. Associations between background variables and mood disorders are displayed in Table 1.

The multinomial logistic regression analyses are shown in Table 2. An association between alcohol consumption and BP remained statistically significant even in the model adjusted for sex, family structure, parental psychiatric disorder, illicit drug use, and YSR total score (Model 4) (OR, 1.22 [95% CI, 1.01–1.47]). The association between alcohol consumption and MD was significant when adjusting for sex, parental psychiatric disorder and family structure (OR, 1.16 [95% CI, 1.10–1.29]) but attenuated after further adjustment.

There were modest associations between frequency of excessive drinking ≥3 times/month and both mood disorders in Model 1: (OR, 1.66 [95% CI, 1.30–2.12]) for MD and (OR, 2.54 [95% CI, 1.34–4.84]) for BP. After adjustment for parental variables the association with BP attenuated, while the association with MD was significant (OR, 1.64 [95% CI, 1.26–2.14]) but attenuated after further adjustment for illicit substance use.

The association between number of cigarettes/day and BP remained statistically significant even in Model 4 (OR, 1.23 [95% CI, 1.01–1.50]), while the association with MD was statistically significant in Model 3 (OR, 1.11 [95% CI, 1.02–1.20]), but not with further adjustments. When examining daily smoking (no/yes), the association with BP was significant in Model 3 (OR, 2.09 [95% CI, 1.12–3.91]), but not the association with MD.

Associations between coffee consumption in adolescence and adulthood mood disorder were not seen.

The inverse probability weighted regression models confirmed our findings (Tables S1 and S2). VIFs in Model 4 for both alcohol consumption and cigarettes smoking were <1.2. When adding alcohol consumption (g/day) as a covariate to the cigarette smoking models and cigarettes/day to the alcohol consumption models the associations attenuated to non-significant (Table S4).

4 | DISCUSSION

In this prospective study based on a large birth cohort linked with registry data, adolescent cigarette smoking was associated with increased risk for subsequent BP, and this was true after adjustment for sex, any parental psychiatric disorder, family structure, illicit substance use, and emotional and behavioral problems at age 15/16. The associations between cigarette smoking and MD attenuated to statistically non-significant in the last adjustment steps. Adolescent mean volume of alcohol consumption (but not frequency of excessive drinking) was associated with subsequent BP after all four steps of adjustments. No associations were observed between adolescent coffee consumption and subsequent mood disorders.

Adolescent cigarette smoking, when examined as number of cigarettes/day, was associated with subsequent risk of BP independently of a range of covariates, including baseline mental health (YSR) and parental psychiatric disorders. In line with our findings, recent Mendelian randomization studies have indicated that cigarette smoking seems to be a causal risk factor for BP.[27,38] Also, a recent Norwegian cohort study reported a prospective association between adolescent smoking and later prescription for mood-stabilizers, a proxy for BP.[39] However, the association between smoking and BP could be bidirectional,[27] and a possible causation from BP to smoking is supported by the fact that smoking is more common among persons with BP than in the general population,[25] and that there is a heavy

genetic load for bipolar disorder.[40] Despite these findings, this is to the best of our knowledge the first study to report an association of adolescent cigarette smoking and subsequent BP, which provide grounds for further research.

Increased risk for MD among smokers has been reported in meta-analyses for both adults[41] and adolescents,[24] suggesting that smoking may be causally linked to MD. However, in our study, the association between cigarette smoking and MD attenuated to a non-significant level when adjusted for YSR total score. Although most previous studies have adjusted for demographics and adolescent alcohol and drug use,[24,42] they have failed to take into account adolescent emotional and behavioral problems like the current study does. Thus, the present study may challenge the causality implied by others, indicating that smoking may be a consequence of major depression rather than a risk factor.

The mean volume of alcohol consumption in adolescence was associated with BP in adulthood, even after multiple adjustments. Frequency of excessive drinking, however, did not predict BP in the same way. There is a lack of studies concerning adolescent alcohol consumption and later BP, as most previous research on adolescent alcohol use has focused on unipolar depression[16,43–45] and depressive symptoms[46] as outcomes.

The association between adolescent alcohol use and subsequent MD was weaker than for BP and was attenuated when adjusting for illicit drug use and YSR, much like what was found for smoking and MD. The findings agree with a previous report from our group,[21] which also found frequency of excessive drinking to be unrelated to later mood disorder. However, several studies identify binge drinking in adolescence as a better predictor than mean volume for adverse mental health outcomes in adolescent age[18] and adulthood.[47] It may be that the present study have too low power for revealing a relationship between frequency of drinking and mood disorders. Furthermore, other studies have found associations between alcohol-related problems and drinking frequency with later MD,[16,20,44] but this could be because these studies set higher limits for alcohol consumption, even addressing alcohol problems, or because the current study adjusted for illicit substance use and emotional and behavioral problems in adolescence.

We observed no association between coffee consumption and subsequent mood disorders in the current study. Previous findings concerning coffee consumption and mood disorders mainly point to an inverse relationship where coffee seem to decrease the risk of MD in adults.[48–51] Less is known about how coffee and caffeine affect adolescents, but positive cross-sectional associations have been reported.[52] Consumption of other caffeinated beverages, such as energy drinks has become substantial,[53] but were not included in the current study as they were not marketed when data collection took place. The coffee exposure in the studied adolescents was relatively low and the association between coffee and mood disorders may be different in samples with higher consumption of caffeinated beverages. Possible subsequent effects of caffeine intake in adolescence is an important issue that still needs more research.

A major strength of the current study is NFBC1986 being one of the largest ongoing birth cohort studies and it has high genetic and ethnic homogeneity. We also used several nationwide registers with little missing information for the mental health outcomes. Mood disorder outcomes included in the study were taken from both primary and specialized health care. This was done to ensure a broad enough capture of outcome. Although the specificity of diagnosis from the two settings is unknown, underdiagnosis of mood disorders may represent a greater concern, as many go undiagnosed.[54] In addition, some patients are more likely to get in contact with the health care system, such as patients with comorbid personality disorders, and thus they may be overrepresented in the registers. Attrition bias and unmeasured confounding were controlled for and did not influence the findings. Furthermore, the data included a wide range of information that made it possible to address many potential confounders. It could be a limitation that we did not have information about symptoms prior to age 15/16, and reversed causality between mental health symptoms and substance use cannot be ruled out. Diagnostic information on comorbid psychiatric disorder at age 15/16 was not included, and self-reported substance use measures as used in this study are known to underestimate substance use[55] and may lead to underestimation of true associations. Lastly, the number of BP cases was potentially too low to produce robust estimates with frequency of excessive drinking. More research with longitudinal design is needed to further examine the individual effects of volume of consumption and pattern of drinking on mood disorders in adulthood.

5 | CONCLUSIONS

Our findings demonstrate that both adolescent cigarette smoking and alcohol consumption are associated with increased risk for subsequent mood disorders. These relationships are relatively well established for MD, but the current study points to the relationships being even more pronounced for BP. We were not able to demonstrate any relationship between coffee consumption and mood disorders.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

PEER REVIEW

The peer review history for this article is available at https://publons.com/publon/10.1111/acps.13506.

DATA AVAILABILITY STATEMENT

NFBC data is available from the University of Oulu, Infrastructure for Population Studies. Permission to use the data can be applied for research purposes via electronic material request portal. In the use of data, we follow the EU general data protection regulation (679/2016) and Finnish Data Protection Act. The use of personal data is based on cohort participants' written informed consent at his/her latest follow-up study, which may cause limitations to its use. Please, contact NFBC project center (nfbcprojectcenter@oulu.fi) and visit the cohort website (www.oulu.fi/nfbc) for more information.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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