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Parental Risk Constellations and Future Alcohol Use Disorder (AUD) in Offspring: A Combined HUNT Survey and Health Registries Study

Jasmina Burdzovic Andreas[1], Fartein Ask Torvik[2, 3], Eivind Ystrom[1, 4, 5], Svetlana Skurtveit[1, 6], Marte Handal[3], Priscilla Martinez[7], Anne-Marie Laslett[8], and Ingunn Olea Lund[1]

[1] Department of Alcohol, Tobacco, and Drugs, Norwegian Institute of Public Health, Oslo, Norway
[2] Center for Fertility and Health, Norwegian Institute of Public Health, Oslo, Norway
[3] Department of Psychology, University of Oslo
[4] PROMENTA Research Center, Department of Psychology, University of Oslo
[5] Pharmacoepidemiology and Drug Safety Research Group, School of Pharmacy, University of Oslo
[6] Norwegian Centre for Addiction Research, University of Oslo
[7] Alcohol Research Group, Public Health Institute, Emeryville, California
[8] Centre for Alcohol Policy Research, LaTrobe University

Objective: We examined the risk of developing a future alcohol use disorder (AUD) among offspring of families with different constellations of parental risk factors. ***Method:*** We analyzed a sample of 8,774 offspring (50.2% male) from 6,696 two-parent families who participated in the Nord-Trøndelag Health Study in Norway when offspring were 13–19 years old in 1995–1997 or 2006–2008. Based on population registry information and parental Nord-Trøndelag Health Study self-reports, families were classified via Latent Profile Analysis into fiver risk constellations reflecting parents' education, drinking quantities and frequencies, and mental health. Information about AUD-related diagnoses, treatments, and prescriptions for all offspring in the period between 2008 and 2016 was obtained from 3 national health registries and pooled to reflect any AUD. The likelihood of AUD in offspring was examined with a set of nested logistic regression models. ***Results:*** Registry records yielded 186 AUD cases (2.1%). Compared with the lowest-risk constellation, offspring from two constellations were more likely to present with AUD in unadjusted analyses. After adjusting for all covariates, including offspring's alcohol consumption and witnessing parental intoxication during adolescence, AUD risk remained elevated and statistically significant (adjusted odds ratio = 2.34, 95% confidence interval = 1.14, 4.85) for offspring from the constellation characterized by at least weekly binge drinking, low education, and poor mental health in both parents. ***Conclusion:*** Weekly binge drinking by both parents was associated with future AUD risk among community offspring in Norway when clustered with additional parental risks such as poor mental health and low educational attainment.

Public Significance Statement

Focus on parental alcohol use disorder (AUD) as a risk factor for offspring's AUD overlooks youth from community samples and the effects of parental drinking habits that are not necessarily part of a clinical disorder. Parental drinking patterns that do not manifest as a clinical disorder may also contribute to the development of AUD in offspring. Weekly binge drinking in both parents increased AUD risk in offspring when combined with additional risk factors such as poor mental health and low educational attainment in parents.

Keywords: alcohol use disorder, community sample, cohort studies, latent profile analysis, risk factors

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Jasmina Burdzovic Andreas https://orcid.org/0000-0002-6730-1321
Fartein Ask Torvik https://orcid.org/0000-0003-3984-5978
Eivind Ystrom https://orcid.org/0000-0003-4390-6171
Marte Handal https://orcid.org/0000-0003-1773-0184
Priscilla Martinez https://orcid.org/0000-0002-6797-1632
Anne-Marie Laslett https://orcid.org/0000-0001-5652-7336
Ingunn Olea Lund https://orcid.org/0000-0001-7412-4776

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Correspondence concerning this article should be addressed to Jasmina Burdzovic Andreas, Department of Alcohol, Tobacco, and Drugs, Norwegian Institute of Public Health, Postboks 222-Skøyen, 0213 Oslo, Norway. Email: jabu@fhi.no

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Parental characteristics and behaviors along with other individual, peer, and community risk and protective factors shape children's developmental outcomes, including negative outcomes such as alcohol-related problems and disorders (Bronfenbrenner, 1977; Chassin et al., 2013; Ennett et al., 2008; Richter & Richter, 2001). Indeed, the proportion of youth at risk for developing alcohol use disorders (AUD) later in life may be considerable, given that these disorders are relatively prevalent in the general adult population (Grant et al., 2015; Rehm et al., 2015) and are more common among offspring of similarly affected parents (Chassin et al., 1999; Holst et al., 2019; Jennison & Johnson, 1998; Johnson & Leff, 1999; Lieb et al., 2002; Mellentin et al., 2016; Sørensen et al., 2011). The official North American and European estimates indicate that between 3.5% and 13.9% of the adult population is affected by an AUD each year (Grant et al., 2015; Rehm et al., 2015; Rehm et al., 2005) and that approximately 10% of children in the United States may be living with an AUD-affected parent (Lipari & Van Horn, 2013). The situation is similar in Norway, where 8.3% of children younger than 18 years—which translated to approximately 90,000 children in 2011—were estimated to have at least one parent who was affected by an AUD within the last year (Torvik & Rognmo, 2011).

It is therefore not surprising that past research has primarily focused on offspring of parents with a clinically defined AUD and corresponding intergenerational transmission mechanisms (Hussong et al., 2008; Lieb et al., 2002; Mellentin et al., 2016; Slutske et al., 2008; Sørensen et al., 2011). However, such a focus somewhat overlooks youth from community samples and the possible adverse effects of parental drinking habits that are not necessarily part of a clinical disorder (Rossow et al., 2016).

Indeed, whether parental non-AUD drinking affects offspring's alcohol use has received relatively sparse research attention: A recent review identified only a handful of causally informative cohort studies addressing this question (Rossow et al., 2016). Because all of the reviewed studies examined non-clinically defined outcomes such as excessive drinking or drinking frequencies in offspring only, it is unknown whether normative parental drinking may be associated with AUD risk in offspring. This is surprising because the number of drinking families greatly exceeds the number of AUD-affected families. Thus, understanding the risks, if any, of other forms of parental alcohol consumption on offspring's AUD risk is imperative (Fischerman, 2000; Manning et al., 2009; Richter et al., 2001). Whether parental drinking outside clinically defined AUD—by itself or in combination with other parental characteristics—may contribute to future AUD risk in offspring were the main questions explored in this investigation.

Addressing such questions requires broad theoretical and methodological perspectives. Emerging research underscores the need to consider the family system as a whole and to examine both parents' drinking (Finan et al., 2018; Haugland et al., 2015; Haugland et al., 2013; Homel & Warren, 2019; Karlsson et al., 2016; Mares et al., 2012; Pedersen & von Soest, 2013; Vermeulen-Smit et al., 2012), as well as for the utilization of the more advanced analytical approaches (Bates, 2000; Bergman & Magnusson, 1997; Malmberg et al., 2012; von Eye & Bergman, 2003). In other words, to understand the associations between AUD in offspring and patterns of parental drinking and other risk factors, we need to understand those maternal and paternal risk patterns first.

This is especially the case because disorders such as AUD tend to have varied developmental origins (Appleyard et al., 2005; Chassin et al., 2013; Kendler, 2019; Kendler et al., 2011), stem from the accumulation of adverse experiences and risks factors (Lee et al., 2014; Pilowsky et al., 2009; Zufferey et al., 2007), and co-occur with poor mental health or low socioeconomic status (Berg et al., 2016; Hussong et al., 2008; Kendler et al., 2014; Nesvåg et al., 2015). Yet the relevant literature frequently utilizes combined parental instead of individual maternal/paternal indices of alcohol use based on the subjective, retrospective, and single-source assessments of risks and relies on the variable-centered instead of person-centered conceptualization of risks (Alati et al., 2014; Cox et al., 2018; Finan et al., 2018; Merline et al., 2008; Olsson et al., 2019; Pedersen & von Soest, 2013). Such approaches do not fully capture the complexity of family systems and risk factors manifested between and within parents. Only a handful of reports have aimed to empirically detect risk patterns based on the alcohol use of both parents and to investigate their prospective associations with various negative outcomes in their offspring (Lund et al., 2019; Vermeulen-Smit et al., 2012). Whereas none of these studies examined AUD specifically, offspring from families with heavy drinking fathers or with two heavy episodic drinking parents were at greater risk for an earlier onset of and heavier alcohol use in adolescence compared with families with lower levels of alcohol use (Vermeulen-Smit et al., 2012).

To bridge these research gaps, we focused on offspring from community samples and examined how various patterns and complex constellations of parental drinking, education, and mental health may influence their AUD risk later in life. We were primarily guided by the cumulative risk model and the argument that accumulation of early negative experiences and multiple stressors increases the odds of later maladjustment (Appleyard et al., 2005), including a range of alcohol-related problems such as alcohol misuse in adolescence and heavy episodic drinking and alcohol dependence in adulthood (Lee et al., 2014; Pilowsky et al., 2009; Zufferey et al., 2007). However, cumulative risk research may be limited in several aspects. These include a reliance on preconceived values for determining whether or not risk is present when computing cumulative risk scores and the implicit interchangeability of risk factors and the associated developmental processes driven by the number, not the nature, of risk factors (McLaughlin & Sheridan, 2016). In this study we extended the traditional cumulative risk model to examine the putative effects of complex risk constellations, defined not only by the number of risk factors but also by their potentially different levels of harm and combinations thereof.

To this end, we: (a) used putative risk constellations derived from a previously conducted latent profile analysis (Lund et al., 2019), (b) included data from survey self-reports and national registries on both parents and considered parental-level socioeconomic status and mental health, (c) analyzed offspring's AUD as objectively recorded in the national population registries years after the risk exposures during adolescence, and (d) accounted for important individual-level covariates to aid causal inferences. Such an integrative approach extends the current literature on cumulative risk and future AUD, both conceptually and methodologically. The results may improve understanding of the AUD risk beyond

those inferred by parental AUD and may be informative for population-based intervention strategies, especially considering the high individual and social costs of AUD (Jacob et al., 2001; Kendler et al., 2017).

Finally, understanding AUD risk outside the intergenerational transmission mechanisms may be of particular relevance in Norway. AUDs are consistently one the most prevalent and comorbid mental health disorders (Kringlen et al., 2001, 2006; Nesvåg et al., 2015), and drinking is an integral part of everyday life in Norway (OsloEconomics Report, 2013). Norwegian drinking patterns mainly consist of hazardous drinking in early adulthood (Erevik et al., 2017) and heavy alcohol consumption on weekends and holidays, especially among men (Horverak & Bye, 2007). In a society in which the lifetime prevalence of AUD ranges between 9.4% and 22% in the adult population (Kringlen et al., 2001, 2006) and in which normative drinking patterns largely reflect occasional (i.e., weekend) consumption of excessive quantities (Horverak et al., 2007), it is highly likely that the proportion of offspring exposed to potentially problematic parental drinking at some point during adolescence greatly exceeds the estimated 8% exposed to parental AUD alone during a calendar year (Torvik et al., 2011). What kind of problematic parental drinking that may be, and in what accumulated risk combinations it may affect future AUD risk in offspring, was our central question.

Specifically, this study builds on our previous work that identified parental risk constellations defined by various levels and combinations of parental drinking with other risk factors to examine whether such risk constellations may affect the future development of AUD in offspring.

Method

Study Design, Data Sources, and Procedures

This report combined the following: (a) survey data obtained from the Nord-Trøndelag Health Studies (HUNT and Young-HUNT; Holmen et al., 2003; Holmen et al., 2014; Krokstad et al., 2013), which were used to identify primary exposures and key covariates, and (b) administrative data obtained from three Norwegian national health registries, which were used as a longitudinal follow-up means to identify primary outcomes of interest (Bakken et al., 2019; Furu, 2008).

HUNT/Young-HUNT

The HUNT/Young-HUNT are general population health surveys in Norway implemented in several cross-sectional waves (Holmen et al., 2003; Holmen et al., 2014; Krokstad et al., 2013), in which all adults older than 20 years (HUNT) and all adolescents between 13 and 19 years of age (Young-HUNT) in Nord-Trøndelag county were invited to participate. This report utilized HUNT waves 2 (administered in 1995–1997) and 3 (administered in 2006–2008), which provided parental self-reports, and concurrently administered Young-HUNT waves, which provided adolescent offspring self-reports.

National Health Registries

Detailed medical information is available on all residents in Norway through obligatory, population-level administrative health registries (Bakken et al., 2019). This study utilized: (a) the Database for Control and Payment of Health Reimbursements Registry for practitioners in primary health care, which provides information on the *International Classification of Primary Care* diagnosis code recorded at each contact with primary health care providers; (b) the Norwegian Prescription Database, which provides information on all dispensed prescription drugs to patients in ambulatory care in Norway; and (c) the Norwegian Patient Registry, which provides information on admission to hospitals and other specialist health care and includes *International Statistical Classification of Diseases and Related Health Problems*, 10th revision, diagnosis codes.

Procedure

Identification and extraction of family relationships (i.e., of adult dyads and any adolescent offspring residing in the same household) and linkages between the Young-HUNT/HUNT surveys and health registries at the individual level were achieved with technical assistance from Statistics Norway (Lund et al., 2015; Lund et al., 2019) and through the utilization of national personal identification numbers (i.e., Social Security numbers). Statistics Norway also provided additional demographic data, such as the attained educational level.

In-depth study details are described elsewhere, including study design and participant selection, data sources, follow-up timeline, definitions of families for analytical purposes, and construction of primary exposures based on parental characteristics (Lund et al., 2015; Lund et al., 2019). Informed consent and assent were obtained for all participants by the original HUNT and Young-HUNT studies, including permission for future linkages with health registries. This study was approved by the Regional Committees for Medical and Health Research Ethics (number 2014/867) and the Norwegian Data Protection Authority (number 38949).

Sample

This report examined a combined sample of 8,774 offspring from 6,696 two-parent families who (a) participated in the Nord-Trøndelag Health Studies (Young-HUNT) in 1995–1997 or in 2006–2008 when they were adolescents and (b) were then followed up via national health registries between 2008 and 2016. All offspring were 13–19 years old at the time of Young-HUNT participation and 14–33 years old in 2008 at the time of the first registry follow-up and initiation of this study.

Measures: Outcome

Offspring AUD

AUD in offspring during the study period of 2008–2016 was identified through the administrative records in three primary national health registries in Norway; all registries and corresponding AUD-relevant codes are shown in Table 1. Relevant records were pooled to reflect the presence of any AUD-related entry for each offspring, where 1 = at least one AUD-relevant record during the study period.

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Table 1
Overview of the National Health Registries and Corresponding Entries Used to Identify AUD in Offspring During Study Period, 2008–2016

Registry	Full name	Classification	Codes (diagnoses/prescription drugs)
1. CPHR	Database for Control and Payment of Health Reimbursements	ICPC codes[a]	P15 (chronic alcohol abuse) P16 (acute alcohol abuse)
2. NorPD	Norwegian Prescription Database	ATC codes[b]	ATC codes starting with N07BB Prescription drugs used to treat alcohol dependence
3. NPR	Norwegian Patient Registry	ICD-10 codes[c]	F10 (alcohol-related disorders)

[a] International Classification of Primary Care. [b] Anatomical Therapeutic Chemical. [c] International Classification of Diseases.

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Primary Exposure

Risk Constellations Based on Parental Education, Drinking, and Mental Health

Educational attainment for both parents was obtained from Statistics Norway and converted into completed years of education for analytical purposes. As part of the HUNT surveys, both mothers and fathers reported their usual alcohol consumption, including frequency (i.e., "How many times a month do you normally drink alcohol?") and quantity (i.e., "How many glasses of beer, wine, or spirits do you usually drink in the course of 2 weeks?"). Both parents also reported their mental health symptoms during the past 14 days on the 14-item Hospital Anxiety and Depression Scale (HADS). The scale was shown to be a robust screening instrument in Norwegian samples; a raw score of 8 or greater is indicative of a clinical disorder and is commonly used as a diagnostic cutoff (Leiknes et al., 2016; Mykletun et al., 2001; Stordal et al., 2001).

These indicators of parental education, drinking, and mental health were previously used in a latent profile analysis (LPA) to derive our primary exposure: the unique constellations of parental risk factors as manifested during offspring's adolescence (Lund et al., 2019). To aid interpretability of results and to avoid reliance on preconceived risk categorizations, all LPA indicators were used in their original format. For example, rather than using dichotomized categories based on the diagnostic cutoffs, we used continuous HADS scores to detect more nuanced profiles of parental mental health symptomatology. Similarly, to detect parental drinking patterns unrestricted by traditional definitions of clinical disorders, we analyzed simple drinking frequencies (number of times per month) and quantities (number of alcohol units per 2 weeks) as reported in the original HUNT surveys by parents.

Specifically, in this sample we have previously identified a total of five mutually exclusive constellations of parental risks using the LPA, a well-established person-centered approach allowing classification and examination of observed individuals according to their shared behavioral and/or other characteristics (Bergman & Magnusson, 1997; Lanza & Rhoades, 2013). We used the LPA to identify unobserved groups of parents (i.e., two-parent families) who were similar in terms of their education, mental health, and alcohol use. Comparable approaches have been used in other reports for classification and identification of typologies of families affected by parental substance abuse (Jääskeläinen et al., 2016; Lowthian et al., 2020) or for closer examination of alcohol use in offspring as a function of such identified patterns in parental drinking (Vermeulen-Smit et al., 2012), parenting behaviors (Latendresse et al., 2009), and socioeconomic characteristics (Skogen et al., 2019). All LPA analyses were conducted in Mplus version 8 software using a default MLR estimator (Lanza et al., 2013; Muthén & Muthén, 2017), and the resulting risk constellations are conceptually described below and in Table 2. All procedures and the original LPA estimates (i.e., means and *SE*; fit indices, etc.) are reported in detail elsewhere (Lund et al., 2019).

LP1 reflected families with the lowest educational attainment in both parents (i.e., no completed high school on average) but no apparent additional risk factors (LP1 = low socieconomic status only; n = 5,966 or 68.0% of offspring). LP2 reflected families with low educational attainment and mental health symptoms in the mild disorder range in both parents. On average, mothers from this group drank 1 day per week and consumed 4 units of alcohol on said day, whereas fathers drank about 2 days per week and consumed 6 units of alcohol on each drinking day (LP2 = multiple risks + weekly binge drinking in both parents; n = 246 or 2.8%). As such, drinking patterns in both LP2 parents aligned with the commonly used binge drinking/heavy episodic drinking definitions (Esser et al., 2014; National Institute of Alcohol Abuse and Alcoholism, 2004). LP3 was selected as a reference group because of its comparably low-risk characteristics for both parents, including the highest educational attainment, sparse and low-quantity drinking (i.e., less than weekly and a bit above 2 units of alcohol per drinking occasion), and average HADS scores in the normal range (LP3 = low risk; n = 1,884 or 21.5%). LP4 captured families in which both parents drank about two times per week and consumed a couple of alcohol units on each drinking occasion but had no other risk factors relative to the remaining sample (LP4 = weekly casual drinkers; n = 598 or 6.8%). The final group, LP5, reflected families with multiple risk factors, including maternal minimal (i.e., a single unit of alcohol) but almost daily alcohol consumption, and paternal average HADS scores in a mild disorder range (LP5 = multiple risks + maternal daily drinking; n = 79 or 0.9%).

Covariates

Demographics

As part of the Young-HUNT survey participation during adolescence, offspring reported their gender and birthday, which was used to compute the age at Young-HUNT participation as well as the age at study entry (i.e., age at first registry follow-up in 2008).

Alcohol-Relevant Experiences During Adolescence

As part of the Young-HUNT survey, offspring reported their alcohol consumption during an ordinary 2-week period. These

Table 2
Description of the Selected Latent Profile (LP) Analysis Solution and Corresponding Parental Risk Constellations

Characteristic	LP1 Low education only	LP2 Multiple risks, including weekly binge drinking in both parents	LP3 Low overall risk	LP4 Casual weekly drinking in both parents	LP5 Multiple risks, including maternal daily low-quantity drinking
Participants, *n* (%)					
Family[a]	4,857 (69.1%)	194 (2.8%)	1,444 (20.5%)	473 (6.7%)	61 (.9%)
Offspring	5,966 (68.0%)	246 (2.8%)	1,884 (21.5%)	598 (6.8%)	79 (.9%)
Completed education (years)[b]					
Maternal	**Less than 12 years**	**Less than 12 years**	More than 12 years	More than 12 years	More than 12 Years
Paternal	**Less than 11 years**	**Less than 12 years**	More than 14 years	More than 12 years	More than 12 years
Maternal drinking (weekly)[c,d]					
Average quantity	1 unit/week	**3.92 units/week**	1.25 units/week	**4.1 units/week**	**6.5 units/week**
Average frequency	0.4 days/week	**0.95 days/week**	0.5 days/week	**2.3 days/week**	**5.4 days/week**
Average alcohol units/occasion	—	**4.1 units/occasion**	—	**1.7 units/occasion**	**1.1 units/occasion**
Paternal drinking (weekly)[c,d]					
Average quantity	2 units/week	**11.2 drinks/week**	2.3 units/week	**4.8 units/week**	**6.6 units/week**
Average frequency	0.7 days/week	**1.9 days/week**	0.9 days/week	**2 days/week**	**3 days/week**
Average alcohol units/occasion	—	**5.95 units/occasion**	—	**2.35 units/occasion**	**2.2 units/occasion**
Mental health (HADS score)[c,e]					
Maternal	Normal range	**Mild disorder range**	Normal range	Normal range	Normal range
Paternal	Normal range	**Mild disorder range**	Normal range	Normal range	**Mild disorder range**

Note. Shown are the conceptual summaries for the LPA-derived parental risk constellations (LP1–LP5); the corresponding LPA procedures and complete results (i.e., means and standard error; fit indices, etc.) were reported in Lund et al. (2019). To aid interpretation, the original estimates based on continuous indicators were reconceptualized here to show average weekly drinking quantities and frequencies as well as the meaningful educational cutoffs (i.e., 12 years of completed education) and disorder severity for HADS average scores (Stordal et al., 2001) for each identified LP. Elevated levels of parental risk factors for a given LP are shown in bold. LP1 and LP3 were not considered to be risky drinking patterns because they reflected less than weekly drinking in both parents; thus, averages per occasion are not shown. HADS = Hospital Anxiety and Depression Scale; LPA = latent profile analysis; HUNT = Nord-Trøndelag Health Studies.
[a] Family refers to 7,029 temporally unique families used for clustering risk exposures of substantive interest. Because some families had multiple offspring, the number of offspring is greater than the number of families for each LP. [b] Obtained from the official Statistics Norway records. [c] Obtained from parental self-reports/HUNT. [d] Number of drinks was defined as the number of glasses of beer, wine, or liquor reported in HUNT surveys. [e] HADS (14-item scale) is a commonly used screening tool for anxiety and depression, in which the raw scale scores ranges translate to these diagnostic categories: 0–7, normal; 8–10, mild; 11–14, moderate; and 15–21, severe disorder (Leiknes et al., 2016; Stordal et al., 2001).

simple counts (i.e., the total number of beer, wine, liquor, etc. alcohol units consumed during this period) were recoded to capture no alcohol intake/past 2-weeks, 1–5 units of alcohol/past 2-weeks, 5 or more units of alcohol/past 2-weeks, and missing information categories. The original Young-HUNT reports concerning parental intoxication were dichotomized to reflect adolescents who explicitly endorsed never witnessing parental alcohol intoxication versus rest (including 127 missing responses).

Analyses

The risk of future AUD in offspring as a function of the LPA-derived parental risk constellations was examined with a nested set of logistic regression models. We first estimated the unadjusted bivariate models between the offspring AUD and all study variables. Then we estimated a set of adjusted models accounting for demographics and risk constellations first (Model 1: gender, age at Young-HUNT participation, age at study entry in 2008 + risk constellations) and then for offspring's alcohol consumption (Model 2: Model 1 + alcohol consumption) and witnessing parental alcohol intoxication (Model 3: Model 2 + witnessing parental intoxication) during adolescence.

All models were estimated with clustered robust errors to account for within-family nesting (i.e., for cases in which multiple children resided in the same family). All reported analyses were conducted in Stata 15 (StataCorp., 2017).

Results

Sample Characteristics

Table 3 summarizes all study variables. The sample was equally distributed by gender (50.2% male); the participants were on average 16 years old at the time of their Young-HUNT participation and on average 24 years old at first registry assessment in 2008. At Young-HUNT participation, 20% reported consuming 5 or more units of alcohol during the usual 14-day period, and 38% unambiguously reported never having seen their parents intoxicated.

A total of 186 offspring (2.1%) presented with at least one AUD entry in at least one health registry during the 2008–2016 follow-up period. Of these, 123 (1.4%) presented with at least one AUD-indicative code in the Control and Payment of Health Reimbursements Registry; 54 in the Norwegian Patient Registry (0.6%); and 46 in the Norwegian Prescription Database (0.5%).

Parental Risk Constellations and Future AUD Risk in Offspring

The unadjusted representation of AUD cases within each risk constellation was as follows: 2.1% in LP1 (127 cases), 4.5% in

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Table 3
Sample Characteristics (N = 8,773) and Study Variables

Sample characteristics	*M* (*SD*)	*N* (%)
Gender (male)[a]	—	4,406 (50.2%)
Age at exposure (age at Young-HUNT participation)[a]	16.05 (1.79)	—
Age at study entry (at first registry follow-up in 2008)	23.80 (5.68)	—
Parental risk constellations (latent profiles)[b,c]		
LP1		5,966 (68.0%)
LP2		246 (2.8%)
LP3 (reference)		1,884 (21.5%)
LP4		598 (6.8%)
LP5		79 (0.9%)
Adolescent alcohol intake/usual 2-week period[a]		
None		4,294 (48.9%)
1–5 drinks		1,902 (21.7%)
5+ drinks		1,807 (20.6%)
No valid report		770 (8.8%)
Witnessing parental alcohol intoxication[a]		
Never	—	3,379 (38.4%)
		Number of cases, *N* (%)
Registries-based outcomes		
CPHR (2008–2014)		123 (1.4%)
NorPD (2008–2016)		46 (0.52%)
NPR (2008–2014)		54 (0.62%)
Presented in at least one registry with an AUD-indicative entry	—	186 (2.1%)

Note. LPA-derived parental risk constellations are as follows: LP1, lowest education only; LP2, multiple risks + weekly binge drinking in both parents; LP3, low overall risk (reference); LP4, casual weekly drinking in both parents; LP5, multiple risks + maternal daily single-drink. LP = latent profile; LPA = latent profile analysis; HUNT = Nord-Trøndelag Health Studies; CPHR = Control and Payment of Health Reimbursements Registry; NorPD = Norwegian Prescription Database; NPR = Norwegian Patient Registry; AUD = alcohol use disorder.
[a] Based on Young-HUNT adolescent offspring self-reports. [b] Based on data obtained from Statistics Norway. [c] Based on HUNT parental self-reports.

LP2 (11 cases), 1.5% in LP3 (28 cases), 2.7% in LP4 (16 cases), and 5.1% in LP5 (4 cases).

Unadjusted Associations

Table 4, Model 0, shows estimates of bivariate associations between AUD in offspring and all study variables. The results indicate elevated likelihoods of future AUD in offspring from all risk constellations when compared with the low-risk constellation (LP3) but were statistically significant at the $p < .05$ level for two constellations only. Specifically, the largest and statistically significant effect sizes were observed for offspring from constellations marked by multiple risks, including LP2 (odds ratio [OR] = 3.10, 95% confidence intrval [CI] = 1.52, 6.31) and LP5 (OR = 3.53, 95% CI = 1.21, 10.33).

Adjusted Associations

Similar patterns were observed in the set of adjusted models, including the adjustments for demographics only (Table 4, Model 1); demographics and the offspring's alcohol intake during adolescence (Table 4, Model 2), and demographics, offspring's alcohol intake, and witnessing of parental intoxication during adolescence (Table 4, Model 3). Future AUD risk remained elevated in offspring from all risk constellations when compared with those from the lowest risk constellation (LP3) in all models, but the strength of the associations and corresponding significance levels were attenuated when compared with those obtained in bivariate analyses (Model 0). Specifically, the results from the fully adjusted Model 3 show that the LP2 and LP5 offspring were more than twice as likely to subsequently present with AUD than were the lowest risk LP3 offspring (Table 4, Model 3); however, only the estimates for LP2 remained statistically significant (adjusted odds ratio [aOR] = 2.34; 95% CI = 1.14, 4.85). Even though AUD risk was also elevated in LP1, LP4 and especially in LP5 offspring, the confidence intervals were wide and not statistically significant: The crude estimates for these constellations were strongly attenuated after accounting for adolescents' alcohol consumption and witnessing parental intoxication during adolescence in final Model 3.

In addition, future AUD was also significantly associated with offspring's alcohol intake and witnessing of parental alcohol intoxication during adolescence (Table 4, Model 3). Specifically, the odds of subsequent AUD were significantly greater among offspring who reported consuming 5 or more units of alcohol during the usual 2-week period (aOR = 1.78, 95% CI = 1.16, 2.73) and significantly lower among those who reported never having seen their parents intoxicated (aOR = .64, 95% CI = .46, .90).

Discussion

We examined the specific risk of future AUD in offspring within the context of non-AUD parental drinking in combination with other risk factors, using information from both parents, multiple and independently collected sources of data, and a prospective study design and key covariates adjustment. The results high-

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Table 4

Estimated AUD Risk in Offspring During 2008–2014 Study Period as a Function of Parental Risk Constellations During Adolescence, N = 8,773

			Adjusted estimates, nested models					
	Unadjusted estimates (Model 0)		Model 1: demographics + parental risk constellations		Model 2: Model 1 + offspring alcohol intake in adolescence		Model 3: Model 2 + offspring witnessing parental intoxication in adolescence	
Variables	*OR* (95% CI)	*p*	aOR (95% CI)	*p*	aOR (95% CI)	*p*	aOR (95% CI)	*p*
Gender (male)	1.35 (1.006, 1.81)	.05	1.34 (0.99, 1.81)	.05	1.30 (0.97, 1.75)	.08	1.30 (0.97, 1.75)	.08
Age at Young-HUNT participation	0.92 (0.85, 1.008)	.08	0.97 (0.88, 1.06)	.50	0.91 (0.82, 1.00)	.07	0.90 (0.81, 0.99)	.04
Age at study entry[a]	0.96 (0.93, 0.98)	<.001	0.96 (0.94, 0.99)	.01	0.96 (0.93, 0.98)	<.01	0.96 (0.93, 0.98)	<.01
Parental risk constellations								
LP1	1.44 (0.95, 2.17)	.08	1.46 (0.96, 2.21)	.07	1.40 (0.92, 2.11)	.11	1.31 (0.86, 1.98)	.20
LP2	3.10 (1.52, 6.31)	<.01	2.90 (1.42, 5.90)	.01	2.71 (1.32, 5.56)	.01	2.34 (1.14, 4.85)	.02
LP3 (reference)	—		—		—		—	
LP4	1.82 (0.98, 3.38)	.057	1.62 (0.87, 3.03)	.13	1.57 (0.84, 2.93)	.16	1.45 (0.76, 2.73)	.26
LP5	3.53 (1.21, 10.33)	.02	2.83 (0.97, 8.27)	.06	2.87 (0.97, 8.52)	.06	2.60 (0.86, 7.79)	.09
Adolescent alcohol intake/2 weeks[b]								
None (reference)	—		—		—		—	
1–5 drinks	0.84 (0.55, 1.28)	.40			1.11 (0.71, 1.73)	.65	1.01 (0.64, 1.59)	.96
5 or more drinks	1.46 (1.02, 2.10)	.04			1.99 (1.31, 3.04)	.01	1.78 (1.16, 2.73)	.01
No valid report	1.91 (1.23, 2.98)	<.01			2.34 (1.43, 3.50)	.01	2.07 (1.32, 3.26)	.01
Never witnessed parental intoxication[b]	0.61 (0.44, 0.85)	.01					0.64 (0.46, 0.90)	.01

Note. LPA-derived parental risk constellations are as follows: LP1, lowest education only; LP2, multiple risks + weekly binge drinking in both parents; LP3, low overall risk (reference); LP4, casual weekly drinking in both parents; LP5, multiple risks + maternal daily single drink. *OR* = odds ratio; CI = confidence interval; aOR = adjusted odds ratio; LP = latent profile; LPA = latent profile analysis; HUNT = Nord-Trøndelag Health Studies; AUD = alcohol use disorder.

[a] Age at first registry follow-up in 2008. [b] Adolescent offspring self-reports/Young-HUNT. All regression models accounted for family-clustering; intercepts are not shown.

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light the utility of person-centered analytical approaches in defining parental risk constellations and in understanding AUD risk in offspring from the general population and community samples, in which both the risk for and the prevalence of AUD may be comparably lower than in treatment or high-risk samples.

When compared with the lowest-risk constellation offspring, offspring from families marked by multiple risk factors had a greater likelihood of AUD during the study period. This is consistent with our theoretical framework and the cumulative risk model proposing that aggregation of risk factors may be one of the key mechanisms negatively affecting child development (Appleyard et al., 2005), including various alcohol-related problems (Lee et al., 2014; Pilowsky et al., 2009; Zufferey et al., 2007). Indeed, most of our identified constellations contained several risks; even the single-risk constellations (i.e., lowest educational attainment in LP1 and casual drinking manifested in LP4) can conceivably be conceptualized in terms of multiple risks because both parents were affected. The constellations associated with offspring's future AUD reflected diverse combinations of risk factors and risk levels, suggesting the need for dimensionality in cumulative risk research in general (McLaughlin et al., 2016) and implying etiological heterogeneity of AUD in this sample specifically (Chassin et al., 2013; Kendler, 2019).

Closer examination of the constellations and our final adjusted model revealed additional details. First, the constellation encompassing weekly binge drinking in both parents (LP2) remained most robustly associated with the subsequent AUD risk in offspring, echoing the salience of paternal problem drinking on offspring's drinking noted in previous studies (Haugland et al., 2013; Holst et al., 2019; Mares et al., 2012; Vermeulen-Smit et al., 2012). Interestingly, the sole study that used latent class analysis to identify patterns of parental drinking and explored their effects on offspring's alcohol use also noted the risk embedded in having both parents who are heavy weekend (i.e., binge) drinkers (Vermeulen-Smit et al., 2012). Second, previous studies reported similar effects of maternal heavy drinking or alcohol misuse (Alati et al., 2014; Haugland et al., 2013; Holst et al., 2019) but not of the maternal daily light drinking in combination with paternal drinking of similar quantity consumed in as half as many days, which emerged as part of our final risk constellation (LP5). Even though the significance levels was attenuated to the statistical trend level in the final model likely because of the relatively small cell size of this constellation and after controlling for covariates, these offspring were two and a half times more likely to present with AUD—the greatest magnitude of AUD risk we observed.

Most importantly, our results show that parental drinking did not have to reach AUD clinical levels to be meaningfully associated with offspring's AUD risk later in life. That is, even though some parental behaviors could be described in terms of binge drinking (i.e., LP2), other parents did drink more frequently but not necessarily excessively (i.e., LP5). Importantly, LP2 largely coincides with the most common drinking pattern in Norway where alcohol consumption takes place almost entirely during weekends but in high quantities (Horverak et al., 2007). These drinking behaviors, as noted above, remained most robustly associated with AUD risk in offspring once

combined with additional risk factors in at least one parent. Indeed, LP2 (as well as LP5) was characterized by additional risks, most notably mental health symptomatology in the mild disorder range in at least one parent. Considering that these specific risk constellations have been previously linked to the risk of anxiety and depression in offspring from this sample (Lund et al., 2019), our results can be interpreted in the context of developmental multifinality (in which a specific set of risk factors contributes to multiple outcomes, such as LP5 to both AUD and anxiety/depression in offspring from this sample) and multicausality and equifinality (in which a specific outcome arises from multiple sets of risks, such as AUD from both LP2 and LP5; Chassin et al., 2013; Cicchetti & Rogosch, 1996; Kendler, 2019). Overall, our results underscore the need to consider non-AUD drinking patterns in both parents in combination with other characteristics and with closer attention to the risk dimensions when examining the risk of future clinical outcomes such as AUD in offspring (McLaughlin et al., 2016; Vermeulen-Smit et al., 2012).

Finally, our results also suggest that future AUD in at least some offspring may not be entirely driven by socialization and witnessing parental drinking and/or intoxication, as suggested both in general literature (Cox et al., 2018; Smit et al., 2018; Smit et al., 2019) and in previous studies based on HUNT samples (Haugland et al., 2015; Haugland et al., 2013). Namely, even though witnessing parental intoxication was a significant risk factor for subsequent AUD in all offspring, such experiences did not entirely attenuate the negative effects of children's exposure to the specific multiple-risk constellation (LP2). Similarly, even though elevated alcohol use during adolescence was associated with doubled odds of future AUD, such behaviors during adolescence did not entirely prevail over the risks inferred by weekly binge drinking of both LP2 parents in combination with additional risks.

Methodological Considerations and Study Limitations

Our results are limited by the parameters of the original HUNT project, which includes the low prevalence of AUD among a community subsample of offspring from two-parent families in which both parents and offspring had participated in the HUNT studies as well as by the inherent limitations of the data sources (i.e., administrative health registries) we used. As such, the generalizability of our findings is limited.

First and foremost, the proportion of AUD cases in this sample was rather low during the study period. This may not be surprising, given that we examined a community sample of relatively young adults. It is thus possible that the number of cases would have increased if additional years of data beyond 2016 were included. In addition, administrative health records are by definition conservative (e.g., capturing the most severe cases, those self-selecting into treatment, etc.) and AUD may be especially underrecorded in Norwegian administrative databases, even though they generally capture the same phenomena as diagnostically assessed AUD in the general population (Torvik et al., 2018). Such biases are inherent in registry-based research (Elnegaard et al., 2017) and could not be addressed here. Nevertheless, our use of multiple health registries should at least to some extent alleviate the concerns about AUD underestimation while at the same time raising the confidence about diagnostic accuracy not always achieved through self-reports (Stockwell et al., 2004).

It should also be noted that we did not aim to generate AUD prevalence estimates but to examine the prospective associations between the offspring's AUD risk and parental-level risk factors examined in novel and complex patterns. Whereas we detected such associations, they should be interpreted with caution, given the low number of AUD cases in this community sample and especially in certain constellations. Indeed, the issue of low prevalence of AUD in this sample primarily translates into the issues of low power because there were only 11 cases (of 246 total) in LP2 and four cases (of 79 total) in LP5. These were our smallest risk constellations in terms of overall membership yet also the ones most robustly associated with future AUD risk in offspring—perhaps testifying to the strength of those risk effects, which were detected despite such small numbers. Even though not necessarily generalizable to the entire population, we contend that these constellations—in terms of their overall size, characteristics, and generated AUD cases—likely accurately captured these phenomena as manifested in two-parent families from the community.

Indeed, our sample of two-parent families with adolescent children who all participated in HUNT surveys was highly selective. Whereas this nonrepresentativeness does not necessarily hinder inferences concerning the observed prospective associations between the accumulated parental-level risks and AUD in offspring (Rothman et al., 2013), such inferences should take into consideration the meaning of those risks in the context of this particular sample. For example, the identified risk levels and risk constellations may be unique to this sample; these may take different forms in different sociocultural settings and have different associations with AUD risk in offspring (Chaiyasong et al., 2018; Laslett et al., 2017; Vermeulen-Smit et al., 2012). Furthermore, our key risks were captured through HUNT surveys only once during adolescence; how their magnitude, constellations, and associations with future AUD may differ across different developmental periods or lengths of exposure is not known. Similarly, our data sources and study design did not facilitate examination of other putative parental risk factors (i.e., other psychopathology, family violence, parenting styles, etc.) or gene-environment hypotheses. Further research is needed to address these questions, including the examination of developmental mechanisms and pathways through which these identified risk constellations impact future AUD outcomes in offspring.

It is also possible that some of the parents from our analytical sample met AUD criteria and that our main results were to some extent driven by such parents with clinically diagnosable disorders. However, past research shows that the majority of binge drinkers—such as those we observed in LP2, for example—do not necessarily meet diagnostic criteria for alcohol dependence (Esser et al., 2014). Furthermore, prior studies utilizing this sample indicate that HUNT responders (when compared with nonresponders) tended to be, if anything, characterized by better health and social outcomes in general (Knudsen et al., 2010; Langhammer et al., 2012; Torvik et al., 2012; Torvik et al., 2013) and by lower likelihood of substance use disorders in particular (Knudsen et al., 2010). Again, our key results should

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therefore be interpreted in the context of relatively well-functioning two-parent families from the community and the relatively low proportion of AUD cases in offspring. And even in such relatively well-adjusted families, parental weekly binge drinking alongside poor mental health and low educational attainment were predictive of future AUD in offspring.

Conclusion

Offspring from families marked by unique combinations of multiple risk factors in parents were more likely to present in the national health registries with AUD later in life, even after accounting for other early alcohol-relevant risk factors. Although the parental drinking we examined did not necessarily meet clinical or diagnostic criteria, weekly binge drinking in both parents was prospectively associated with the AUD risk in offspring when part of specific risk constellations. Examination of both maternal and paternal risk factors using person-centered analytical approaches can inform our understanding of future AUD risk in youth from community samples.

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