



Parental Alcohol Use and the Alcohol Misuse of their Offspring in a Finnish Birth Cohort: Investigation of Developmental Timing

Gilbert R. Parra¹ · Irina Patwardhan² · W. Alex Mason³ · Mary B. Chmelka² · Jukka Savolainen⁴ · Jouko Miettunen^{5,6} · Marjo-Riitta Järvelin^{6,7,8}

Received: 9 January 2020 / Accepted: 2 April 2020 / Published online: 6 May 2020
© Springer Science+Business Media, LLC, part of Springer Nature 2020

Abstract

There is a positive association between parental alcohol use and the alcohol use of their offspring. It is less clear whether this relation exists at different developmental periods. The purpose of the current study was to examine the associations between parental alcohol use at two developmental periods (prenatal and adolescence) and the alcohol misuse of their offspring at two developmental periods (adolescence and young adulthood). Data from the Northern Finland Birth Cohort 1986 (NFBC1986; $n = 6963$; 51% of offspring were girls) were used. The NFBC1986 is a population-based study of individuals born during a 1-year period in Finland. Multi-informant (parent, teacher, and youth) and multi-method (surveys and population registers) data were collected at four developmental periods (prenatal, childhood, adolescence, and young adulthood). The findings indicated that parents' alcohol use was stable from the prenatal period to adolescence. Mothers' and fathers' (based on mothers' perceptions) alcohol use during the prenatal period and adolescence were directly related to adolescents' heavy drinking. Prenatal alcohol use by mothers and fathers were related to young adults' alcohol use disorder indirectly (but not directly) through mothers' and fathers' alcohol use during adolescence and then through adolescents' heavy drinking. The results suggest that early and ongoing screening for alcohol use by mothers and fathers could help identify individuals at risk for heavy drinking and alcohol-related problems during adolescence and young adulthood.

Keywords Parental alcohol use · Heavy drinking · Alcohol use disorder · Adolescence · Young adulthood · Developmental timing

Introduction

Heavy alcohol use during adolescence and young adulthood is associated with a wide range of negative psychosocial outcomes (Danielsson et al. 2012; Fergusson et al. 2013). One important strategy for addressing this public health concern is continuing to refine knowledge of factors that place individuals at risk for heavy drinking and alcohol-related problems. There is a positive association between parental alcohol use and the alcohol use of their offspring (Rossow et al. 2016). It is less clear whether this relation exists at different developmental periods (Hussong et al. 2012). The purpose of the current study was to examine the associations between parental alcohol use at two developmental periods (prenatal and adolescence) and the alcohol misuse of their offspring at two developmental periods (adolescence and young adulthood).

The majority of studies examining the associations between parents' and their offspring's alcohol use assess lifetime history of parents' alcohol involvement or do not

✉ Gilbert R. Parra
gparra2@unl.edu

¹ Department of Child, Youth and Family Studies, University of Nebraska-Lincoln, 257 Louise Pound Hall, Lincoln, NE 68588, USA

² Boys Town Translational Research Center for Child and Family Studies, 378 Bucher Drive, Boys Town, NE 68010, USA

³ University of Tennessee Health Science Center, Memphis, TN, USA

⁴ Wayne State University, Detroit, MI, USA

⁵ Center for Life Course Health Research, University of Oulu, Oulu, Finland

⁶ Medical Research Center Oulu, Oulu University Hospital and University of Oulu, Oulu, Finland

⁷ Department of Epidemiology and Biostatistics, MRC PHE Centre for Environment and Health, School of Public Health, Imperial College London, London, UK

⁸ Unit of Primary Care, Oulu University Hospital, Oulu, Finland

specify a timeframe for parents' drinking (e.g., Diggs et al. 2017; Finan et al. 2018; Randolph et al. 2018). Moreover, in cross-sectional and relatively short-term longitudinal research designs, parental alcohol use typically is examined during the same developmental period as the alcohol use of their offspring (e.g., Rossow et al. 2016). Together, these studies have provided important contributions to knowledge of the relation between parents' and their offspring's alcohol use. The approaches that have generally been used to assess parents' alcohol use, however, limit the conclusions that can be drawn about *when* during development parents' alcohol use is most strongly related to the alcohol misuse of their offspring (i.e., developmental timing). A focus on developmental timing has been highlighted as critically important to understanding whether key risk factors have long-term (enduring) or relatively short-term (transient) effects (Haltigan et al. 2013). To examine developmental timing, a research design that assesses parents' alcohol use during at least two developmentally distinct points in time is needed.

Studies exist that allow conclusions to be drawn about when during development parents' alcohol use is related to the alcohol misuse of their offspring. Some of these longitudinal studies used a lifetime history measure of parents' alcohol involvement at a baseline assessment and then obtained time specific (usually past year) assessments of parents' alcohol involvement at later points in time. For example, Hussong et al. (2012) measured mothers' and fathers' lifetime alcohol use disorder in late childhood/early adolescence (baseline assessment). In addition, at baseline and two additional annual waves, both parents completed a past year measure of alcohol use disorder symptoms. The findings indicated that both mothers' and fathers' lifetime history of alcohol use disorder in late childhood/early adolescence were related to adolescents' heavy alcohol use. Fathers', but not mothers', lifetime history of alcohol use disorder in late childhood/early adolescence also was associated with adolescents' alcohol use. Importantly, neither mothers' nor fathers' past year alcohol use disorder symptoms was related to either adolescents' alcohol use or adolescents' heavy drinking. In another study, lifetime history of maternal alcohol use disorder when children were age 9 was not directly related to children's early onset of alcohol use (age 11; Johnson et al. 2019). Lifetime history of maternal alcohol use disorder was related to children's early onset of alcohol use indirectly through several mechanisms including financial strain, maternal depressive symptoms, and negative family climate. Interestingly, lifetime history of maternal alcohol use disorder when children were age 9 was not related to past year maternal alcohol use when children were ages 9, 10, or 11. Past year maternal alcohol use when children were age 11 was not directly related to child early onset of alcohol use (Johnson et al.

2019). Together, these studies indicate that, after accounting for parents' lifetime history of alcohol use through late childhood, parents' use during adolescence may not be directly related to adolescents' alcohol use.

Relatively few long-term longitudinal studies have examined the impact of alcohol use by parents during a specific developmental period on adolescents' and young adults' alcohol use and alcohol related problems. There are, however, several notable exceptions. For example, Mahedy et al. (2018) found that mothers' and fathers' alcohol use when children were age 12 were associated with adolescents' alcohol use at age 14 and adolescents' alcohol use at age 18. In addition, Alati et al. (2008) found an increased risk of early drinking during adolescence for those who had been exposed to maternal alcohol use during pregnancy. Studies that have measured parents' alcohol use prior to adolescents' alcohol use onset found that parents' alcohol use was positively related to youth's alcohol initiation (Cranford et al. 2010; Duncan et al. 2006; Latendresse et al. 2008) and later use (Latendresse et al. 2008). Given the relatively small literature and complex set of findings, additional long-term longitudinal studies are needed to better understand how developmental timing of parents' alcohol use impacts adolescents' and young adults' alcohol misuse.

The prenatal period and adolescence are two times in development when parents' alcohol use is likely to impact adolescents' and young adults' alcohol misuse. Maternal drinking during pregnancy has been found to have deleterious effects on the physical, cognitive, behavioral, and emotional outcomes of offspring (Easey et al. 2019). There is a surprisingly small literature on the association between maternal drinking during pregnancy and adolescents' and young adults' alcohol misuse. As noted, there is evidence that a relation exists (Baer et al. 2003; Cornelius et al. 2016; Goldschmidt et al. 2019). Cornelius et al. (2016) highlighted research that suggests offspring exposed to maternal drinking during pregnancy may be particularly vulnerable to the effects of alcohol. It is also likely that genetic/biological factors other than prenatal alcohol exposure contribute to the association between maternal prenatal alcohol use and adolescents' heavy drinking (Kendler et al. 2015). A review of the literature did not reveal any studies that examined the relation between fathers' prenatal alcohol use and adolescents' or young adults' alcohol misuse. Given the extensive literature on the heritability of alcohol misuse, genetic/biological factors are likely one set of influences that explain this association (Verhulst et al. 2015). Notably, a recent review of 11 studies suggested that paternal alcohol consumption during pregnancy is related to maternal alcohol consumption during pregnancy (McBride and Johnson 2016).

Heavy alcohol use often begins during adolescence (Chung et al. 2018). Research has shown that parents'

alcohol use during adolescence is associated with adolescents' alcohol misuse (Homel and Warren 2019). Both genetic and psychosocial influences appear to play a role in this association. Adolescents' alcohol use is, to some degree, heritable (Waaktaar et al. 2018), particularly as involvement progresses to heavier and more problematic use (Maes et al. 1999). Parenting and family environment factors also likely help explain associations of mothers' and fathers' alcohol use with that of their adolescent offspring. Parents who drink convey attitudes favorable toward alcohol and model its use to their children, and often provide access to alcoholic beverages in the home, all of which are well-established risk factors for alcohol use during adolescence (Hawkins et al. 1992).

Most research on the relations between parents' alcohol use and the alcohol use of their offspring has not included the assessment of both mothers' and fathers' alcohol use or has combined parents' reports (Poelen et al. 2009). The inclusion of information about both parents' alcohol involvement is important because research indicates that both maternal and paternal alcohol involvement impact the alcohol use of their offspring (Kerr et al. 2012). The relative impact of each parent's alcohol use, however, is poorly understood. Information about both mothers' and fathers' alcohol use may better account for genetic/biological risk than either mothers' or fathers' alcohol use alone. In addition, consideration of mothers' and fathers' alcohol use is important because each parent could model different levels of alcohol consumption and alcohol-related behaviors. Parents also may communicate different levels of approval of their offspring's alcohol use and/or engage in different alcohol-related parenting practices (e.g., monitoring alcohol use; Donovan 2018).

Alcohol misuse during adolescence and young adulthood is multidetermined (Chassin et al. 2013). To rule out alternative explanations for the unique associations between parental alcohol use and the alcohol use of their offspring, it is important to account for key risk factors. Youth gender is one well-established risk factor for alcohol misuse. Research consistently indicates that boys and men are more likely to engage in heavy drinking and experience alcohol related problems than girls and women (Schulte et al. 2009).

Contextual factors during the prenatal/birth period are risk factors for the alcohol misuse of youth. A cumulative risk approach has been put forth as a method for capturing youth's level of risk given that risk factors tend to co-occur (Evans et al. 2013). In this approach, a cumulative risk index is created by taking the sum of multiple dichotomized risk variables. Research indicates that cumulative contextual risk during the prenatal/birth period is related to a wide range of psychosocial difficulties including elevated levels of alcohol involvement among youth (Evans et al. 2013; Mason et al. 2016).

Children's proneness to behavioral undercontrol is a particularly strong predictor of alcohol misuse across development (Chassin et al. 2013). Youth who engage in externalizing behaviors such as teasing others and disobedience at a young age are at heightened risk of experiencing alcohol related problems later in development (Kendler et al. 2013). Moffitt's life course theory of delinquency (1993) posits that one pathway toward persistent behavior problems including alcohol misuse involves the existence of elevated levels of externalizing behaviors during childhood.

Peer relationships are important for the socialization of drinking behaviors during adolescence (Donovan 2018). Research indicates that adolescents who are rejected by prosocial peers tend to affiliate with peers who engage in deviant behaviors (Kelly et al. 2012). Deviant peer affiliation is a robust predictor of alcohol misuse during adolescence and young adulthood (Cambron et al. 2018).

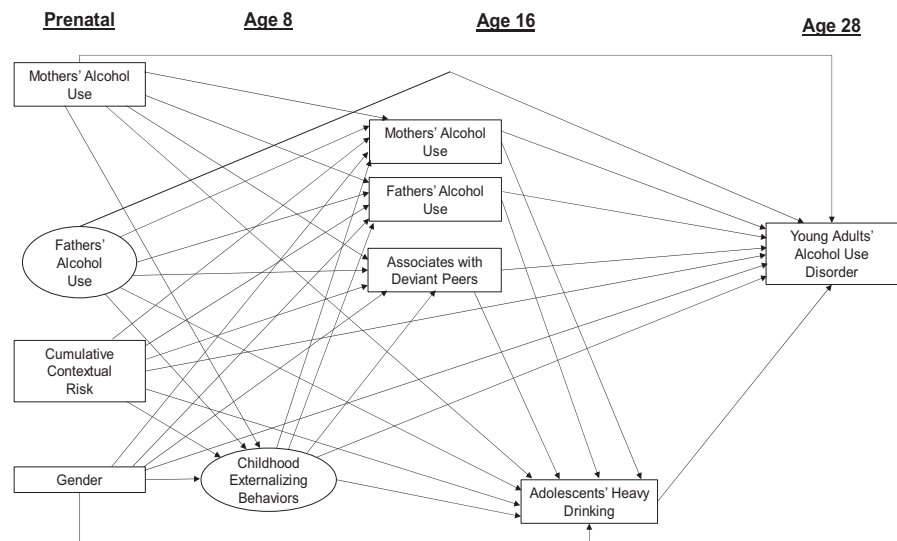
Current Study

There is an extensive literature documenting the positive association between parental alcohol use and the alcohol misuse of their offspring. However, relatively few studies have investigated this relation from a developmental perspective. A developmental perspective is important because it can provide key information about whether parental alcohol use at one point in time has enduring or transient effects and whether there are periods in development when parental alcohol use is particularly influential. A long-term longitudinal study that spans multiple developmental periods is needed to investigate these issues. Given that research indicates both mothers' and fathers' alcohol use are related to the alcohol use of their offspring, information about both mothers' and fathers' alcohol use should be considered.

The current study was guided by two questions. First, are mothers' and fathers' alcohol use at two developmental periods (prenatal and adolescence) unique predictors of their offspring's alcohol misuse at two developmental periods (adolescence and young adulthood)? Second, are there relations from mothers' and fathers' alcohol use to offspring's alcohol misuse that reflect complex developmental pathways?

A multivariate structural equation model was designed to answer these two questions (see Fig. 1). As shown, it was expected that mothers' and fathers' alcohol use during the prenatal period and adolescence would be directly related to adolescents' heavy drinking and young adults' alcohol use disorder. It was also hypothesized that mothers' and fathers' prenatal alcohol use would be indirectly related to adolescents' heavy drinking and young adults' alcohol use disorder through mothers' and fathers' alcohol use during adolescence. It was further expected that mothers' and fathers' alcohol use during adolescence would be directly

Fig. 1 Conceptual model



and indirectly (through adolescents' heavy drinking) related to young adults' alcohol use disorder.

The model included factors in addition to parents' alcohol use that have been associated with adolescents' and young adults' alcohol misuse. These variables were conceptualized as covariates and included to account for factors at different developmental periods that are related to adolescents' and young adults' alcohol misuse. The factors were gender, cumulative contextual risk in the prenatal/birth period, conduct problems in childhood, and associations with deviant peers in adolescence.

Methods

Participants and Procedures

This study used data from the Northern Finland Birth Cohort 1986 (NFBC1986), a population-based study of individuals born during a one-year period in Finland. The original cohort included 9432 children born alive, whose expected date of birth fell between July 1, 1985 and June 30, 1986 (98.5% of all deliveries taking place in the two northernmost provinces of Finland). Details regarding the NFBC1986 data collection are available elsewhere (Hurtig et al. 2007; Miettunen et al. 2014). The current analyses used data collected at prenatal/birth, childhood, adolescence, and young adulthood.

Prenatal/birth

At the study's outset, mothers were provided a prenatal background questionnaire at their first antenatal visit to the local prenatal clinic (on average at the 12th

gestational week), to be returned by their 24th gestational week. Midwives completed a subsequent pregnancy questionnaire at mothers' last antenatal visit to the clinic, or during the first home visit by the midwife after delivery. Midwives and/or medical staff at the prenatal clinics completed additional information on pregnancy and delivery.

Childhood

In 1993–1994, when children were 7–8 years old, a postal questionnaire regarding school behavior and performance was provided to each child's homeroom teacher in the spring of their first year of school.

Adolescence

In 2001–2002, when the participants were 15–16 years old, separate postal questionnaires were mailed to the adolescents and their parents. The parent survey contained items about the mothers', fathers', and adolescents' health, social background and living habits. The survey was completed either by the parents together (54%), mother (39.8%), father (4.3%) or other (1.9%). The adolescent survey covered topics such as family, school, behavior, health, and activities. During this same time, adolescents were also invited to a clinical examination, where they filled out an additional questionnaire regarding eating habits, stress, sexual behavior, substance use, and mental well-being. By this time, 217 were deceased, immigrated, or had an unknown address. Written informed consent was obtained from parents and assent from children. The NFBC1986 study was approved by the ethical committee of the Northern Ostrobothnia Hospital District.

Young adulthood

Substance-related diagnoses through 2013 were available through Finnish national registers. These data can be missing due to immigration or deaths, but otherwise are complete. The final analysis sample of 6963 participants was comprised of all consented youth with adolescent self-report data (74% of live births), with one randomly selected child from each set of participating non-singletons. Fifty-one percent of the participants in the final analysis sample were girls, with an average age in adolescence of 16.0 (14.58 to 16.96) years.

Measures

Mothers' alcohol use—prenatal

On the prenatal/pregnancy questionnaires, mothers self-reported about their consumption of alcohol during pregnancy. If the mother responded “yes” to the question, “Have you had alcohol drinks during this pregnancy?”, the following quantity items were collected: “Amount of alcohol drinks before 16th week of pregnancy”, and “Amount of alcohol drinks after 16th week of pregnancy”. Response options for both quantity items were (1) “5 drinks per week”, (2) “5–20 drinks per week”, and (3) “over 20 drinks per week”. Of those mothers who drank alcohol while pregnant ($n = 787$, 11.8%), over 95% reported 5 drinks per week and none reported over 20 drinks weekly. Due to this limited response variation, each quantity variable was collapsed into a dichotomous prenatal alcohol use (1) or non-use (0) variable. The mothers' alcohol use—prenatal variable was coded 1 if either the dichotomous before 16th week of pregnancy or after 16th week of pregnancy variables were coded 1 and coded 0 if both the dichotomous before 16th week of pregnancy and after 16th week of pregnancy variables were coded 0.

Fathers' alcohol use—prenatal

Fathers' prenatal alcohol use was a two-indicator latent variable representing the fathers' alcohol use during pregnancy, as reported by mothers on the prenatal/pregnancy questionnaires. If mother responded “yes” to the question, “Does your husband drink alcohol?”, first frequency and then quantify items were collected. *Fathers' prenatal alcohol frequency* was collected using the question, “How often does he (husband) drink alcohol?”, with response options of (1) “daily”, (2) “once a week”, (3) “2–3 times per month”, (4) “once a month”, and (5) “less often”. This variable was reverse coded so that higher scores represent higher frequency of drinking. Further, after passing two

levels of qualifying questions, *fathers' prenatal alcohol quantity* was collected using the question, “Can you give figures for the amount that he (husband) drinks?”, with response options of (1) “less than 5 drinks a week”, (2) “5 to 20 drinks a week”, and (3) “over 20 drinks a week”. If the father did not use alcohol during the mother's pregnancy, then both the frequency and quantity items were coded as 0. Internal consistency for these two items was acceptable ($\alpha = 0.77$). Research has shown that wives' perceptions of their husbands' alcohol use is related to husbands' reported alcohol use (Rodriguez and Neighbors 2015). Moreover, mothers' perceptions provide important information about their husbands' alcohol use.

Mothers' alcohol use when adolescents were age 16

Mothers' alcohol use when adolescents were age 16 is a manifest variable from the parent postal questionnaire. The item was “Does the mother currently use any alcohol?”. Response options were (1) “Has never used”, (2) “No, has stopped”, (3) “Yes, less than once a month”, (4) “Yes, once a month”, (5) “Yes, 2–3 times a month”, (6) “Yes, once a week”, (7) “Yes, a few times a week”, and (8) “Yes, daily”. Response options 1 and 2 were recoded to 0 because both reflect no current drinking. Response options 3, 4, 5, 6, 7, and 8 were recoded to 1, 2, 3, 4, 5 and 6, respectively. Thus, this variable could range from 0 to 6.

Fathers' alcohol use when adolescents were age 16

Fathers' alcohol use when adolescents were age 16 is a manifest variable from the parent postal questionnaire. The item was “Does the father use any alcohol?”, with the same response set as the parallel mother item. Consistent with the mother alcohol use at age 16 variable, response options 1 and 2 were recoded to 0 because both reflect no current drinking. Response options 3, 4, 5, 6, 7, and 8 were recoded to 1, 2, 3, 4, 5 and 6, respectively. Thus, this variable could range from 0 to 6.

Externalizing behaviors

The measure of externalizing behaviors was represented as a latent variable with four indicators ($\alpha = 0.85$) from the teacher rated Rutter Children Behavior Questionnaire (RCBQ; Rutter 1967): *destroys* (“Often ruins and breaks his/her own or other's things”); *fights* (“Fights every so often or quarrels often with other children”), *disobedient* (“Is often disobedient”); and *teases* (“Teases other children”). Response options for all items were (1) “Doesn't apply”, (2) “Applies somewhat”, and (3) “Certainly applies”.

Associates with deviant peers

Peer deviance was measured using 1 item from the adolescent postal questionnaire. The item was “I hang around with kids who get in trouble”, with response options of (1) “not true”, (2) “somewhat or sometimes true”, and (3) “very true or often true”.

Adolescents’ heavy drinking

Adolescents’ heavy drinking was measured with 1 item from the adolescent clinical exam survey. The question was “How many times in your life have you been drunk?”, with the following response options: (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 times or more”.

Young adults’ alcohol use disorder

Young adults’ alcohol use disorder is a dichotomous indicator based on ICD-10 substance abuse and dependence *diagnoses* available from national health registries. These data reflect diagnoses through 2013, or when participants were 27–28 years of age. Four registers provided source data: (1) Care Register for Health Care (inpatient treatments until 2013), (2) Finnish outpatient registers (specialized care 1998–2013, primary care 2011–2013), (3) Social Insurance Institution registers: reimbursable medicines (until 2005), and (4) Finnish Center for Pensions: disability pensions (until 2013).

Cumulative contextual risk

A measure of cumulative contextual risk was created by summing up the scores from seven dichotomous parent-reported characteristics of family structure and socioeconomic disadvantage. With the exception of birth weight (provided by medical staff at time of delivery), all measures were collected through the pregnancy questionnaires completed by mothers (for details about the indicators of the cumulative contextual risk variable, see Parra et al. 2017). The indicators, described in more detail below, included: (1) *low birth weight*, (2) *teenage mother*, (3) *single mother*, (4) *multiple unions*, (5) *low maternal education*, (6) *economic exclusion*, and (7) *material deprivation*. Each indicator was coded 1 to represent presence of the risk and 0 to represent absence of the risk. It is notable that cumulative contextual risk generated an observed range of 0–5 but was recorded to 0–3 due to low frequency of high numbers of risks in this sample.

Low birth weight was coded 1 if the child was born weighing under 2500 grams (Zegers-Hochschild et al. 2009). *Teenage mother* was coded 1 if the mother gave birth

to the participant at age 19 or younger. *Single mother* was coded 1 if the mother was unmarried, widowed, divorced, or not cohabitating with a partner. *Multiple unions* was coded 1 if the mother had at least one prior registered union, such as marriage or cohabitation. *Low maternal education* was coded 1 if the mother completed fewer than 9 years of comprehensive schooling (Grades 1–9). *Economic exclusion* was coded 1 if the highest occupational status of the adult member of household was either unskilled worker (manual labor), unemployed, or on disability pension. *Material deprivation* was coded 1 if the household had fewer than two of these four items: washing machine, telephone, flushing toilet, or indoor bathroom.

Gender

Participants’ gender was coded 1 for boys and 0 for girls.

Data Analyses

A multivariate model structural equation model was estimated using *Mplus* 8.3 (Muthén and Muthén 1998–2017). The weighted least squares mean- and variance-adjusted (WLSMV) estimator was used to derive parameter estimates. First, a measurement model was conducted to examine overall model fit and whether manifest indicators of the two latent variables (prenatal fathers’ alcohol use and childhood externalizing behaviors) had acceptable factor loadings. Second, the hypothesized structural model was estimated (see Fig. 1). Key direct paths were (1) prenatal mothers’ and fathers’ alcohol use to adolescents’ heavy drinking, (2) prenatal mothers’ and fathers’ alcohol use to young adults’ alcohol use disorder, (3) mothers’ and fathers’ alcohol use when adolescents were age 16 to adolescents’ heavy drinking, and (4) mothers’ and fathers’ alcohol use when adolescents were age 16 to young adults’ alcohol use disorder. The model includes several indirect pathways that could add to understanding of the complex relations between parents’ alcohol use at two developmental periods and offspring’s alcohol misuse at two developmental periods. Specifically, indirect pathways from prenatal mothers’ and fathers’ alcohol use to young adults’ alcohol use disorder through mothers’ and fathers’ alcohol use when adolescents were age 16 and then through adolescents’ heavy drinking were estimated. To examine indirect effects, bootstrapped confidence intervals (95%) based on 5000 bootstrap samples were computed (MacKinnon et al. 2004).

Sample sizes for each measure included in the study appear in Table 1. The amount of missing data ranged from 0% (gender, cumulative contextual risk, and young adults’ alcohol use disorder) to 36.79% (fathers’ prenatal alcohol quantity). The largest amount of missing data was for

Table 1 Descriptive statistics and estimates of associations for study variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Prenatal														
1. Mothers' alcohol use ^a														
Fathers' alcohol use														
2. Frequency	0.26*													
3. Quantity	0.27*	0.80*												
4. Cumulative contextual risk	0.04*	0.04*	0.08*											
5. Gender ^a	-0.01	-0.01	-0.02	-0.03*										
Age 8														
Childhood externalizing behaviors														
6. Breaks things	0.03*	0.02	0.02	0.05*	0.16*									
7. Fights or quarrels	0.01	0.03*	0.03*	0.07*	0.21*	0.47*								
8. Is disobedient	0.01	0.04*	0.03	0.07*	0.22*	0.50*	0.65*							
9. Teases other children	0.02	0.03*	0.01	0.07*	0.23*	0.48*	0.75*	0.67*						
Age 16														
10. Mothers' alcohol use	0.23*	0.42*	0.44*	-0.02	-0.02	0.00	0.02	0.01	0.01					
11. Fathers' alcohol use	0.18*	0.55*	0.55*	0.00	-0.02	0.01	0.02	0.02	0.02	0.69*				
12. Associates with deviant peers	0.03*	0.08*	0.09*	0.03*	0.01	0.04*	0.05*	0.04*	0.04*	0.07*	0.08*			
13. Adolescents' heavy drinking	0.11*	0.28*	0.35*	0.08*	-0.05*	0.05*	0.10*	0.08*	0.08*	0.26*	0.29*	0.28*		
Age 28														
14. Young adults' alcohol use disorder ^a	0.02	0.05*	0.04*	0.03*	0.04*	0.06*	0.06*	0.05*	0.06*	0.03	0.02	0.09*	0.09*	
Mean/prevalence	9.2%	1.48	0.73	0.33	49.0%	1.06	1.21	1.20	1.18	1.93	2.57	1.20	2.41	2.4%
Standard deviation	-	1.25	0.67	0.62	-	0.26	0.47	0.46	0.43	1.53	1.75	0.45	2.19	-
Range	0-1	0-4	0-3	0-3	0-1	1-3	1-3	1-3	1-3	0-6	0-6	1-3	0-6	0-1
<i>n</i>	6471	5932	4401	6963	6963	6441	6440	6431	6423	5844	5490	6476	6152	6963

Associations between two dichotomous variables are Phi values

* $p < 0.05$

^aPoint Biserial Correlations unless otherwise noted

measures of fathers' alcohol use. Attrition analyses were conducted in which individuals who had missing data on a specific variable were compared to those who had complete data on that variable. Comparisons were made on the three variables that had complete data (i.e., gender, cumulative contextual risk, and alcohol use disorder during young adulthood). Chi-square tests were conducted for gender and young adults' alcohol use disorder. Independent samples *t*-tests were conducted for cumulative contextual risk. The findings from attrition analyses indicated that there was one (out of 11) gender difference between those with missing data and those without missing data. Specifically, boys were more likely to have missing data on the measure of associates with deviant peers than girls. There were six differences on the cumulative contextual risk variable between those with missing data and those without missing data. Specifically, individuals with missing data on the measures of prenatal mothers' alcohol use, prenatal fathers' alcohol use quantity, mothers' alcohol use when adolescents were age 16, fathers' alcohol use when adolescents were age 16, associates with deviant peers, and adolescents'

heavy drinking tended to have higher scores on the measure of cumulative contextual risk than those without missing data on those measures. There were seven differences on the young adults' alcohol use disorder measure between those with missing data and those without missing data. Specifically, individuals with an alcohol use disorder diagnosis during young adulthood were less likely to have missing data on the measures of prenatal mothers' alcohol use, prenatal fathers' alcohol use frequency, prenatal fathers' alcohol use quantity, mothers' alcohol use when adolescents were age 16, fathers' alcohol use when adolescents were age 16, associates with deviant peers, and adolescents' heavy drinking than those individuals without an alcohol use disorder diagnosis. Together, findings from the attrition analyses indicated there were differences between individuals with missing data on study measures and those without missing data. As noted, the WLSMV estimator in Mplus was used. "WLSMV allows missingness to be a function of observed covariates (but not observed outcomes as in ML estimation) and is more efficient in dealing with missing categorical data than listwise deletion"

(Savolainen et al. 2018, p. 19). The WLSMV estimator allowed the sample size in the structural model to be 6471.

Results

Descriptive Statistics

Descriptive statistics and estimates of associations for study variables are reported in Table 1. As shown, mothers' and fathers' (based on mothers' perceptions) prenatal alcohol use was positively associated with adolescents' heavy drinking (r s ranged from 0.11 to 0.35, p s < 0.05). Further, both mothers' and fathers' (based primarily on mothers' perceptions) alcohol use when adolescents were age 16 were positively associated with adolescents' heavy drinking. Fathers', but not mothers', prenatal alcohol use was significantly associated with young adults' alcohol use disorder. Neither mothers' nor fathers' alcohol use when adolescents were age 16 was associated with young adults' alcohol use disorder. Adolescents' heavy drinking was related to young adults' alcohol use disorder.

Multivariate Model

Prior to estimating the hypothesized multivariate model, a measurement model was estimated that included two latent variables (prenatal fathers' alcohol use and externalizing childhood behaviors at age 8) and correlations among latent

and manifest variables in the model. Results from the measurement model indicated good model fit: χ^2 ($n = 6963$) = 93.11, $df = 40$, $p < 0.001$, RMSEA = 0.01, SRMR = 0.01, CFI = 0.996, TLI = 0.99. Factor loadings for the prenatal fathers' alcohol use latent variable were 0.86 and 0.90 (standardized coefficients) for frequency and quantity variables, respectively. Factor loadings for the externalizing childhood behaviors at age 8 latent variable were 0.85, 0.59, 0.83, and 0.81 (standardized coefficients) for the teases other children, breaks things, fights or quarrels, and is disobedient variables, respectively. All factor loadings were statistically significant.

The hypothesized multivariate model provided a reasonable fit to the data: χ^2 ($n = 6471$) = 1257.07, $df = 46$, $p < 0.001$, RMSEA = 0.06, SRMR = 0.05, CFI = 0.90, TLI = 0.81. The modification indices suggested adding two parameters: a correlation between the residual variances of mothers' alcohol use when adolescents were age 16 and fathers' alcohol use when adolescents were age 16 and a correlation between the residual variances of breaks things and is disobedient. Both of those correlations were estimated. The modified model provided a good fit to the data: χ^2 ($n = 6471$) = 910.49, $df = 44$, $p < 0.001$, RMSEA = 0.06, SRMR = 0.04, CFI = 0.93, TLI = 0.86.

Parameter estimates for the multivariate model are presented in Table 2. To highlight key findings, Fig. 2 presents only the statically significant paths. The findings revealed that mothers' ($\beta = 0.79$) and fathers' ($\beta = 0.44$) prenatal alcohol use were significantly associated with mothers'

Table 2 Parameter estimates for multivariate structural equation model

	Childhood externalizing behaviors (age 8)	Mothers' alcohol use (age 16)	Fathers' alcohol use (age 16)	Associates with deviant peers (age 16)	Adolescents' heavy drinking (age 16)	Young adults' alcohol use disorder (age 28)
Prenatal						
Mothers' alcohol use	0.07/0.03	0.79/1.21*	0.61/1.06*	0.09/0.04*	0.24/0.51*	0.05/0.05
Fathers' alcohol use	0.03/0.01	0.44/0.65*	0.59/1.00*	0.10/0.04*	0.23/0.48*	0.09/0.08
Cumulative contextual risk	0.09/0.06*	-0.04/-0.10*	-0.02/-0.06	0.03/0.02*	0.06/0.20*	0.04/0.06
Gender	0.55/0.21*	-0.02/-0.03	-0.05/-0.08	-0.01/0.00	-0.13/-0.29*	0.14/0.14*
Age 8						
Childhood externalizing behaviors		0.01/0.03	0.01/0.04	0.06/0.07*	0.09/0.50*	0.13/0.34*
Age 16						
Mothers' alcohol use					0.08/0.12*	0.05/0.03
Fathers' alcohol use					0.06/0.08*	-0.09/-0.05
Associates with deviant peers					0.25/1.24*	0.10/0.22*
Adolescents' heavy drinking						0.18/0.08*

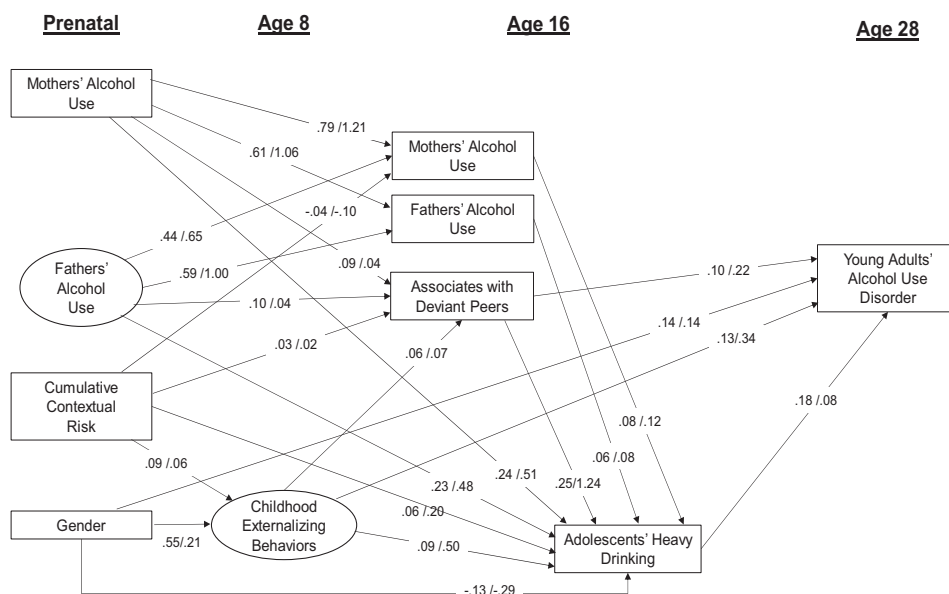
STDY standardization parameters reported for dichotomous covariates

$N = 6471$

Values are standardized/unstandardized coefficients

* $p < 0.05$

Fig. 2 Multivariate Structural Equation Model. Note. $n = 6471$. Parameters are standardized/unstandardized estimates. STDY standardization parameters reported for dichotomous covariates. All parameters shown are statically significant ($p < 0.05$)



alcohol use when adolescents were age 16. Similarly, mothers' ($\beta = 0.61$) and fathers' ($\beta = 0.59$) prenatal alcohol use were significantly associated with fathers' alcohol use when adolescents were age 16.

The results further indicated that mothers' ($\beta = 0.24$) and fathers' ($\beta = 0.23$) prenatal alcohol use were significantly associated with adolescents' heavy drinking. Both mothers' ($\beta = 0.08$) and fathers' ($\beta = 0.06$) alcohol use when adolescents were age 16 were significantly associated with adolescents' heavy drinking. There were no significant associations between mothers' ($\beta = 0.05$) or fathers' ($\beta = 0.09$) prenatal alcohol use and young adults' alcohol use disorder. Neither mothers' ($\beta = 0.05$) nor fathers' ($\beta = -0.09$) alcohol use when adolescents were age 16 was directly associated with young adults' alcohol use disorder. The findings showed that adolescents' heavy drinking was related to young adults' alcohol use disorder ($\beta = 0.18$).

The results related to indirect effects are shown in Table 3. As can be seen, all but 1 indirect effect tested were found to be statistically significant. Notably, the indirect pathways from prenatal mothers' alcohol use to both mothers' and fathers' alcohol use when adolescents were age 16 to adolescents' heavy drinking to young adults' alcohol use disorder were found to be significantly different from zero. Similarly, the indirect pathways from prenatal fathers' alcohol use to both mothers' and fathers' alcohol use when adolescents were age 16 to adolescents' heavy drinking to young adults' alcohol use disorder were found to be significantly different from zero. A statistically significant indirect pathway from fathers' prenatal alcohol use to associates with deviant peers to adolescents' heavy drinking to young adults' alcohol use disorder was found.

In terms of findings for the covariates, cumulative contextual risk had significant positive associations with childhood externalizing behaviors ($\beta = 0.09$), associates with deviant peers ($\beta = 0.03$), and adolescents' heavy drinking ($\beta = 0.06$) as well as a significant negative association with mothers' alcohol use when adolescents were age 16 ($\beta = -0.04$). Coefficients for gender as a covariate showed that being male increased risk for childhood externalizing behaviors ($\beta = 0.55$) and young adults' alcohol use disorder ($\beta = 0.14$) as well as decreased risk for adolescents' heavy drinking ($\beta = -0.13$). Childhood externalizing behavior was related to associates with deviant peers ($\beta = 0.06$), adolescents' heavy drinking ($\beta = 0.09$), and young adults' alcohol use disorder ($\beta = 0.13$). Associates with deviant peers was associated with adolescents' heavy drinking ($\beta = 0.25$) and young adults' alcohol use disorder ($\beta = 0.10$).

Discussion

Parents' alcohol use is positively associated with the alcohol misuse of their offspring. Less clear is whether parental alcohol use at one point in time has long-term (enduring) or relatively short-term (transient) effects and whether there are periods in development when parental alcohol use is particularly influential. The investigation of parental alcohol use from a developmental perspective can help refine knowledge of offspring's risk for alcohol misuse. That refined understanding has the potential to provide key insights into the optimal timing of intervention efforts designed to address alcohol-related problems during

Table 3 Indirect effects based on pathways from parents' alcohol use at two developmental periods to the alcohol misuse of their offspring at two developmental periods

	Estimate	95% CI	
		Lower	Upper
Prenatal mothers' alcohol use to			
1. Mothers' alcohol use (16) to Adolescents' heavy drinking	0.141 ^a	0.072	0.212
2. Mothers' alcohol use (16) to Adolescents' heavy drinking to Young adults' alcohol use disorder	0.012 ^a	0.005	0.021
3. Fathers' alcohol use (16) to Adolescents' heavy drinking	0.082 ^a	0.018	0.147
4. Fathers' alcohol use (16) to Adolescents' heavy drinking to Young adults' alcohol use disorder	0.007 ^a	0.001	0.014
5. Associates with deviant peers to Adolescents' heavy drinking to Young adults' alcohol use disorder	0.004	0.000	0.009
Prenatal Fathers' alcohol use to			
6. Mothers' alcohol use (16) to Adolescents' heavy drinking	0.076 ^a	0.039	0.112
7. Mothers' alcohol use (16) to adolescents' heavy drinking to Young adults' alcohol use disorder	0.006 ^a	0.003	0.011
8. Fathers' alcohol use (16) to Adolescents' heavy drinking	0.076 ^a	0.017	0.135
9. Fathers' alcohol use (16) to Adolescents' heavy drinking to Young adults' alcohol use disorder	0.006 ^a	0.001	0.013
10. Associates with deviant peers to Adolescents' heavy drinking to Young adults' alcohol use disorder	0.004 ^a	0.002	0.007
Mothers' alcohol use (16) to			
11. Adolescents' heavy drinking to Young adults' alcohol use disorder	0.010 ^a	0.004	0.017
Fathers' alcohol use (16) to			
12. Adolescents' heavy drinking to Young adults' alcohol use disorder	0.006 ^a	0.001	0.013

n = 6471

CI confidence intervals

^aIndicates estimate is significantly different from 0 based on bootstrapped 95% CI not including 0

adolescence and young adulthood. The present study examined the associations between parental alcohol use at two developmental periods (prenatal and adolescence) and the alcohol misuse of their offspring at two developmental periods (adolescence and young adulthood). To address issues of developmental timing, data from a large, long-term longitudinal (prenatal to age 28) Finnish birth cohort study were used. Most research on the relations between parents' alcohol use and the alcohol use of their offspring has not included the assessment of both mothers' and fathers' alcohol use or has combined parents' reports. The relative impact of each parent's alcohol use thus is poorly understood. This study extends prior research by examining both mothers' and fathers' (based on mothers' perceptions) alcohol use at two distinct developmental periods.

Mothers' and fathers' alcohol use were found to be stable from the prenatal period to adolescence. These findings are notable because surprisingly few studies have examined the stability of both mothers' and fathers' alcohol use over time, especially over an extended period of time (Johnson et al. 2019; Kerr et al. 2012; Liu et al. 2015; Tran et al. 2018). Tran et al. (2018) found high levels of stability in mothers' alcohol use over a 14-year period for mothers who abstained from alcohol use or drank at low levels during the

prenatal period. Mothers' alcohol use was also highly stable when children were ages 9 to 11 in an intergenerational study of the sequelae of maternal and family functioning (Johnson et al. 2019). In another intergenerational study, fathers' alcohol use was found to be moderately stable from their own adolescence to adulthood (Kerr et al. 2012). Stability findings from the current study are consistent with prior work and highlight the influence of prenatal parental alcohol use on mothers' and fathers' drinking behaviors when their offspring were adolescents.

Mothers' and fathers' (based on mothers' perceptions) alcohol use during the prenatal period and adolescence were found to be unique predictors of adolescents' heavy drinking. These findings add a developmental perspective to knowledge of the relation between mothers' and fathers' alcohol use and adolescents' heavy drinking. Mothers' and fathers' prenatal alcohol use both seem to have enduring (compared to transient) effects given that they were related to adolescents' heavy drinking 16 years later. Gestational exposure to alcohol and genetic/biological factors are likely reasons for the enduring prenatal effects (Cornelius et al. 2016). The findings that mothers' and fathers' alcohol use during adolescence were also unique predictors of adolescents' heavy drinking are important. They show that

mothers' and fathers' alcohol use at two different development periods separated by 16 years both influence the heavy drinking of adolescents. The unique effects of parental alcohol use during adolescence could reflect the proximal effects of alcohol-related socialization within the family (Donovan 2018).

Another way that mothers' and fathers' prenatal alcohol use exerted their influence on adolescents' heavy drinking was by their impact on mothers' and fathers' alcohol use during adolescence. Specifically, mothers' and fathers' prenatal use were associated with adolescents' heavy drinking indirectly through both mothers' and fathers' alcohol use during adolescence. The stability of parents' alcohol use across their offspring's development thus is important for understanding the heavy drinking of adolescents and, again, may reflect an enduring home environment in which there are lenient alcohol-related parenting practices and parents model the misuse of alcohol. It is important to note that the effects of parental alcohol use were observed after controlling for gender, cumulative contextual risk, childhood externalizing behaviors, and associations with deviant peers, which are all established risk factors for heavy alcohol use during adolescence.

Neither mothers' nor fathers' alcohol use during the prenatal period or adolescence were directly related to young adults' alcohol use disorder. However, prenatal mothers' and fathers' alcohol use were related to young adults' alcohol use disorder indirectly through mothers' and fathers' alcohol use during adolescence and then through adolescents' heavy drinking. These indirect effects suggest that parental prenatal alcohol use contributes to a cascade of negative consequences across development (Eiden et al. 2016). The findings suggest that early and ongoing screening for parental alcohol use could help to identify individuals at risk for heavy drinking and alcohol-related problems during adolescence and young adulthood. Likewise, early and sustained multi-component, ecological interventions that target parental alcohol use are needed to reduce the risk of alcohol misuse by their offspring. Given that relatively few early intervention efforts for offspring alcohol misuse have addressed fathers' alcohol use, this focus seems particularly important.

Gender, childhood externalizing behaviors, and affiliating with deviant peers were predictors of young adults' alcohol use disorder. These results are consistent with a large literature that indicates men compared to women are at higher risk of experiencing alcohol related problems in young adulthood (Schulte et al. 2009) and that early child behavior problems and affiliating with deviant peers are salient predictors of young adults' alcohol-related problems (Kendler et al. 2013). One unique finding was a complex pathway from fathers' prenatal alcohol use to alcohol use disorder in young adulthood through associations with

deviant peers and then adolescents' heavy drinking. This finding provides support for a key part of the social control pathway toward offspring's alcohol misuse described by Donovan (2018). Here, fathers' but not mothers' prenatal alcohol use appears to have facilitated friendship formation with deviant peers among offspring, perhaps by contributing to rejection from mainstream peers earlier in development or by promoting attitudes favorable to deviance. Additional research is needed to better understand the mechanisms underlying this unique pathway and whether the pathway indeed only exists for fathers.

Findings from the current study should be interpreted in the context of several limitations. First, fathers' prenatal alcohol use was based on mothers' perceptions. For fathers' alcohol use when adolescents were age 16, 54% of mothers and fathers completed the survey together and most of the remaining data for this variable were based on mothers' reports. Mothers' perceptions of fathers' alcohol use were included because they provide an important perspective on fathers' use (Rodriguez and Neighbors 2015). Future research should include fathers' reports of their own alcohol use. Second, mothers' and fathers' alcohol use were assessed using different items during the prenatal period and adolescence. Third, the measures of parents' alcohol involvement focused on use. It is possible that different patterns of association would have been observed if the focus was on parents' alcohol-related problems. Fourth, several constructs were assessed using single item measures. Fifth, there was a notable amount of missing data for alcohol use variables for fathers. Sixth, the study did not account for several family-related factors that could influence the relation between parental alcohol use and the alcohol use of their offspring. For example, the quality of the parent-child relationship and children's sense of felt security in their families may be important factors that help explain the impact of parental alcohol use but were unmeasured in this birth cohort.

Conclusion

Relatively few studies on the association between parental alcohol use and the alcohol misuse of their offspring have investigated this relation from a developmental perspective. A focus on *when* during development parents' alcohol use is most strongly related to the alcohol misuse of their offspring (i.e., developmental timing) is important for understanding whether parental alcohol use has long-term (enduring) or relatively short-term (transient) effects. The current study has several notable strengths including a long-term longitudinal design that spanned four distinct developmental periods (28 years), information about mothers' and fathers' alcohol use, multi-informant and multi-method

data, and the statistical control of individual, family, and peer risk factors. The results showed that mothers' and fathers' alcohol use at two different development periods (prenatal and adolescence) separated by 16 years were both unique predictors of adolescents' heavy drinking. Moreover, prenatal alcohol use by mothers and fathers were related to young adults' alcohol use disorder indirectly (but not directly) through mothers' and fathers' alcohol use during adolescence and then through adolescents' heavy drinking. The results indicated that mothers' and fathers' prenatal alcohol use have enduring effects by contributing to a cascade of negative consequences across development.

Acknowledgements We would like to thank Gail Smith for her valuable contributions to the project.

Authors' Contributions GP worked with the research team to conceive the study, assisted with statistical analysis and interpretation of the results, and drafted the manuscript; IP worked with the research team to conceive the study, performed the statistical analysis and assisted with the interpretation of the results, and helped to draft the manuscript; WAM worked with the research team to conceive the study, facilitated the development of study measures; assisted with statistical analysis and interpretation of the results, and helped to draft the manuscript; MC worked with the research team to conceive the study, facilitated the development of study measures; assisted with statistical analysis and interpretation of the results, and helped to draft the manuscript; JS worked with the research team to conceive the study and facilitated the development of study measures; JM worked with the research team to conceive the study, participated in its design and coordination, and helped to draft the manuscript; MRJ worked with the research team to conceive the study and participated in its design and coordination. All authors read and approved the final manuscript.

Funding The analyses were supported by National Institute on Drug Abuse (NIDA), National Institutes of Health, Grant # R01 DA038450. Support for the Northern Finland Birth Cohort was provided by the European Commission (Grant DynaHEALTH—H2020—633595), EU QLG1-CT-2000-01643 (EUROBLCS) Grant no. E51560, NorFA Grant no. 731, 20056, 30167, Academy of Finland (#268336), and USA/NIH 2000 G DF682 Grant no. 50945. The content is solely the responsibility of the authors and does not necessarily represent the official views of the funding agencies. The funders played no role in the research design, analysis, or writing and submission process. With support from the grants, all authors contributed to drafting the manuscript. The authors disclose no conflict of interest.

Data Sharing and Declaration This manuscript's data will not be deposited.

Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

Ethical Approval The study was approved by the ethical committee of the Northern Ostrobothnia Hospital District and The study was performed in accordance with the ethical standards as laid down in the 1964 Declaration of Helsinki and its later amendments.

Informed Consent Informed consent was obtained from research participants.

Publisher's note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

References

- Alati, R., Clavarino, A., Najman, J. M., O'Callaghan, M., Bor, W., Al Mamun, A., & Williams, G. M. (2008). The developmental origin of adolescent alcohol use: findings from the Mater University Study of pregnancy and its outcomes. *Drug and Alcohol Dependence*, *98*(1–2), 136–143.
- Baer, J. S., Sampson, P. D., Barr, H. M., Connor, P. D., & Streissguth, A. P. (2003). A 21-year longitudinal analysis of the effects of prenatal alcohol exposure on young adult drinking. *Archives of General Psychiatry*, *60*(4), 377–385. <https://doi.org/10.1001/archpsyc.60.4.377>.
- Cambron, C., Kosterman, R., Catalano, R. F., Guttmanova, & Hawkins, J. D. (2018). Neighborhood, family, and peer factors associated with early adolescent smoking and alcohol use. *Journal of Youth and Adolescence*, *47*(2), 369–382. <https://doi.org/10.1007/s10964-017-0728-y>.
- Chassin, L., Sher, K. J., Hussong, A., & Curran, P. (2013). The developmental psychopathology of alcohol use and alcohol disorders: research achievements and future directions. *Development and Psychopathology*, *25*(4pt2), 1567–1584. <https://doi.org/10.1017/S0954579413000771>.
- Chung, T., Creswell, K. G., Bachrach, R., Clark, D. B., & Martin, C. S. (2018). Adolescent binge drinking: Developmental context and opportunities for prevention. *Alcohol Research: Current Reviews*, *39*(1), 5–15.
- Cornelius, M. D., De Genna, N., Goldschmidt, L., Larkby, C., & Day, N. (2016). Adverse environmental exposures during gestation and childhood: Predictors of adolescent drinking. *Substance Use & Misuse*, *51*(10), 1253–1263. <https://doi.org/10.3109/10826084.2016.1162812>.
- Cranford, J. A., Zucker, R. A., Jester, J. M., Puttler, L. I., & Fitzgerald, H. E. (2010). Parental alcohol involvement and adolescent alcohol expectancies predict alcohol involvement in male adolescents. *Psychology of Addictive Behavior*, *24*(3), 386–396. <https://doi.org/10.1037/a0019801>.
- Danielsson, A., Wennberg, P., Hibell, B., & Romelsjö, A. (2012). Alcohol use, heavy episodic drinking and subsequent problems among adolescents in 23 European countries: does the prevention paradox apply? *Addiction*, *107*(1), 71–80. <https://doi.org/10.1111/j.1360-0443.2011.03537.x>.
- Diggs, O. N., Neppl, T., Jeon, S., & Lohman, B. J. (2017). The association of harsh parenting, parent-child communication, and parental alcohol use with male alcohol use into emerging adulthood. *Journal of Adolescent Health*, *61*(6), 736–742. <https://doi.org/10.1016/j.jadohealth.2017.06.025>.
- Donovan, J.E. (2018). A framework for studying parental socialization of child and adolescent substance use. In H.E. Fitzgerald & L.I. Puttler (Eds.), *Alcohol use disorders: a developmental science approach to etiology* (pp. 143–180). New York, NY: Oxford University Press. <https://doi.org/10.1093/oso/9780190676001.001.0001>.
- Duncan, S. C., Duncan, T. E., & Strycker, L. A. (2006). Alcohol use from ages 9-16: a cohort-sequential latent growth model. *Drug and Alcohol Dependence*, *81*(1), 71–81. <https://doi.org/10.1016/j.drugalcdep.2005.06.001>.

- Easey, K. E., Dyer, M. L., Timpson, N. J., & Munafo, M. R. (2019). Prenatal alcohol exposure and offspring mental health: a systematic review. *Drug and Alcohol Dependence*, *197*, 344–353. <https://doi.org/10.1016/j.drugalcdep.2019.01.007>.
- Eiden, R. D., Lessard, J., Colder, C. R., Livingston, J., Casey, M., & Leonard, K. E. (2016). Developmental cascade model for adolescent substance use from infancy to late adolescence. *Developmental Psychology*, *52*(10), 1619–1633. <https://doi.org/10.1037/dev0000199>.
- Evans, G. W., Li, D., & Whipple, S. S. (2013). Cumulative risk and child development. *Psychological Bulletin*, *139*(6), 1342–1396. <https://doi.org/10.1037/a0031808>.
- Fergusson, D. M., Boden, J. M., & Horwood, L. J. (2013). Alcohol misuse and psychosocial outcomes in young adulthood: Results from a longitudinal birth cohort studied to age 30. *Drug and Alcohol Dependence*, *133*(2), 513–519. <https://doi.org/10.1016/j.drugalcdep.2013.07.015>.
- Finan, L. J., Simpson, E., Schulz, J., & Ohannessian, C. M. (2018). Parental problem drinking and emerging adult problem behavior: the moderating role of parental support. *Journal of Child and Family Studies*, *27*(4), 1175–1185. <https://doi.org/10.1007/s10826-017-0953-8>.
- Goldschmidt, L., Richardson, G. A., De Genna, N. M., Cornelius, M. D., & Day, N. L. (2019). Prenatal alcohol exposure and offspring alcohol use and misuse at 22 years of age: a prospective longitudinal study. *Neurotoxicology and Teratology*, *71*, 1–5. <https://doi.org/10.1016/j.ntt.2018.11.001>.
- Haltigan, J. D., Roisman, G. I., & Fraley, R. C. (2013). The predictive significance of early caregiving experiences for symptoms of psychopathology through midadolescence: enduring or transient effect. *Development and Psychopathology*, *25*(1), 209–221. <https://doi.org/10.1017/S0954579412000260>.
- Hawkins, J. D., Catalano, R. F., & Miller, J. Y. (1992). Risk and protective factors for alcohol and other drug problems in adolescence and early adulthood: implications for substance abuse prevention. *Psychological Bulletin*, *112*(1), 64–105. <https://doi.org/10.1037/0033-2909.112.1.64>.
- Hommel, J., & Warren, D. (2019). The relationship between parent drinking and adolescent drinking: Differences for mothers and fathers and boys and girls. *Substance Use & Misuse*, *54*(4), 661–669. <https://doi.org/10.1080/10826084.2018.1531429>.
- Hurtig, T., Ebeling, H., Taanila, A., Miettunen, J., Smalley, S. L., McGough, J. J., Loo, S. K., Järvelin, M.-R., & Moilanen, I. K. (2007). ADHD symptoms and subtypes: relationship between childhood and adolescent symptoms. *Journal of the American Academy of Child and Adolescent Psychiatry*, *46*(12), 1605–1613. <https://doi.org/10.1097/chi.0b013e318157517a>.
- Hussong, A. M., Huang, W., Serrano, D., Curran, P. J., & Chassin, L. (2012). Testing whether and when parent alcoholism uniquely affects various forms of adolescent substance use. *Journal of Abnormal Psychology*, *40*(8), 1265–1276. <https://doi.org/10.1007/s10802-012-9662-3>.
- Johnson, A. K., Fulco, C. J., & Augustyn, M. B. (2019). Intergenerational continuity in alcohol misuse: Maternal alcohol use disorder and the sequelae of maternal and family functioning. *Psychology of Addictive Behaviors*, *33*(5), 442–456. <https://doi.org/10.1037/adb0000476>.
- Kelly, A. B., O'Flaherty, M., Toumbourou, J. W., Homel, R., Paton, G. C., White, A., & Williams, J. (2012). The influence of families on early adolescent school connectedness: evidence that this association varies with adolescent involvement in peer drinking networks. *Journal of Abnormal Child Psychology*, *40*(3), 437–447. <https://doi.org/10.1007/s10802-011-9577-4>.
- Kendler, K. S., Gardner, C. O., Edwards, A., Hickman, M., Heron, J., Macleod, J., Lewis, G., & Dick, D. M. (2013). Dimensions of parental alcohol use/problems and offspring temperament, externalizing behaviors and alcohol use/problems. *Alcoholism: Clinical and Experimental Research*, *37*(12), 2118–2127. <https://doi.org/10.1111/acer.12196>.
- Kendler, K. S., Ji, J., Edwards, A. C., Ohlsson, H., Sundquist, J., & Sundquist, K. (2015). An extended Swedish national adoption study of alcohol use disorder. *JAMA Psychiatry*, *72*(3), 211–218. <https://doi.org/10.1001/jamapsychiatry.2014.2138>.
- Kerr, D. C. R., Capaldi, D. M., Pears, K. C., & Owen, L. D. (2012). Intergenerational influences on early alcohol use: Independence from the problem behavior pathway. *Development & Psychopathology*, *24*(3), 889–906. <https://doi.org/10.1017/S0954579412000430>.
- Latendresse, S. J., Rose, R. J., Viken, R. J., Pulkkinen, L., Kaprio, J., & Dick, D. M. (2008). Parenting mechanisms in links between parents' and adolescents' alcohol use behaviors. *Alcoholism: Clinical and Experimental Research*, *32*(2), 322–330. <https://doi.org/10.1111/j.1530-0277.2007.00583.x>.
- Liu, W., Mumford, E. A., & Petras, H. (2015). Maternal patterns of postpartum alcohol consumption by age: A longitudinal analysis of adult urban mothers. *Prevention Science*, *16*(3), 353–363. <https://doi.org/10.1007/s11121-014-0522-y>.
- MacKinnon, D. P., Lockwood, C. M., & Williams, J. (2004). Confidence limits for the indirect effect: Distribution of the product and resampling methods. *Multivariate Behavioral Research*, *39*(1), 99–128. https://doi.org/10.1207/s15327906mbr3901_4.
- Maes, H. H., Woodard, C. E., Murrelle, L., Meyer, J. M., Silberg, J. L., Hewitt, J. K., Rutter, M., Simonoff, E., Pickles, A., Carbonneau, R., Neale, M. C., & Eaves, L. J. (1999). Tobacco, alcohol and drug use in eight- to sixteen-year-old twins: the virginia twin study of adolescent behavioral development. *Journal of Studies on Alcohol*, *60*(3), 293–305. <https://doi.org/10.15288/jsa.1999.60.293>.
- Mahedy, L., MacArthur, G. J., Hammerton, G., Edwards, A. C., Kendler, K. S., Macleod, J., & Heron, J. (2018). The effect of parental drinking on alcohol use in young adults: the mediating role of parental monitoring and peer deviance. *Addiction*, *113*(11), 2041–2050. <https://doi.org/10.1111/add.14280>.
- Mason, W. A., January, S. A., Chmelka, M. B., Parra, G. R., Savolainen, J., Miettunen, J., Marjo-Riitta Järvelin, M., Taanila, A., & Moilanen, I. (2016). Cumulative contextual risk at birth in relation to adolescent substance misuse, conduct problems, and risky sex: general and specific predictive associations in a Finnish Birth Cohort. *Addictive Behaviors*, *58*, 161–166. <https://doi.org/10.1016/j.addbeh.2016.02.031>.
- McBride, N., & Johnson, S. (2016). Fathers' role in alcohol-exposed pregnancies: systematic review of human studies. *American Journal of Preventive Medicine*, *51*(2), 240–248. <https://doi.org/10.1016/j.amepre.2016.02.009>.
- Miettunen, J., Murray, G. K., Jones, P. B., Mäki, P., Ebeling, H., Taanila, A., Joukamaa, M., Savolainen, J., Törmänen, S., Järvelin, M.-R., Veijola, J., & Moilanen, I. (2014). Longitudinal associations between childhood and adulthood externalizing and internalizing psychopathology and adolescent substance use. *Psychological Medicine*, *44*(8), 1727–1738. <https://doi.org/10.1017/S0033291713002328>.
- Moffitt, T. E. (1993). Adolescence-limited and life-course-persistent antisocial behavior: a developmental taxonomy. *Psychological Review*, *100*(4), 674–701. <https://doi.org/10.1037/0033-295X.100.4.674>.
- Muthén, L. K., & Muthén, B. O. (1998–2017). *Mplus user's guide*. Eighth Edition Los Angeles, CA: Muthén & Muthén .
- Parra, G. R., Smith, G., Mason, W. A., Savolainen, J., Chmelka, M. B., Miettunen, J., & Järvelin, M. (2017). Tests of linear and nonlinear relations between cumulative contextual risk at birth and psychosocial problems during adolescence. *Journal of Adolescence*, *60*, 64–73. <https://doi.org/10.1016/j.adolescence.2017.07.010>.

- Poelen, E. A., Engels, R. C., Scholte, R. H., Boomsma, D. I., & Willmsen, G. (2009). Predictors of problem drinking in adolescence and young adulthood. *European Child and Adolescent Psychiatry*, *18*(6), 345–352. <https://doi.org/10.1007/s00787-009-0736-x>.
- Randolph, K. A., Cheatham, L. P., Weiss, U. K., & Williams, J. (2018). Exposure to parent and peer alcohol use and the risk of drinking onset and escalation among adolescents. *Child and Adolescent Social Work Journal*, *35*(2), 97–106. <https://doi.org/10.1007/s10560-017-0516-2>.
- Rodriguez, L. M., & Neighbors, C. (2015). An interdependent look at perceptions of spousal drinking problems and marital outcomes. *Alcohol*, *49*(6), 597–605. <https://doi.org/10.1016/j.alcohol.2015.05.002>.
- Rossow, I., Felix, L., Keating, P., & McCambridge, J. (2016). Parental drinking and adverse outcomes in children: A scoping review of cohort studies. *Drug and Alcohol Review*, *35*(4), 397–405. <https://doi.org/10.1111/dar.12319>.
- Rossow, I., Keating, P., Felix, L., & McCambridge, J. (2016). Does parental drinking influence children's drinking: A systematic review of prospective cohort studies. *Addiction*, *111*(2), 204–217. <https://doi.org/10.1111/add.13097>.
- Rutter, M. (1967). A children's behaviour questionnaire for completion by teachers: preliminary findings. *Journal of Child Psychology and Psychiatry*, *8*(1), 1–11. <https://doi.org/10.1111/j.1469-7610.1967.tb02175.x>.
- Savolainen, J., Eisman, A., Mason, W. A., Schwartz, J. A., Miettunen, J., & Marjo-Riitta Järvelin, M. (2018). Socioeconomic disadvantage and psychological deficits: pathways from early cumulative risk to late-adolescent criminal conviction. *Journal of Adolescence*, *65*, 16–24. <https://doi.org/10.1016/j.adolescence.2018.02.010>.
- Schulte, M. T., Ramo, D., & Brown, S. A. (2009). Gender differences in factors influencing alcohol use and drinking progression among adolescents. *Clinical Psychology Review*, *29*(6), 535–547. <https://doi.org/10.1016/j.cpr.2009.06.003>.
- Tran, N. T., Clavarino, A., Williams, G. M., & Najman, J. M. (2018). Gender difference in offspring's alcohol use disorder by 21 years: a longitudinal study of maternal influences. *Substance Use & Misuse*, *53*(5), 705–715. <https://doi.org/10.1080/10826084.2017.1363233>.
- Verhulst, B., Neale, M. C., & Kendler, K. S. (2015). The heritability of alcohol use disorders: A meta-analysis of twin and adoption studies. *Psychological Medicine*, *45*(5), 1061–1072. <https://doi.org/10.1017/S0033291714002165>.
- Waaktaar, T., Kan, K. J., & Torgersen, S. (2018). The genetic and environmental architecture of substance use development from early adolescence into young adulthood: a longitudinal twin study of comorbidity of alcohol, tobacco and illicit drug use. *Addiction*, *113*(4), 740–748. <https://doi.org/10.1111/add.14076>.
- Zegers-Hochschild, F., Adamson, G.D., de Mouzon, J., Ishihara, O., Mansour, R., Nygren, K., Sullivan, E., Vanderpoel, S., International Committee for Monitoring Assisted Reproductive Technology, & World Health Organization. (2009). The international committee for monitoring assisted reproductive technology (ICMART) and the World Health Organization (WHO) revised glossary on ART terminology, 2009. *Fertility and Sterility*, *92*(5), 1520–1524. <https://doi.org/10.1016/j.fertnstert.2009.09.009>.

Gilbert R. Parra is an Associate Professor at the University of Nebraska-Lincoln. His major research interests include family factors and processes and the mental health of youth.

Irina Patwardhan is a Research Scientist at the Boys Town Translational Research Center. Her major research interests include development of self-regulation in childhood and adolescence.

W. Alex Mason was Senior Director of the Boys Town Child and Family Translational Research Center (now Professor of Preventive Medicine, University of Tennessee Health Science Center). His major research interests include the developmental etiology and prevention of adolescent and young adult substance misuse and cooccurring problems.

Mary B. Chmelka is a Director of Data Support at the Boys Town Translational Research Center. Her major research interests include adolescent substance misuse and comorbid problems.

Jukka Savolainen is professor of sociology and criminology at Wayne State University. His research is focused on the etiology of crime, violence, and victimization. Much of this work is based on data from Finland, Norway, and other European countries.

Jouko Miettunen is a Professor of Clinical Epidemiology, University of Oulu, Oulu, Finland. He is working mainly on psychiatric and psychological topics in the Northern Finland Birth Cohort studies. He is currently working also as a vice-dean of the University of Oulu Graduate School, Oulu, Finland. He has experience from several national and international projects relating to life course epidemiology. His background is in statistics and epidemiology. Marjo-Riitta Järvelin is Professor and Chair in Life Course Epidemiology at Imperial College London. Her major research interests include the genetic and early life environmental origins of multi-factorial diseases and disorders.