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

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**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* = 6,963; 51% of offspring were girls) were used. The NFBC1986 is a population-based study of individuals born during a one-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, Longitudinal

## Introduction

Heavy alcohol use during adolescence and young adulthood is associated with a wide range of negative psychosocial outcomes (Danielsson, Wennberg, Hibell, & Romelsjö, 2012; Fergusson, Boden, & Horwood, 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, Keating, Felix & McCambridge, 2016). It is less clear whether this relation exists at different developmental periods (Hussong, Huang, Serrano, Curran, & Chassin, 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 specify a timeframe for parents' drinking (e.g., Diggs, Neppl, Jeon, & Lohman, 2017; Finan, Simpson, Schulz, & Ohannessian, 2018; Randolph, Cheatham, Weiss, & Williams, 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, Felix, Keating, & McCambridge, 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, Roisman, & Fraley, 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, Fulco, & Augustyn, 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, Zucker, Jester, Puttler, & Fitzgerald, 2010; Duncan, Duncan, & Strycker, 2006; Latendresse, Rose, Viken, Pulkkinen, Kaprio, & Dick, 2008) and later use (Latendress 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, Dyer, Timpson, & Munafo, 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, Sampson, Barr, Connor, Streissguth, 2003; Cornelius, De Genna, Goldschmidt, Larkby, & Day, 2016; Goldschmidt, Richardson, De Genna, Cornelius, & Day, 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, Ji, Edwards, Ohlsson, Sundquist, & Sundquist, 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, Neale, & Kendler, 2015). Notably, a recent review of 11 studies suggested that paternal alcohol consumption during pregnancy is related to maternal alcohol consumption during pregnancy (McBride & Johnson, 2016).

Heavy alcohol use often begins during adolescence (Chung, Creswell, Bachrach, Clark, & Martin, 2018). Research has shown that parents' alcohol use during adolescence is associated with adolescents' alcohol misuse (Homel & Warren, 2019). Both genetic and psychosocial influences appear to play a role in this association. Adolescents' alcohol use is, to some degree, heritable (Waaktaar, Kan, & Torgersen, 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 convene 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, Catalano, & Miller, 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, Engels, Scholte, Boomsma, & Willemsen, 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, Capaldi, Pears, & Owen 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, Sher, Hussong, & Curran, 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, Ramo, & Brown, 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, Li, & Whipple, 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). Moffit'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, O'Flaherty, Toumbourou, Homel, Paton, White, & Williams, 2012). Deviant peer affiliation is a robust predictor of alcohol misuse during adolescence and young adulthood (Cambron, Kosterman, Catalano, Guttmannova, & Hawkins, 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 Figure 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 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 9,432 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 12$^{th}$ gestational week), to be returned by their 24$^{th}$ 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 6,963 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 16$^{th}$ week of pregnancy", and "Amount of alcohol drinks after 16$^{th}$ 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 ($\alpha = .77$). Research has shown that wives' perceptions of their husbands' alcohol use is related to husbands' reported alcohol use (Rodriguez & 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 ($\alpha = .85$) 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 socio-economic 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 2,500 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 (Muthen and Muthen 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 Figure 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 5,000 bootstrap samples were computed (MacKinnon, Lockwood, & Williams, 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 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" (Savolainena, Eisman, Mason, Schwartz, Miettunen, & Marjo-Riitta Järvelin, 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 .11 to .35, *p*s < .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: $\chi^2$ ($n$ = 6963) = 93.11, $df$ = 40, $p$ < .001, RMSEA = .01, SRMR = .01, CFI = 0.996, TLI = 0.99. Factor loadings for the prenatal fathers' alcohol use latent variable were .86 and .90 (standardized coefficients) for frequency and quantity variables, respectively. Factor loadings for the externalizing childhood behaviors at age 8 latent variable were .85, .59, .83, and .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: $\chi^2$ ($n$ = 6471) = 1257.07, $df$ = 46, $p$ < .001, RMSEA = .06, SRMR = .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: $\chi^2$ ($n$ = 6471) = 910.49, $df$ = 44, $p$ < .001, RMSEA = .06, SRMR = .04, CFI = 0.93, TLI = 0.86.

Parameter estimates for the multivariate model are presented in Table 2. To highlight key findings, Figure 2 presents only the statically significant paths. The findings revealed that mothers' (β = .79) and fathers' (β = .44) prenatal alcohol use were significantly associated with mothers' alcohol use when adolescents were age 16. Similarly, mothers' (β = .61) and fathers' (β

= .59) prenatal alcohol use alcohol use were significantly associated with fathers' alcohol use when adolescents were age 16.

The results further indicated that mothers' (β = .24) and fathers' (β = .23) prenatal alcohol use were significantly associated with adolescents' heavy drinking. Both mothers' (β = .08) and fathers' (β = .06) alcohol use when adolescents were age 16 were significantly associated with adolescents' heavy drinking. There were no significant associations between mothers' (β =.05) or fathers' (β =.09) prenatal alcohol use and young adults' alcohol use disorder. Neither mothers' (β = .05) nor fathers' (β = -.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 (β = .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 (β = .09), associates with deviant peers (β = .03), and adolescents' heavy drinking (β = .06) as well as a significant negative association with

mothers' alcohol use when adolescents were age 16 (β = -.04). Coefficients for gender as a covariate showed that being male increased risk for childhood externalizing behaviors (β = .55) and young adults' alcohol use disorder (β = .14) as well as decreased risk for adolescents' heavy drinking (β = -.13). Childhood externalizing behavior was related to associates with deviant peers (β = .06), adolescents' heavy drinking (β = 0.09), and young adults' alcohol use disorder (β = .13). Associates with deviant peers was associated with adolescents' heavy drinking (β = .25) and young adults' alcohol use disorder (β = .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 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, Mumford, & Petras, 2015; Tran, Clavarino, Williams & Najman, 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, Lessard, Colder, Livingston, Casey, Leonard, 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 & 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.

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**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 | .26* | | | | | | | | | | | | | |
| 3. Quantity | .27* | .80* | | | | | | | | | | | | |
| 4. Cumulative Contextual Risk | .04* | .04* | .08* | | | | | | | | | | | |
| 5. Gender[a] | -.01 | -.01 | -.02 | -.03* | | | | | | | | | | |
| **Age 8** | | | | | | | | | | | | | | |
| Childhood Externalizing Behaviors | | | | | | | | | | | | | | |
| 6. Breaks Things | .03* | .02 | .02 | .05* | .16* | | | | | | | | | |
| 7. Fights or Quarrels | .01 | .03* | .03* | .07* | .21* | .47* | | | | | | | | |
| 8. Is Disobedient | .01 | .04* | .03 | .07* | .22* | .50* | .65* | | | | | | | |
| 9. Teases Other Children | .02 | .03* | .01 | .07* | .23* | .48* | .75* | .67* | | | | | | |
| **Age 16** | | | | | | | | | | | | | | |
| 10. Mothers' Alcohol Use | .23* | .42* | .44* | -.02 | -.02 | .00 | .02 | .01 | .01 | | | | | |
| 11. Fathers' Alcohol Use | .18* | .55* | .55* | .00 | -.02 | .01 | .02 | .02 | .02 | .69* | | | | |
| 12. Associates with Deviant Peers | .03* | .08* | .09* | .03* | .01 | .04* | .05* | .04* | .04* | .07* | .08* | | | |
| 13. Adolescents' Heavy Drinking | .11* | .28* | .35* | .08* | -.05* | .05* | .10* | .08* | .08* | .26* | .29* | .28* | | |
| **Age 28** | | | | | | | | | | | | | | |
| 14. Young Adults' Alcohol Use Disorder[a] | .02 | .05* | .04* | .03* | .04* | .06* | .06* | .05* | .06* | .03 | .02 | .09* | .09* | |

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 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 |
| *n* | 6471 | 5932 | 4401 | 6963 | 6963 | 6441 | 6440 | 6431 | 6423 | 5844 | 5490 | 6476 | 6152 | 6963 |

Note. [a]Point Biserial Correlations unless otherwise noted.
Associations between two dichotomous variables are *Phi* values.
*$p < .05$.

**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 | .07 / .03 | .79 / 1.21* | .61 / 1.06* | .09 / .04* | .24 / .51* | .05 / .05 |
| Fathers' Alcohol Use | .03 / .01 | .44 / .65* | .59 / 1.00* | .10 / .04* | .23 / .48* | .09 / .08 |
| Cumulative Contextual Risk | .09 / .06* | -.04 / -.10* | -.02 / -.06 | .03 / .02* | .06 / .20* | .04 / .06 |
| Gender | .55 / .21* | -.02 / -.03 | -.05 / -.08 | -.01 / .00 | -.13 / -.29* | .14 / .14* |
| **Age 8** | | | | | | |
| Childhood Externalizing Behaviors | | .01 / .03 | .01 / .04 | .06 / .07* | .09 / .50* | .13 / .34* |
| **Age 16** | | | | | | |
| Mothers' Alcohol Use | | | | | .08 / .12* | .05 / .03 |
| Fathers' Alcohol Use | | | | | .06 / .08* | -.09 / -.05 |
| Associates with Deviant Peers | | | | | .25 / 1.24* | .10 / .22* |
| Adolescents' Heavy Drinking | | | | | | .18 / .08* |

Note. $N = 6471$.
Values are standardized / unstandardized coefficients.
STDY standardization parameters reported for dichotomous covariates.
*$p < .05$.

**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 | .141* | .072 | .212 |
| 2. Mothers' Alcohol Use (16) to Adolescents' Heavy Drinking to Young Adults' Alcohol Use Disorder | .012* | .005 | .021 |
| 3. Fathers' Alcohol Use (16) to Adolescents' Heavy Drinking | .082* | .018 | .147 |
| 4. Fathers' Alcohol Use (16) to Adolescents' Heavy Drinking to Young Adults' Alcohol Use Disorder | .007* | .001 | .014 |
| 5. Associates with Deviant Peers to Adolescents' Heavy Drinking to Young Adults' Alcohol Use Disorder | .004 | .000 | .009 |
| **Prenatal Fathers' Alcohol Use to** | | | |
| 6. Mothers' Alcohol Use (16) to Adolescents' Heavy Drinking | .076* | .039 | .112 |
| 7. Mothers' Alcohol Use (16) to Adolescents' Heavy Drinking to Young Adults' Alcohol Use Disorder | .006* | .003 | .011 |
| 8. Fathers' Alcohol Use (16) to Adolescents' Heavy Drinking | .076* | .017 | .135 |
| 9. Fathers' Alcohol Use (16) to Adolescents' Heavy Drinking to Young Adults' Alcohol Use Disorder | .006* | .001 | .013 |
| 10. Associates with Deviant Peers to Adolescents' Heavy Drinking to Young Adults' Alcohol Use Disorder | .004* | .002 | .007 |
| **Mothers' Alcohol Use (16) to** | | | |
| 11. Adolescents' Heavy Drinking to Young Adults' Alcohol Use Disorder | .010* | .004 | .017 |
| **Fathers' Alcohol Use (16) to** | | | |
| 12. Adolescents' Heavy Drinking to Young Adults' Alcohol Use Disorder | .006* | .001 | .013 |

Note. $n$ = 6471. CI = Confidence Intervals.

*Indicates estimate is significantly different from 0 based on bootstrapped 95% CI not including 0.

**Figure 1**

*Conceptual Model*

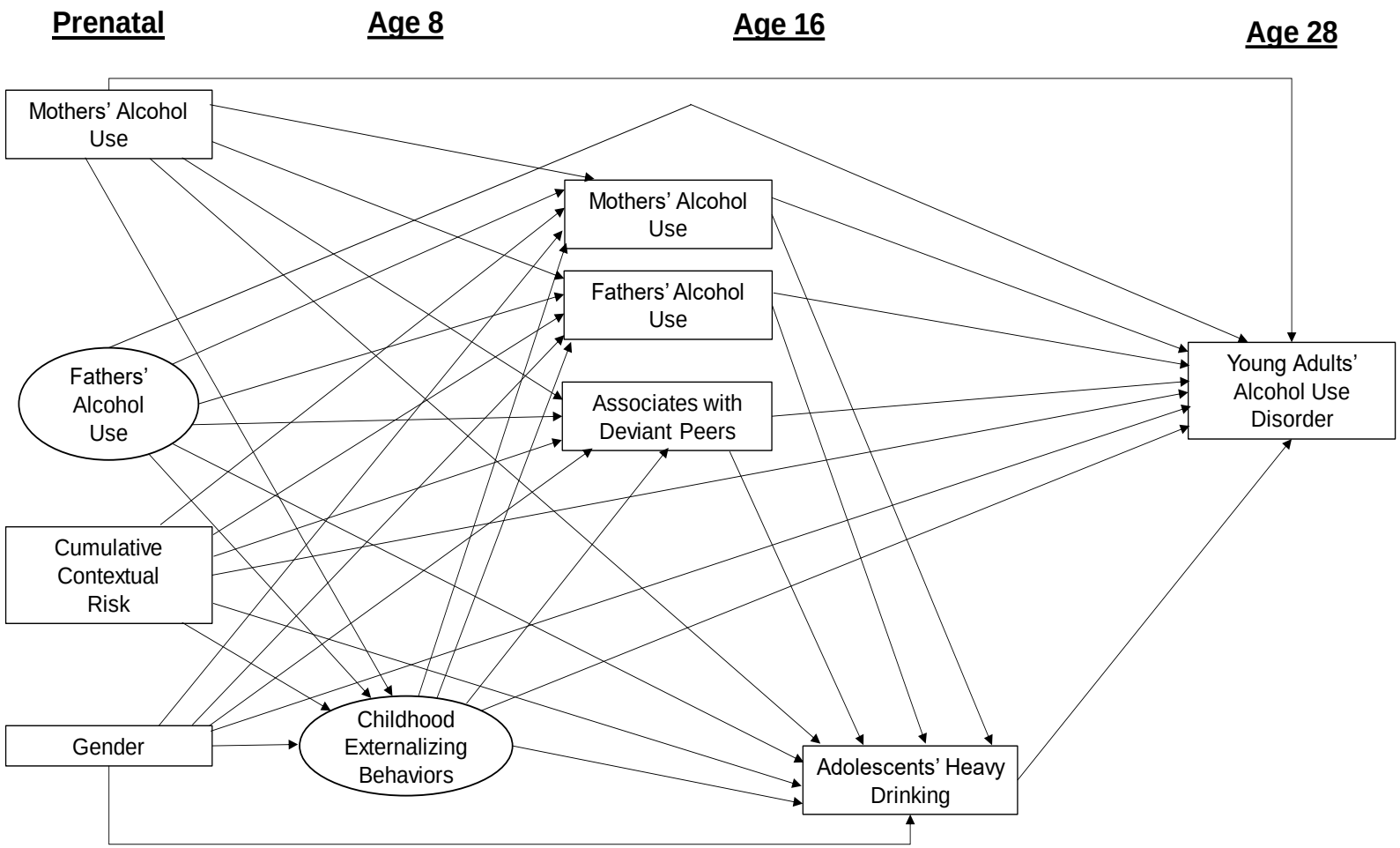
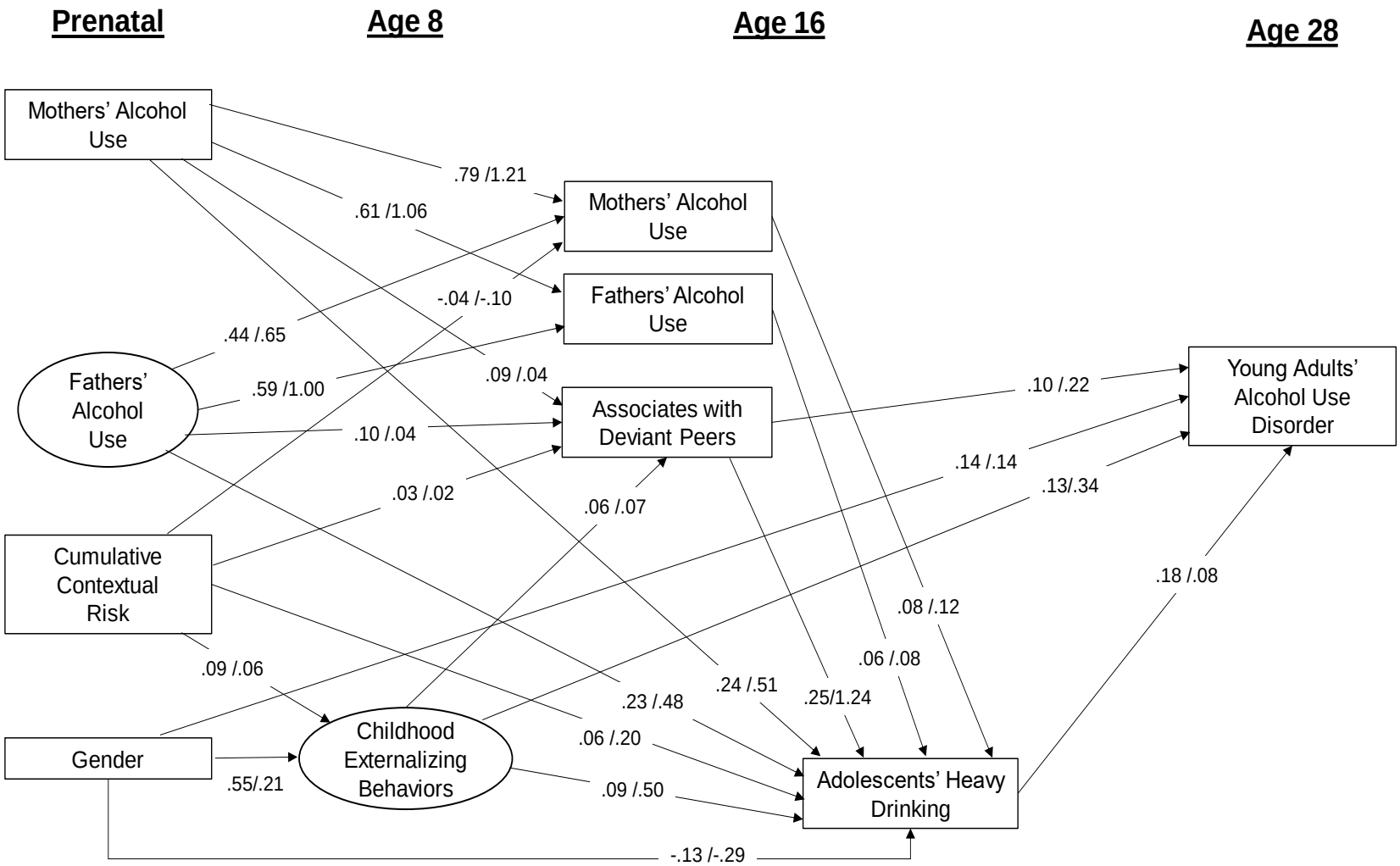


**Figure 2**
*Multivariate Structural Equation Model*




Note. $n$ = 6471. Parameters are standardized / unstandardized estimates.
STDY standardization parameters reported for dichotomous covariates.
All parameters shown are statically significant ($p < .05$).