



Time-specific and cumulative effects of exposure to parental externalizing behavior on risk for young adult alcohol use disorder



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HIGHLIGHTS

- Parental externalizing behavior predicts offspring AUD in young adulthood.
- Timing of parental EB exposure does not differentially impact AUD risk.
- Sustained parental EB exposure results in higher offspring AUD rates.

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ABSTRACT

Background: Previous studies indicate that parental externalizing behavior (EB) is a robust risk factor for alcohol use disorder (AUD) in their children, and that this is due to both inherited genetic liability and environmental exposure. However, it remains unclear whether the effects of exposure to parental EB vary as a function of timing and/or chronicity.

Methods: We identified biological parents with an alcohol use disorder, drug abuse, or criminal behavior, during different periods of their child's upbringing, using Swedish national registries. Logistic regression was used to determine whether the effect of parental EB exposure during different developmental periods differentially impacted children's risk for young adult AUD (ages 19–24). In addition, we tested how multiply affected parents and/or sustained exposure to affected parents impacted risk.

Results: While parental EB increased risk for young adult AUD, timing of exposure did not differentially impact risk. Having a second affected parent increased the risk of AUD additionally, and sustained exposure to parental EB across multiple periods resulted in a higher risk of young adult AUD than exposure in only one period.

Conclusions: In this well-powered population study, there was no evidence of "sensitive periods" of exposure to national registry-ascertained parental EB with respect to impact on young adult AUD, but sustained exposure was more pathogenic than limited exposure. These findings suggest developmental timing does not meaningfully vary the impact, but rather there is a pervasive risk for development of young adult AUD for children and adolescents exposed to parental EB.

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1. Introduction

Parental externalizing behaviors (EB), including substance use disorders and antisocial or criminal behavior, are strong predictors of similar

outcomes in children (Chassin, Pitts, DeLucia, & Todd, 1999; Connell & Goodman, 2002; Hicks, Foster, Iacono, & McGue, 2013; Kendler et al., 2013; Kendler, Ohlsson, Sundquist, & Sundquist, 2016a; Marmorstein, Iacono, & McGue, 2009; Merikangas, Dierker, & Szatmari, 1998; Sorensen et al., 2011). This risk is conferred by both genetic liability and environmental exposures, as evidenced by genetically informative studies of adoptees and twins (Bohman, Sigvardsson, & Cloninger, 1981; Cloninger, Bohman, & Sigvardsson, 1981; Kendler et al., 2015),

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and by studies exploring mediation across factors (Sher, Gershuny, Peterson, & Raskin, 1997). Alcohol use disorder (AUD), which is moderately heritable (Verhulst, Neale, & Kendler, 2015), falls within the spectrum of outcomes associated with exposure to parental EB; however little is known about factors that may moderate these effects, such as timing and duration of exposure. In the current study of young adult AUD, we draw on tenets from developmental psychopathology to investigate patterns and effects of exposure to parental EB during upbringing, with an emphasis on differences in risk conferred by exposure during sensitive developmental periods and by exposures that accumulate over time. A better understanding of these temporal effects can inform targeted prevention of AUD.

Sensitive developmental periods refer to life phases such as middle childhood and adolescence during which exposures to risk factors and stressors may be particularly salient. For example, sensitive periods have been examined with respect to trauma exposure and later depression (McCutcheon et al., 2009) and post-traumatic stress disorder (McCutcheon et al., 2010). In the case of parental EB and young adult AUD, heightened risk is expected given instability likely to be experienced by children and adolescents exposed to parental EB, the role of parental modeling of EB (White, Johnson, & Buyske, 2000), and the stress-sensitive and dynamic neurodevelopmental processes at play during these early years (Andersen et al., 2008; Pechtel, Lyons-Ruth, Anderson, & Teicher, 2014). This question has been previously addressed in the context of child physical abuse. A recent study of US young adults found physical abuse starting in adolescence increased risk for pathological drinking behaviors including AUD (Shin, Chung, & Rosenberg, 2016). This is noteworthy, as early childhood represents a developmental period especially sensitive to the impact of child abuse on subsequent functioning (Cicchetti & Toth, 1995). However, some studies have found that abuse exposure during adolescence leads to later alcohol misuse (Smith et al., 2005; Thornberry et al., 2001). With respect to parental EB exposure, previous research has demonstrated that parental AUD has both proximal and distal effects on children's externalizing behavior (Hussong, Huang, Curran, Chassin, & Zucker, 2010), of which AUD is a potential manifestation; it remains unclear whether those effects differ as a function of timing of exposure.

Another important aspect of parental EB exposure concerns its duration or chronicity. Accumulated risk refers to the build-up over time of repeated/sustained exposure to AUD risk factors. Compounding of risks engendered in early developmental stages impacts later health and development (Henrich, 2006). This concept is similar to the *cumulative risk hypothesis* (Sameroff, Seifer, Baldwin, & Baldwin, 1993), although here we are examining chronic exposure to a constellation of related risk factors—grouped as exposure to parental EB—rather than exposure to diverse risk factors. In the study of sequelae of child abuse, chronic exposure was associated with significantly increased risk of heavy drinking and AUD, and this risk was greater than that seen for time-limited child abuse (Shin et al., 2016). This line of questioning also can be applied to the impact of chronicity of parental EB exposure on AUD risk. Children of parents with EB likely experience more sustained exposure to a variety of environmental stressors than do their peers (Hussong et al., 2008). As noted above, evidence that adoptive parental EB confers risk for AUD (Kendler, Neale, Heath, Kessler, & Eaves, 1994; Kendler et al., 2012; Kendler et al., 2015) confirms that environmental exposure is a risk factor. Because parental EB can remit, children exposed to parental EB during only some developmental periods may be at a lower risk for later AUD than their counterparts with sustained exposure.

Exposure to multiple affected parents can also be considered a form of cumulative risk. Prior studies have examined the effects of having one versus two parents with psychopathology with respect to offspring outcomes. While some studies have reported (non-additive) interactions between maternal and paternal psychopathology (Brennan, Hammen, Katz, & Le Brocque, 2002), others have not (Cimino, Cerniglia, & Paciello, 2015; Johnson, Cohen, Kasen,

Smailes, & Brook, 2001). Thus, clarification is needed regarding the impact of multiple affected parents.

In the current study, we examine whether parental EB impacts the likelihood of young adult AUD differentially as a function of timing and chronicity of exposure. We also test the effects of cumulative exposure from two perspectives. First, how does having one versus two affected parents impact risk of young adult AUD, and does the gender of the affected parent differentially impact risk? Second, is parental EB exposure across multiple developmental periods more pathogenic than time-limited exposure? We explore these questions using the population of Sweden, for which data are available on substance abuse and criminal behavior in parents, as well as on AUD in their children. We anticipated that having a parent exhibit EB during adolescence would have a stronger influence on AUD risk than a similar exposure during early childhood. We further hypothesized that cumulative exposure to parental EB would act as a more potent risk factor than limited exposure.

2. Materials and methods

2.1. Sample

These analyses are based on the Swedish population. The following national registries were used: 1) the Swedish Hospital Discharge Register, which included hospitalizations for people in Sweden from 1964 through 2010, classified by the main discharge diagnosis and eight secondary diagnoses; 2) the Multi-Generation Register linking children born after 1932 to their parents; 3) the National Census Registry, which provided information on education at 5-year increments (1960–1990); 4) the Total Population Registry, which included annual data on education from 1990 to 2009; and 5) the Swedish Crime Register, containing all convictions in lower court from 1973 to 2011.

Linking was based on individual Swedish 10-digit personal identification numbers, which are assigned at birth or immigration for all Swedish residents and used in all official records for his or her lifetime. This number was replaced by a serial number to guarantee confidentiality for all individuals. The study was approved by the ethics committee in Lund, Sweden; subject consent was waived.

As described above and previously (Kendler et al., 2012; Kendler et al., 2015; Kendler et al., 2016a), information from various national registries is available across different time frames, which impacts the cohorts for which specific combinations of data are available. The current analyses incorporate the risk conferred by biological parental behavior; accordingly, we were limited to cohorts for whom biological parental data was available. We therefore examined outcomes for individuals born in Sweden between 1970 and 1984, for whom data on young adult (age 19–24) AUD were available. These individuals were divided into three cohorts to account for potential differences in the social environment over time. We included individuals who did not emigrate or die before age 24 and who had two registered biological parents in the multi-generation register. No further limitations were made for the parents.

2.2. Measures

2.2.1. Outcome variable

We defined AUD from the inpatient register, which covers the young adult period for all three cohorts, using ICD codes for alcohol abuse and related disorders as described elsewhere (Kendler et al., 2015). Briefly, these include codes for alcohol related disorders such as abuse and dependence (F10), alcoholic liver disease (K70), alcohol induced pancreatitis (K85.2 and K86.0), and toxic effects of alcohol (T51), among other diagnoses. The current analyses focus on AUD during young adulthood (between ages 19–24).

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