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The relation of parent alcohol disorder to young adult drinking outcomes mediated by parenting: Effects of developmentally limited versus persistent parent alcohol disorder

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ABSTRACT

Background: Parent alcohol use disorder (AUD) is a well-established risk factor for the development of offspring AUD and is associated with poor parenting. However, few studies have examined heterogeneity in trajectories of parental AUD and its influence on adolescent offspring drinking, and no studies to date have considered the differential risk to offspring conferred by parental AUDs that are limited to early adulthood. Specifically, AUDs limited to the period of emerging adulthood may confer less risk to a child's environment as recovery following emerging adulthood coincides with the typical ages of entry into the parenting role. The present study tested whether parental AUDs developmentally limited to emerging adulthood (DLAUD) transmit less risk for alcohol problems and alcohol consumption in offspring compared to offspring of parents with AUDs spanning across multiple developmental periods (persistent AUD), as mediated by positive parenting strategies.

Method: Pathways were examined using longitudinal mediation models (N = 361) comparing offspring with parental DLAUD, persistent AUD, and no AUD.

Results: Parents with DLAUD do not transmit the same risk for alcohol problems to offspring as parents with persistent AUD (B = 0.173, SE = 0.067, $p < .05$); more offspring alcohol problems were associated with persistent AUD than with DLAUD. Positive parenting mediated the transmission of risk from parental AUD to offspring alcohol problems (B = 0.040, SE = 0.019, $p < .05$) and consumption (B = 0.019, SE = 0.011, $p < .05$) only when comparing persistent AUD vs. no parental AUD.

Conclusion: Findings suggest that the developmental period in which parents' recovery occurs is a useful way to categorize “recovered” AUDs versus current AUDs.

1. Introduction

Given the societal costs associated with problematic drinking and alcohol use disorder (Mokdad et al., 2004), the study of alcohol misuse across the lifetime has considerable public health importance. A well-established risk factor for alcohol use and misuse is having a parent with alcohol use disorder (AUD; Sher et al., 1991; Chassin et al., 1992; McGue, 1994). However, despite the wealth of research which shows that parental AUD is linked to the development of alcohol problems and AUD in offspring, prior research has often relied on an oversimplified definition of parental AUD. For example, parent AUD has been defined as having at least one parent with AUD (e.g., Nirenberg et al., 1990; Rogosch et al., 1990), or having at least one parent in treatment for AUD (Ohannessian et al., 1995). Yet this definition overlooks potentially important types of heterogeneity within parental AUD and possible differential associations with adolescent offspring alcohol use.

Although few studies have considered heterogeneity in the definition of parental AUD, some existing studies indicate differential pathways of risk. Types of heterogeneity such as a number of alcoholic parents, recovered vs. current AUD, and comorbid vs. single AUD diagnosis have been examined in the literature. Results suggest risk for internalizing and externalizing symptoms is greater for participants with two parents with AUD as compared to participants with only one or no parents with AUD (Hussong et al., 2008a,b; Edwards et al., 2006). Chassin et al. (1991) found that children of parents with a recent AUD had higher levels of externalizing symptoms than did children of parents with recovered AUD. However, alcohol use of children of parents with recovered AUD was still elevated as compared to children of parents with no AUD (Chassin et al., 1991). Children of parents with AUD and depression or AUD and antisocial symptoms displayed greater risk for internalizing symptomology and externalizing behaviors as compared to children of parents with AUD and no antisocial symptoms

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or depression (Hussong et al., 2008a,b). Additionally, parental AUDs can also be meaningfully subtyped based on drinking patterns and severity of dependence symptoms (Jellinek, 1960; Sher et al., 1991).

These studies demonstrate that heterogeneity in parental AUD diagnosis is associated with differential risk for adolescent offspring alcohol use and psychopathology. The purpose of the current study is to consider a novel classification of heterogeneity in parents' AUD as either "developmentally limited" vs. "persistent," and examine associations with offspring alcohol use. In particular, developmentally limited refers to parental AUDs that are limited to one period of parents' development compared to a disorder that persists over time and spans multiple developmental stages. In the current study, we consider developmentally limited AUD to be during the period of emerging adulthood, defined as between the ages of 18–25 (Arnett, 2007). Therefore, parents with developmentally limited AUD will not meet criteria for a diagnosis after the period of emerging adulthood (i.e., age 26). Parents with persistent AUD will continue to meet criteria for a diagnosis of AUD beyond this age. Emerging adulthood developmentally limited parental AUD is a special case of a "recovered" parental AUD in that recovery has occurred during a particular developmental stage, in this case prior to adulthood. Similarly, persistent AUD will often (but not always) be synonymous with "current" parental AUD.

Zucker and Noll (1987) originally described "developmentally limited" AUD (DLAUD) as one of four different trajectories of AUD. DLAUD was described as time-limited, related to heavy peer drinking in late adolescence and emerging adulthood, with recovery associated with entry into adult familial and career roles (Zucker and Noll, 1987; Maggs and Schulenberg, 2004). Thus, DLAUD, as defined by Zucker and Noll (1987), is specific to emerging adulthood when adult role occupancy typically occurs (Bachman et al., 1997). As alcohol consumption and alcohol problems typically reach their peak and then begin to decline during emerging adulthood (Johnston et al., 2004; Sher et al., 1999), individuals who recover during this developmental period may follow a relatively normative trajectory of drinking. Currently, most literature on "recovered" AUDs does not define the period in which individuals recovered from their disorder (Sobell et al., 2000). However, given our knowledge about developmentally limited disorders, the developmental period in which recovery occurs is theoretically important. For example, Moffitt (1993) identified that antisocial behavior has two distinct typologies: one that is limited to adolescence, and one that is life-course-persistent. In this case, the developmentally limited disorder of "adolescence-limited" antisocial behavior is thought to be less severe, as well as having a different etiology and trajectory than the "life-course-persistent" antisocial behavior (Moffitt, 1993). Similarly, it is possible that DLAUD has a different trajectory and etiology than persistent AUD.

If the etiology of DLAUD is different than the etiology of persistent AUD, it is possible that there will be differences in the risk conveyed to offspring, respectively. There are multiple reasons to believe that parental DLAUD would confer risk to offspring differently than a persistent parent AUD. Previous studies have shown that developmentally limited disorders conveyed decreased risk for consequences and decreased heritability than persistent disorders. Chassin et al. (2008) found that children of developmentally limited cigarette smokers were less likely to have ever smoked than the children of parents who were persistent smokers. Barnes et al. (2011) found that life-course persistent antisocial behavior is more strongly influenced by genetic factors than was adolescence-limited antisocial behavior, suggesting an increased likelihood of genetic risk to the next generation for individuals with life-course persistent antisocial behavior. Taken together, these results suggest that persistent AUD may be more heritable and show greater intergenerational transmission than does DLAUD (Zucker, 1986; Meier et al., 2013). Therefore, understanding the different trajectories of DLAUD as compared to persistent AUD could benefit intervention efforts, as parents with DLAUD may not need intervention that focuses on improving parenting skills.

Another important way that parents with DLAUD vs. persistent AUDs may differ is in their general parenting behaviors. Although no study to date has compared parenting specifically in DLAUD versus persistent AUD groups, there is evidence of poor parenting among parents with AUD. Compared to parents without AUD, parents with AUD are less consistent in rule enforcement (Tarter et al., 1993), show poor monitoring (Latendresse et al., 2008; Dishion and Loeber, 1985; Ary et al., 1999) and lower levels of warmth and nurturance than parents with no AUD (White et al., 2000; Brook and Brook, 1990). Moreover, there is some evidence that parenting behaviors and the family environment improve in parents who have recovered from substance use disorders (such as AUD) as compared to parents who have current substance use disorders (Bountress and Chassin, 2015; Thornberry et al., 2003), though one study suggests that parenting does not recover for fathers with recovered AUD (DeLucia et al., 2001). Still, persistent parental AUD may be related to lower rates of positive parenting, which in turn could be related to increased alcohol use and alcohol problems in children of parents with AUD. Parents with DLAUD likely do not have adolescent children while they are actively diagnosed with AUD, resulting in a greater likelihood of positive parenting during adolescence and lower risk of their children developing the AUD. Thus, parenting behaviors in parents with DLAUD may be less compromised than parents with persistent AUD.

In turn, less positive parenting has been linked to increased risk for offspring alcohol use (Ary et al., 1999). Positive parenting is of particular importance during early adolescence, which is considered a "critical period" of adolescence (Steinberg, 1991). During this time, increased parental monitoring and high levels of warmth and affection are linked to lower rates of alcohol use (Steinberg, 1991). Thus, parenting could be a mediator of the effects of parent's persistent AUD on offspring drinking.

Accordingly, the current study aimed to test whether positive parenting mediated the effects of persistent parental AUD versus parental DLAUD on emerging adulthood drinking and alcohol problems. We hypothesize that offspring of parents with persistent AUD will have more alcohol consumption and alcohol problems than offspring of parents with DLAUD and no AUD. Second, we hypothesize that offspring of parents with DLAUD will have higher rates of alcohol consumption and alcohol problems than offspring of parents with no AUD. Finally, we considered the effects of parent anxiety/depression, parent drug use disorder, and parent antisociality, as well as parent education in addition to offspring age, sex, and ethnicity.

2. Method

2.1. Participants

Participants were from the third generation of a multigenerational, longitudinal study of families with AUD and matched controls (see Chassin et al., 1992 for details of the original study). The original study began with 454 families with one adolescent (generation 2, or G2) and their parents (G1s). G1s and G2s were interviewed over six waves of data collection. Beginning at wave 5, the third generation (G3s) were also interviewed, as well as siblings of the G2s. G3s were also interviewed three times after wave six (waves 7–9). Participants included in the current study are G3s who reported about their drinking behaviors at least once between the ages of 18–25. The report of drinking was taken from anytime between wave 7 to wave 9. Because of age heterogeneity in the G3s, the wave where the G3 was between the ages of 18–25 (or closest to the mean age, if there was more than one wave that included a drinking report) was used in order to specifically examine alcohol problems and consumption during this age (the average age at which reports of drinking was assessed in this study was 20.4). Biological, custodial parents of the G3s selected for the current study were coded as either no AUD, DLAUD, or persistent AUD. There were few