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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.

Keywords

Alcohol use disorder; Parenting; Developmentally-limited

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Conflict of interest

No conflict declared.

Contributors

Every author listed on this manuscript was involved in the article preparation. AS was involved in writing, analyses, and literature review. DP was involved in the writing and editing of the manuscript. KE was involved in the editing and writing of the manuscript. LC was involved in the editing and writing of the manuscript. All authors have approved of the final article.

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 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 significant differences between included and excluded participants (see Supplemental document Table S1). Characteristics of the G3s included in the study are presented in Table 1. Overall,

59.8% were non-Hispanic Caucasian, and 51.2% were female; 178 participants had parents with no history of AUD, 59 had parents with DLAUD, and 124 had parents with persistent AUD.

2.2. Recruitment

Recruitment for the original study is described in detail in Chassin et al. (1992). Children of parents with AUD and their families were recruited using community telephone screenings, court records, and health maintenance organization (HMO) questionnaires. Matched control families (no parent AUD) were recruited via telephone surveys and were recruited in the same neighborhoods as the families with parents diagnosed with AUD. Control families were matched on child's age, family composition, ethnicity, SES, and neighborhood. Adult participants gave consent, and adolescents gave assent to be interviewed as a part of the larger longitudinal study. The Arizona State University Institutional Review Board approved all study protocols.

2.3. Measures

2.3.1. Offspring alcohol consumption and alcohol problems—Offspring (G3) alcohol consumption was a latent variable with three indicators. Past year typical quantity (1=Zero drinks to 9=Nine or more drinks) and frequency (1=Never to 8=Every day) questions were taken from Sher's (1987) Alcohol, Health, and Behavior Study Questionnaire. A third question created for the current study and designed to tap into early markers for high quantity drinking episodes (Chung et al., 2017) asked, "How often did you drink 3 or more drinks at one time in the past year?" with responses ranging from 1=Never to 8=Every day. These three items formed the offspring past year alcohol consumption latent factor. Fit statistics were not available due to a fully saturated model; however, all loadings were strong and significant (Range 0.62–0.81; $p < .01$).

The lifetime alcohol problems outcome measure was also a latent variable formed from indicators of alcohol-related social consequences and dependence symptoms from Sher's (1987) Alcohol, Health, and Behavior Study. Cumulative lifetime problems by emerging adulthood were used as an outcome due to low base rates in past year problems.¹ Examples of alcohol-related social consequences included "Experienced complaints from family or friends," and "Missed school or work." Dependence symptoms were taken from the DSM-IV diagnostic criteria including examples such as withdrawal symptoms and inability to cut down on drinking. Offspring lifetime problems latent factor score was created using a count of all alcohol-related social consequences, and all dependence symptoms, treated as count variables. Though fit statistics are not available due to fully saturated models, loadings for both latent factors were strong and significant (Range 0.64–.82; $p < .01$).

2.3.2. Positive parenting—Children reported on their mother and father's parenting in early adolescence, when G3 s were still living at home (wave 6; G3 mean age=11.6) to prospectively predict later alcohol outcomes. For the purposes of this study, only report on

¹Means of past year alcohol problems: No AUD=0.60, DLAUD=0.65, Persistent AUD=0.99. See online Supplement S3* for analysis of past year alcohol problems. An additional and separate factor was created for past year problems using past year alcohol-related social consequences and dependence symptoms.

biological, custodial parents were used to ensure report of parenting matched parent AUD status. If data was available for only one biological custodial parent, only that parent was used in analyses. Specifically, children completed measures of parental support, parental consistency of discipline, and parental monitoring. Consistency of discipline was taken from the Children's Report of Parental Behavior Inventory (CRPBI; Schaefer, 1965). These 10 items assessed parent's consistency of discipline on a scale ranging from 1=strongly disagree to 5=strongly agree and included items such as "My parents soon forget the rules he/she has made." Items were scored so that higher levels indicated more consistent discipline, and coefficient alpha was 0.74. Child's report of parental support was taken from the Network of Relations Inventory (Furman and Buhrmester, 1985), and included 7 items such as "How much can you count on your parent to be there when you need them." These items were rated (1=little to none, 5=the most possible) and were scored such that higher scores indicate higher levels of support and coefficient alpha was 0.88. Parental monitoring was taken from a measure created by Lamborn et al. (1991), which assessed what adolescents think parents know about their activities and interests. This included 5 items such as "How much does your parent know about who your friends are," and were scored on a scale from 1 (didn't know at all) to 5 (knew all the time). These items were scored so that higher scores indicate higher levels of parental monitoring and coefficient alpha was 0.83. Means across children's ratings of both parents on the summary scores of these three constructs were used to create a latent factor. Fit statistics were not available for the factor, as the model was fully saturated. However, all loadings were strong and significant (Range 0.6–0.8; $p < .01$).

2.3.3. Parental AUD—Parental diagnoses of DSM-IV alcohol dependence and abuse were assessed with the Diagnostic Interview Schedule (C-DIS; Robins et al., 1981) or other parent report using Family History-Research Diagnostic Criteria if only one parent was interviewed (FH-RDC; Andreasen et al., 1977) over the course of waves 4, 5, and 6 of data collection (no AUD=178, DLAUD=59, persistent AUD=124). The average age at each respective wave was 21.1, 25.7, and 34.1. Parents were categorized as no AUD if neither parent had ever met criteria for alcohol dependence or abuse at any of the three measurement waves. Parents were coded as DLAUD if one or more parents were diagnosed with alcohol dependence or abuse in the past year between the ages of 18–25² but no parent was diagnosed after those ages. Finally, parents were coded as persistent AUD if one or more parent maintained a past year diagnosis through the age of 30 or older.

2.3.4. Demographics—G3 participants reported on their age, gender, and ethnicity (Table 1). These variables were used as covariates, as they have all been identified as related to alcohol consumption and alcohol problems in children of parents with AUDs (Kim and Lee, 2011; Hussong et al., 2008a,b; Edwards et al., 2006; Caetano and Kaskutas, 1995). Race/ethnicity is a dichotomous variable (1=Non-Hispanic Caucasian, 2=Hispanic).

2.3.5. Parental anxiety/depression—Parental anxiety/depression (dichotomous yes/no anxiety or depression diagnoses, see Table 1) was assessed using the C-DIS (Robins et al.,

²Four individuals had a diagnosis of AUD at age 26–27 but not at age 30. For the purposes of the study, these individuals were categorized as DLAUD, as they recovered from their disorder before the age of 30.

1981). If either G2 parent had a lifetime diagnosis of anxiety or depression at wave 4, 5, or 6 (average parent ages=21.1, 25.7, and 34.1), this was coded as yes. If there was no diagnosis of lifetime parental anxiety or depression at any wave, this was coded as no. This variable was included as a covariate in analyses.

2.3.6. Parental antisociality—Clinical diagnoses of parent antisocial personality disorder were not possible because we did not assess conduct problems before age 18 in the C-DIS. However, parental antisociality symptoms were assessed using the C-DIS (Robins et al., 1981). All symptoms that either parent endorsed over all three waves of parental data collection (waves 4, 5, and 6; average parent ages 21.1, 25.7, and 34.1) were added together to create this count variable, which was included as a covariate (see Table 1).

2.3.7. Parent drug use disorder—Lifetime DSM-IV drug abuse or dependence disorder was assessed at all three waves of parental data collection (waves 4, 5, and 6; average ages 21.1, 25.7, and 34.1) using the C-DIS or Family History Research Diagnostic Criteria (FH-RDC; Andreasen et al., 1977). Drugs assessed were cocaine, inhalants, opiates, PCP, sedatives, amphetamines, hallucinogens, and cannabis. A diagnosis of drug use disorder of either parent at any wave was coded as yes (Table 1). This variable was included as a covariate in analyses.³

2.3.8. Parental education—The highest self-reported education of either parent was taken from wave 6, when parents were an average age of 34.1 (and might have been expected to have attained their maximum level of education) and included as a covariate (see Table 1). This response scale ranged from 1=8th grade or less to 11=completed graduate/professional school, and was treated as a continuous variable, where higher scores indicate higher education.

2.4. Data analytic plan

Structural equation modeling (SEM) with full information maximum likelihood (FIML) was used to conduct all data analyses. We examined all statistical assumptions relevant to these analyses a priori. Mediation models were estimated using Mplus 7.11 (Muthén and Muthén, 2015). Variables that were not normally distributed were addressed using maximum likelihood estimation with robust standard errors (MLR) in Mplus. Within the sample, there were young adults from the same family. In order to account for the clustering within the data, the family level variable was included and specified in Mplus, which adjusts the standard errors appropriately in multilevel data.

First, preliminary models were estimated testing the effects of the covariates (young adult age, ethnicity, gender, parent education, parent anxiety/depression, parent antisociality, and parent drug use disorder) on both the mediator (positive parenting) and the outcome variables. Non-significant covariates were then trimmed from the model to achieve the most parsimonious model fit (Little and Wang, 1996). Prior to running the full mediation model,

³Drug use disorder in parents was considered as a binary, yes/no lifetime diagnosis variable. However, it was also considered as a categorical variable, with parents identified as no drug use disorder, DLAUD drug use disorder, and persistent drug use disorder. Models were estimated with drug use disorder defined both ways and results did not differ.

the effect of parental AUD group on the outcome variable was estimated, in a model including all significant covariates. To understand which parental AUD group significantly differed from one another, dummy coding was utilized. Parental AUD group was dummy coded two ways: first with no parental AUD as a reference group, and then with parental DLAUD group as a reference group, to directly compare parental DLAUD to persistent parental AUD.

Next, positive parenting was tested as a mediator of the relation between parental AUD group and alcohol problems/alcohol use. Separate models were estimated to test positive parenting as a mediator of each outcome (i.e., lifetime alcohol problems, past year alcohol consumption). Dummy coding was used again to assess differences between parental AUD group, also in two models with no parental AUD, and then parental DLAUD as a reference group. Indirect effects were estimated using Mplus 7.11 (Muthén and Muthén, 2015). Full information maximum likelihood was used to account for missing data in all models.

3. Results

3.1. Descriptive data

Means, and standard deviations for the full sample are presented in Table 1 (see online Supplement S2 for a correlation table). Parental AUD groups were compared on all variables using ANOVAs with Tukey post hoc comparisons and chi-square tests with post hoc Bonferroni correction. The persistent parental AUD group and the parental DLAUD group showed significantly more parental anxiety/depression and parental drug use disorder than the no parental AUD group. The persistent group also showed significantly lower education than the no parental AUD group. The persistent parental AUD group showed significantly more parental antisociality than the no parental AUD group, and the parental DLAUD group showed significantly more parental antisociality than the no parental AUD group. There were no significant group differences in child age, gender, or ethnicity (see Table 1). The persistent parental AUD group showed significantly more offspring lifetime alcohol problems than the parental DLAUD group and the no parental AUD group. The persistent parental AUD group also showed more offspring alcohol consumption than the no parental AUD group, and less positive parenting than the no parental AUD group.

3.2. Full mediation model predicting offspring alcohol consumption

Covariates included in the model were parent antisociality, parental drug use disorder, offspring age and offspring ethnicity. In the direct model without the mediator included, the persistent parental AUD group had significantly more offspring alcohol consumption than the no parental AUD group ($B=0.139$, $SE=0.068$, $p < .05$). There were no significant differences among any other parental AUD groups. Then, the full mediation model was estimated, examining the effect of parental AUD group on offspring alcohol consumption mediated by positive parenting (see Table 2). The data were a good fit to the model, $\chi^2=2.898$, $p=.234$; $RMSEA=0.035$; $CFI=0.99$; $SRMR=0.012$. The persistent parental AUD group showed significantly less positive parenting than the no parental AUD group ($B=-0.134$, $SE=0.053$; $p < .05$). There were no other group differences in positive parenting. Positive parenting significantly predicted offspring alcohol consumption, such that higher

positive parenting predicted less offspring past year alcohol consumption ($B=-0.140$, $SE=0.055$; $p < .05$). In the full model, after the inclusion of the mediator, there was no longer a significant difference among any of the parental AUD groups on offspring past year alcohol consumption. Finally, there was a significant indirect effect of parental AUD group on offspring alcohol consumption only when comparing the no parental AUD group to the persistent parental AUD group ($B=0.019$, $SE=0.011$, $p < .05$), however the direct effect of the persistent vs. no parental AUD group did not remain significant after inclusion of positive parenting as a mediator. Thus, positive parenting fully mediated the relation between parental AUD group and offspring alcohol consumption.

3.3. Full mediation model predicting offspring lifetime alcohol problems

A second model estimated the effect of parental AUD group on offspring lifetime alcohol problems mediated by positive parenting. Covariates that remained in the model were parent antisociality, offspring age, and offspring ethnicity. In the direct model estimated prior to adding the mediator, the persistent parental AUD group had significantly more offspring lifetime alcohol problems than the no parental AUD group, ($B=0.194$, $SE=0.047$, $p < .01$), and the persistent parental AUD group had significantly more offspring lifetime alcohol problems than the parental DLAUD group ($B=0.173$, $SE=0.067$, $p < .05$).

The full mediation model was then estimated, using parental AUD group as a predictor of lifetime alcohol problems latent construct via the positive parenting latent construct (see Table 3). This model was a good fit to the data, $\chi^2=3.048$, $p=.218$; RMSEA=0.038; CFI=0.99; SRMR=0.017. The parental DLAUD group did not significantly differ from either the persistent or no parental AUD group on positive parenting. However, the persistent parental AUD group showed significantly less positive parenting than the no parental AUD group ($B=-0.134$, $SE=0.053$; $p < .05$). The effect of positive parenting on offspring lifetime alcohol problems was significant, such that higher positive parenting scores predicted fewer offspring lifetime alcohol problems ($B=-0.191$, $SE=0.056$; $p < .01$). The difference between the persistent parental AUD group and the no parental AUD group in offspring lifetime alcohol problems remained significant after the inclusion of the mediator, and the difference between the parental DLAUD group and the persistent parental AUD group on offspring lifetime alcohol problems also remained significant.

Finally, there was a significant indirect effect of parental AUD group on offspring lifetime alcohol problems through positive parenting only when comparing the no parental AUD group to the persistent parental AUD group ($B=0.040$, $SE=0.019$, $p < .05$), and the direct effect of persistent vs. no parental AUD group on offspring alcohol problems remained significant in the full model, indicating partial mediation when comparing these two groups. Positive parenting partially mediated the relation between parental AUD and lifetime alcohol problems only when comparing persistent parental AUD to no parental AUD.

4. Discussion

The current findings suggest a continuum of risk based on the heterogeneity of parental AUD diagnosis when considering DLAUD versus persistent parental AUD diagnosis. Therefore, differentiating DLAUD parent, AUD may be useful in describing the effects of

parent alcohol disorder with greater precision. The pattern of findings that emerged was that the parental DLAUD group remained an intermediate group in between the persistent and no parental AUD groups. The DLAUD parental AUD group was found to be at a significantly lower risk for offspring lifetime alcohol problems than the persistent parental AUD group. This finding is similar to results in Chassin et al. (2008), which indicated that parents who were identified as developmentally limited cigarette smokers had adolescents at low risk to be smokers themselves whereas early onset persistent smoking showed the most intergenerational transmission. Thus, DLAUD may be more normative and less severe, and less heritable than persistent AUD (Edwards and Kendler, 2013; Meier et al., 2013; Zucker and Noll, 1987).

The parental DLAUD group did not significantly differ from either the persistent parental AUD group or the no parental AUD group in either positive parenting or offspring alcohol consumption. This is consistent with prior work, such that alcohol problems, in particular, are more likely to be associated with parental AUD, externalizing behaviors and conduct problems over and above alcohol consumption (Lieb et al., 2002; Stice et al., 1998). Therefore, dysregulated individuals with externalizing symptomology are more likely to drink in risky ways and thus have alcohol problems, though levels of consumption may not differ from others (Stice et al., 1998). The parental DLAUD group may consume similar amounts of alcohol as the persistent parental AUD group, but experience fewer problems as they may be less dysregulated. The pattern of findings in the current study suggest that previous reports of higher rates of alcohol consumption and lower rates of positive parenting in offspring with a history of lifetime parental AUD as compared to offspring with no history of lifetime AUD (Chassin et al., 1992) are likely driven by differences between persistent parental AUD versus no parental AUD.

Importantly, this pattern of findings is similar to but not identical to previous literature comparing recovered parental AUDs to current parental AUDs. In the current study, the DLAUD group never significantly differed from the no parental AUD group. In contrast, in previous studies, the recovered group has still been found to be at significantly higher risk than the no parental AUD group (Chassin et al., 1991). Differences between the current findings and studies of 'recovered' parent AUD may mean that the developmental period of parental recovery is important to elucidate differential transmission of risk.

Moreover, the difference between the persistent parental AUD group and the no parental AUD group on offspring lifetime alcohol problems and alcohol consumption can be partially explained by positive parenting. This finding replicates previous studies in which parental AUD prospectively predicted offspring alcohol problems and consumption through a lack of positive parenting (Chassin et al., 1992; Ary et al., 1999). Notably, there was only evidence of mediation when comparing the persistent parental AUD group to the no parental AUD group, suggesting that positive parenting strategies are particularly important for parents whose AUDs persist past emerging adulthood. Theoretically, these parents are likely to be more impaired in terms of their parenting than those with no AUD. If their disorder is concurrent with their role as parents, they are likely to struggle in fulfilling their parental responsibilities, resulting in less positive parenting (Latendresse et al., 2008).

In addition, positive parenting did not explain the significant difference between the persistent parental AUD group and the DLAUD parental AUD group in offspring lifetime alcohol problems. This may be because general parenting is not as important in the development of alcohol-related problems as alcohol-specific socialization and parenting practices for these two groups. Effects of alcohol-specific parenting practices have been shown to be distinct from general parenting practices (Handley and Chassin, 2013), and in the smoking literature, parents who quit smoking have been shown to have as strong anti-smoking socialization and parenting as parents who never smoked (Chassin et al., 2005). Therefore, DLAUD parents may have alcohol-specific socialization practices for their offspring that are more similar to parents with no AUD. Moreover, the small differences between DLAUD and persistent AUD groups in positive parenting might have been detected with larger sample size, as the mean levels of positive parenting indicated that the DLAUD group fell between the persistent and no AUD groups.

Methodologically, the current study has important strengths including latent variables as outcomes, prospective prediction in longitudinal mediational models, as well as direct ascertainment of parent AUD over multiple waves and consideration of important covariates. However, there are also limitations to consider. The lack of significant differences between the parental DLAUD group and the other parental AUD groups in alcohol consumption and positive parenting could be due to a lack of statistical power. The parental DLAUD group was the smallest of all three groups and contained fewer than 100 participants. In addition, base rates of past year alcohol problems were extremely low in the sample, and therefore we chose to use lifetime alcohol problems instead. Future studies with more participants may also consider past year alcohol problems to assess the current state of offspring emergent adults better.

Another limitation is that the current study did not measure whether or not offspring currently live with their parents and the living situation may impact their current alcohol consumption. Future studies may want to consider assessing the living situation of the offspring. Moreover, as noted earlier, parental DLAUD represents a special case of parental AUD recovery, and the current study cannot compare the effects of parental AUD recovery during emerging adulthood from recovery during other developmental periods. Further research is necessary to make that distinction. Additionally, other types of heterogeneity will be important to consider, such as the number of parents with AUD, and severity of AUD. Future studies will need to consider how to best measure diagnoses of AUD to consider the potential for maximum heterogeneity.

In summary, the current study adds to the literature by suggesting that heterogeneity in parental AUD diagnosis that considers recovery in emerging adulthood may convey differential risk for children of parents with AUD. Results suggest that simply considering lifetime parental AUD as a dichotomous variable may be imprecise and results may be driven by persistent parental AUDs. Further investigation of these AUD group differences is important in informing prevention efforts, as it is possible that the offspring of parents with DLAUD may not need to be the focus of prevention programs despite a positive parental AUD history. Furthermore, a focus on positive parenting practices as a preventative measure for offspring substance use may only be beneficial for parents with persistent AUD, and thus

prevention and intervention programs can be tailored to parents with persistent AUD vs. DLAUD. Understanding more about the developmental period in which recovery from AUD occurs, and how recovery during the particular period may affect the next generation could potentially illuminate specific subgroups for intervention.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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References

- Andreasen NC, Endicott J, Spitzer RL, Winokur G. The reliability and validity of the family history method using family history research diagnostic criteria (FH-RDC). *Arch Gen Psychiatr*. 1977; 34:1229–1235. [PubMed: 911222]
- Arnett JJ. Emerging adulthood: what is it, and what is it good for? *Child Dev Perspect*. 2007; 2:68–73.
- Ary DV, Duncan TE, Biglan A, Metzler CW, Noell JW, Smolkowski K. Development of adolescent problem behavior. *J Abnorm Child Psych*. 1999; 27:41–150.
- Bachman, JG., Wadsworth, KN., O'Malley, PM., Johnston, LD., Schulenberg, JE. *The Impact of New Freedoms and New Responsibilities*. LE Associates; New Jersey: 1997. Smoking, Drinking, and Drug Use in Young Adulthood.
- Barnes JC, Beaver KM, Boutwell BB. Examining the genetic underpinnings to Moffitt's developmental taxonomy: a behavioral genetic analysis. *Criminology*. 2011; 49:923–954.
- Bountress K, Chassin L. Risk for behavior problems in children of parents with substance use disorders. *Am J Orthopsychiat*. 2015; 85:275. [PubMed: 25985113]
- Brook, DW., Brook, JS. *Drug and Alcohol Abuse Prevention*. Humana Press; Totowa, NJ: 1990. The etiology and consequences of adolescent drug use; p. 339–362.
- Caetano R, Kaskutas LA. Changes in drinking patterns among whites, blacks and Hispanics, 1984–1992. *J Stud Alcohol*. 1995; 56:558–565. [PubMed: 7475037]
- Chassin L, Rogosch F, Barrera M Jr. Substance use and symptomatology among adolescent children of alcoholics. *J Abnorm Psychol*. 1991; 100:449–463. [PubMed: 1757658]
- Chassin L, Barrera M Jr, Bech K, Kossak-Fuller J. Recruiting a community sample of adolescent children of alcoholics: a comparison of three subject sources. *J Stud Alcohol*. 1992; 53:316–319. [PubMed: 1619925]
- Chassin L, Presson CC, Rose J, Sherman SJ, Davis MJ, Gonzalez JL. Parenting style and smoking-specific parenting practices as predictors of adolescent smoking onset. *J Pediatr Psychol*. 2005; 30:333–344. [PubMed: 15863430]
- Chassin L, Presson C, Seo DC, Sherman SJ, Macy J, Wirth RJ, Curran P. Multiple trajectories of cigarette smoking and the intergenerational transmission of smoking: a multigenerational, longitudinal study of a Midwestern community sample. *Health Psychol*. 2008; 27:819–828. [PubMed: 19025278]
- Chung T, Creswell KG, Bachrach R, Clark DB, Matrin CS. Adolescent binge drinking: developmental context and opportunities for prevention. *Alcohol Res Curr Rev*. 2017; 39(1):e1–e11.

- DeLucia C, Belz A, Chassin L. Do adolescent symptomatology and family environment vary over time with fluctuations in paternal alcohol impairment? *Dev Psych.* 2001; 37:207.
- Dishion TJ, Loeber R. Adolescent marijuana and alcohol use: the role of parents and peers revisited. *Am J Drug Alcohol Abuse.* 1985; 11:11–25. [PubMed: 4061428]
- Edwards AC, Kendler KS. Alcohol consumption in men is influenced by qualitatively different genetic factors in adolescence and adulthood. *Psychol Med.* 2013; 43:1857–1868. [PubMed: 23282961]
- Edwards EP, Eiden RD, Colder C, Leonard KE. The development of aggression in 18–48 month old children of alcoholic parents. *J Abnorm Child Psychol.* 2006; 34:393–407. [PubMed: 16649001]
- Furman W, Buhrmester D. Children's perceptions of the personal relationships in their social networks. *Dev Psychol.* 1985; 21:1016–1024.
- Handley ED, Chassin L. Alcohol-specific parenting as a mechanism of parental drinking and alcohol use disorder risk on adolescent alcohol use onset. *J Stud Alcohol Drugs.* 2013; 74:684–693. [PubMed: 23948527]
- Hussong AM, Bauer DJ, Huang W, Chassin L, Sher KJ, Zucker RA. Characterizing the life stressors of children of alcoholic parents. *J Fam Psychol.* 2008a; 22:819–832. [PubMed: 19102603]
- Hussong AM, Flora DB, Curran PJ, Chassin LA, Zucker RA. Defining risk heterogeneity for internalizing symptoms among children of alcoholic parents. *Dev Psychopathol.* 2008b; 20:165–193. [PubMed: 18211733]
- Jellinek, EM. *The Disease Concept of Alcoholism.* Hillhouse Press; New Haven, CT: 1960.
- Johnston LD, Bachman JG, O'Malley PM, Schulenberg JE. *Monitoring the Future: A Continuing Study of American Youth (12th-Grade Survey), 2000. Codebook for 12th Grade, Form 1.* 2004
- Kim HK, Lee MY. Factors influencing resilience of adult children of alcoholics among college students. *J Korean Acad Nurs.* 2011; 41:642–651. [PubMed: 22143213]
- Lamborn SD, Mounts NS, Steinberg L, Dornbusch SM. Patterns of competence and adjustment among adolescents from authoritative, authoritarian, indulgent, and neglectful families. *Child Dev.* 1991; 62:1049. [PubMed: 1756655]
- Latendresse SJ, Rose RJ, Viken RJ, Pulkkinen L, Kaprio J, Dick DM. Parenting mechanisms in links between parents' and adolescents' alcohol use behaviors. *Alcohol Clin Exp Res.* 2008; 32:322. [PubMed: 18162066]
- Lieb R, Merikangas KR, Höfler M, Pfister H, Isensee B, Wittchen HU. Parental alcohol use disorders and alcohol use and disorders in offspring: a community study. *Psychol Med.* 2002; 32(1):63–78. [PubMed: 11883731]
- Little RJ, Wang Y. Pattern-mixture models for multivariate incomplete data with covariates. *Biometrics.* 1996;98. [PubMed: 8934587]
- Maggs JL, Schulenberg JE. Trajectories of alcohol use during the transition to adulthood. *Alcohol Res.* 2004; 28:195.
- McGue, M. Genes, environment, and the etiology of alcoholism. In: Zucker, RA, Boyd, GM., Howard, J., editors. *Development of Alcohol Problems: Exploring the Biopsychosocial Matrix of Risk.* Rockville, MD: 1994. p. 1-40. NIAAA Research Monograph No. 26
- Meier MH, Caspi A, Houts R, Slutske WS, Harrington H, Jackson KM, Belsky DW, Poulton R, Moffitt TE. Prospective developmental subtypes of alcohol dependence from age 18–32 years: Implications for nosology, etiology, and intervention. *Dev Psychopathol.* 2013; 25:785. [PubMed: 23880392]
- Moffitt TE. Adolescence-limited and life-course-persistent antisocial behavior: a developmental taxonomy. *Psychol Rev.* 1993; 100:674. [PubMed: 8255953]
- Mokdad AH, Marks JS, Stroup DF, Gerberding JL. Actual causes of death in the United States, 2000. *JAMA.* 2004; 291:1238. [PubMed: 15010446]
- Muthén LK, Muthén B. *Mplus. The Comprehensive Modelling Program for Applied Researchers: User's Guide.* 2015:5.
- Nirenberg TD, Liepmann MR, Begin AM, Maisto SA, Liebermann MP. Family history of alcoholism in males: absence of distinguishing features for treatment matching. *Int J Addict.* 1990; 25:1195. [PubMed: 2090623]

- Ohannessian CM, Stabenau JR, Hesselbrock VM. Childhood and adulthood temperament and problem behaviors and adulthood substance use. *Addict Behav.* 1995; 20:77. [PubMed: 7785484]
- Robins LN, Helzer JE, Croughan J, Ratcliff KS. National Institute of mental health diagnostic interview schedule: its history, characteristics and validity. *Arch Gen Psychiatry.* 1981; 38:381. [PubMed: 6260053]
- Rogosch F, Chassin L, Sher KJ. Personality variables as mediators and moderators of family history risk for alcoholism: conceptual and methodological issues. *J Stud Alcohol.* 1990; 51:310. [PubMed: 2359303]
- Schaefer ES. Children's reports of parental behavior: an inventory. *Child Dev.* 1965:413. [PubMed: 14300862]
- Sher KJ, Walitzer KS, Wood PK, Brent EE. Characteristics of children of alcoholics: putative risk factors, substance use and abuse, and psychopathology. *J Abnorm Psychol.* 1991; 100:427. [PubMed: 1757657]
- Sher, KJ., Trull, TJ., Bartholow, BD., Vieth, A., Leonard, KE., Blane, HT. *Personality and Alcoholism: Issues, Methods, and Etiological Processes Psychological Theories of Drinking and Alcoholism.* Guilford Press; New York: 1999. p. 105
- Sher, KJ. Questionnaire for the Alcohol, Health, and Behavior Study. Columbia: University of Missouri; 1987. Unpublished Manuscript
- Sobell LC, Ellingstad TP, Sobell MB. Natural recovery from alcohol and drug problems: methodological review of the research with suggestions for future directions. *Addiction.* 2000; 95:749. [PubMed: 10885050]
- Steinberg, L. Adolescent Transitions and Alcohol and Other Drug Use Prevention in Preventing Adolescent Drug Use: From Theory to Practice. Gopelrud, EN., editor. Office for Substance Use Prevention; Maryland: 1991. p. 13-51.
- Stice E, Barrera M Jr, Chassin L. Prospective differential prediction of adolescent alcohol use and problem use: examining the mechanisms of effect. *J Abnorm Psychol.* 1998; 107(4):616. [PubMed: 9830249]
- Tarter RE, Blackson T, Martin C, Loeber R, Moss HB. Characteristics and correlates of child discipline practices in substance abuse and normal families. *Am J Addict.* 1993; 2:18.
- Thornberry TP, Freeman-Gallant A, Lizotte AJ, Krohn MD, Smith CA. Linked lives: the intergenerational transmission of antisocial behavior. *J Abnorm Child Psychol.* 2003; 31:171. [PubMed: 12735399]
- White HR, Johnson V, Buyske S. Parental modeling and parenting behavior effects on offspring alcohol and cigarette use: a growth curve analysis. *J Subst Abuse.* 2000; 12:287. [PubMed: 11367605]
- Zucker RA, Noll RB. The interaction of child and environment in the early development of drug involvement: a far ranging review and a planned very early intervention. *Drugs Soc.* 1987; 2:57.
- Zucker, RA. *Nebraska Symposium on Motivation.* University of Nebraska Press; 1986. The four alcoholisms: a developmental account of the etiologic process.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at <https://doi.org/10.1016/j.drugalcdep.2018.03.027>.

Table 1

Descriptive Statistics for All Variables by Parental Alcohol Use Disorder Group.

	No Parental AUD			DL Parental AUD			Persistent Parental AUD			Total Sample, N=361		
	N	Mean	SD	N	Mean	SD	N	Mean	SD	N	Mean	SD
Offspring Alcohol Consumption	178	-0.19 ^a	0.96	59	0.02 ^{ab}	0.92	124	0.28 ^b	0.88	361	0.00	0.95
Offspring Lifetime Alcohol Problems	178	-0.13 ^a	0.68	59	-0.05 ^a	0.62	124	0.24 ^b	0.82	361	0.137	0.74
Offspring Age	178	20.32 ^a	2.14	59	20.40 ^a	2.00	124	20.46 ^a	2.18	361	20.40	2.30
Positive Parenting	178	0.08 ^a	0.89	59	-0.15 ^{ab}	0.87	124	-0.31 ^b	0.82	361	-0.092	0.88
Parent Antisociality	178	1.95 ^a	1.19	59	3.25 ^b	2.52	124	4.06 ^c	2.47	361	2.89	2.43
Parent Education	178	6.78 ^a	0.19	59	6.59 ^{ab}	0.33 ^a	124	5.78 ^b	0.21	361	6.39	2.37
	N	%		N	%		N	%		N	%	
Offspring Ethnicity	178	64.0% Caucasian ^a		59	59.3% Caucasian ^a		124	54.0% Caucasian ^a		361	59.8% Caucasian	
Offspring Gender	178	54.5% Female ^a		59	42.4% Female ^a		124	50.8% Female ^a		361	51.2% Female	
Parent Anxiety/Depression Dx	178	65.2% No Dx ^a		59	45.8% No Dx ^b		124	46% No Dx ^b		361	55% No Dx	
Parent Drug Use Disorder Dx	178	87.6% No Dx ^a		59	42.4% No Dx ^b		124	34.7% No Dx ^b		361	62.0% No Dx	

Note: Dx refers to diagnosis; No Dx means absence of any diagnosis; Parent antisociality is a symptom count, where higher scores indicate more symptoms.

Note: Means with different subscripts within parental AUD groups are significantly different at $p < .05$ using ANOVA with Tukey post hoc between group comparisons and chi square tests with Bonferroni correction post hoc between group comparisons.

Note: Offspring alcohol consumption, offspring lifetime alcohol problems, and positive parenting are factor scores, where negative scores indicate low levels, scores of 0 indicate average levels, and positive scores indicate high levels of alcohol consumption, alcohol problems, and positive parenting.

Note: Raw variables of alcohol consumption are as follows: no AUD=1.9, DLAUD=2.4, persistent AUD=2.8; Raw variables of alcohol problems are no AUD=1.0, DLAUD=1.2, persistent AUD=2.0; and raw variable mean levels of positive parenting are no AUD=4.1; DLAUD=3.9; persistent AUD=3.8.

Note: Parent Education is an ordered categorical variable where higher scores indicate higher levels of education; mean level in total sample indicates graduated high school and completed some vocational/technical school.

Standardized Coefficients of the Effect of Parental Alcohol Use Group on Offspring Alcohol Consumption through Positive Parenting Strategies, N=361.

Table 2

Predictor/Covariates	Mediator		Dependent Variable	
	Positive Parenting Strategies		Offspring Alcohol Consumption	
	B	SE	B	SE
Positive Parenting	–	–	–0.140	0.055
Offspring Ethnicity	–0.125	0.051	–	–
Offspring Age	–0.275	0.051	–0.275	0.044
Parent Antisociality	–0.134	0.060	–	–
Parent Drug Use Disorder	–	–	0.125	0.057
Comparison of Parental AUD groups without Positive Parenting Strategies (Baseline Model)				
None vs. DL	–	–	0.021	0.060
None vs. Persistent	–	–	0.139	0.068
DL vs. Persistent	–	–	0.111	0.068
Comparison of Parental AUD Groups with Positive Parenting Strategies				
None vs. DL	–0.066	0.061	0.015	0.058
None vs. Persistent	–0.134	0.053	0.122	0.066
DL vs. Persistent	–0.049	0.081	0.103	0.066

Note. Non-significant covariate paths were trimmed for most parsimonious model fit and are represented with –.

Note. See Fig. 1 in Supplementary materials for specific paths with beta coefficients.

* $p < 0.05$.

** $p < 0.01$.

Table 3

Standardized Coefficients of the Effect of Parental Alcohol Use Group on Offspring Lifetime Alcohol Problems through Positive Parenting Strategies, N=361.

Predictor/Covariates	Mediator		Dependent Variable	
	Positive Parenting Strategies		Offspring Lifetime Alcohol Problems	
	B	SE	B	SE
Positive Parenting	–	–	–0.191	0.056
Offspring Ethnicity	–0.125	0.051	–	–
Offspring Age	–0.275	0.051	0.071	0.021
Parent Antisociality	–0.134	0.060	–	–
Comparison of Parental AUD groups without Positive Parenting Strategies (Baseline Model)				
None vs. DL	–	–	0.021	0.064
None vs. Persistent	–	–	0.194	0.063
DL vs. Persistent	–	–	0.173	0.067
Comparison of Parental AUD Groups				
None vs. DL	–0.066	0.061	0.013	0.047
None vs. Persistent	–0.134	0.053	0.187	0.062
DL vs. Persistent	–0.049	0.081	0.170	0.066

Note. Non-significant covariate paths were trimmed for most parsimonious model fit and are represented with –.

Note. Regression of parental AUD groups on the mediator of positive parenting strategies remain identical to the model shown in Table 2, however the effect of parental AUD group on the outcome, as well as effect of the mediator on the outcome, differs.

Note. See Fig. 2 in Supplementary materials for specific paths with beta coefficients.

*
p < .05.

**
p < .01.