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Anxiety disorders and risk for alcohol use disorders: The moderating effect of parental support

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ABSTRACT

Background: There have been mixed findings on the temporal relation between anxiety disorders and alcohol use disorders (AUDs), suggesting that the pathway to AUDs may differ among individuals. The aim of the current study was to test whether parental support moderated the association between anxiety disorders and the development of AUDs. We also tested whether our effects differed as a function of age of AUD onset.
Methods: 817 individuals were assessed for lifetime diagnoses of psychopathology during 4-waves between adolescence (mean age = 16) and adulthood (mean age = 30).
Results: Proportional hazards model analyses indicated that baseline anxiety disorders interacted with baseline perceived maternal support to prospectively predict onset of AUDs. At high levels of maternal support, anxiety disorders were associated with a *reduced* risk for AUD onset (HR = 0.74, 95% CI = 0.55–1.00). However, this effect was more robust for AUDs that developed prior to age 20. At low levels of maternal support, anxiety disorders were associated with an *increased* risk for AUD onset (HR = 1.65, 95% CI = 1.21–2.26). This effect was present for AUDs that developed across adolescence and adulthood. Paternal support was not associated with AUDs and did not interact with anxiety disorders.
Conclusions: Prevention and intervention efforts targeted at maternal support in adolescents with anxiety disorders may be valuable, as this may represent a factor that has a significant impact on the developmental course of AUDs.

1. Introduction

Lifetime diagnoses of alcohol abuse and dependence are common (Hasin et al., 2007) and associated with serious adverse consequences (Rehm et al., 2009; Mokdad et al., 2004; Wilcox et al., 2004). Numerous risk factors for alcohol use disorders (AUDs) have been identified (Sher et al., 2005; Tucker et al., 2008; Patterson et al., 2000). However, the identification of at-risk populations continues to be a major public health issue and there is a critical need to examine interactions among risk factors to improve prevention and intervention approaches.

There are multiple developmental pathways to AUD onset. One of the most robust trajectories is via externalizing personality traits and disorders (see Zucker, 2006, 2008 for reviews). Broadly, evidence indicates that externalizing disorders, such as attention-deficit-hyperactivity disorder (ADHD) and conduct disorder (CD; Charach et al., 2011; Nock et al., 2006), as well as externalizing traits, such as impulsivity and sensation seeking (Verdejo-García et al., 2008), are associated with AUD onset. It has been theorized that externalizing traits/symptoms emerge early in development and are associated with an inability or unwillingness to inhibit behaviors despite negative consequences (Iacono et al., 2008). These traits, coupled with a high-risk environment, are thought to ultimately propel individuals along the externalizing pathway to AUD onset (Zucker, 2006).

Notably, research indicates that not all individuals develop AUDs via the externalizing pathway and that there may be a separate internalizing pathway (see Hussong et al., 2011). Studies have shown that internalizing personality traits and disorders, including anxiety, depression and neuroticism, also lead to the onset of AUDs (Dixit and Crum, 2000; Kushner et al., 2012; Littlefield et al., 2010). Although the mechanisms underlying the internalizing pathway

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are not fully understood, evidence suggests that individuals with high levels of negative affect engage in alcohol use to alleviate their aversive affective states (Bolton et al., 2009). The use of alcohol as a means of avoidance-based coping becomes negatively reinforced over time, increasing the likelihood and maintenance of AUDs (Crum et al., 2013; Baker et al., 2004).

To date, the literature on the role of anxiety disorders in the development of AUDs has been mixed, calling into question the etiological validity of the internalizing pathway. Several studies have demonstrated that anxiety disorders increase the risk for AUD onset (Buckner et al., 2009; Zimmermann et al., 2003), whereas others have reported weak effects (Crum and Pratt, 2001). Some studies have also found the opposite relation – anxiety disorders and symptoms are associated with a *reduced* risk for AUDs (Eggleston et al., 2004; Kaplow et al., 2001). Considering these disparate findings, it is likely that key factors influence the direction and strength of the anxiety to AUD pathway. More specifically, it is possible that anxiety disorders can act as either risk or protective factors, as a function of different traits or features (i.e., moderators). It is necessary to consider that individuals with anxiety disorders are often behaviorally inhibited (Clauss and Blackford, 2012) and consequently may fail to affiliate with peers (Booth-LaForce et al., 2012). Since drinking often occurs in social settings (particularly in young adulthood), it is possible that some individuals with anxiety disorders are avoidant of social situations and novel stimuli (i.e., alcohol) which *decreases* their likelihood of developing AUDs (see Kagan, 2007).

One important developmental variable which may influence the association between anxiety disorders and AUDs is parental support. Parental support has been defined in several ways but typically encompasses parental displays of companionship, intimacy, affection, and instrumental aid. Research indicates that adolescents with low levels of parental support have reduced self-esteem and poor social skills which make them vulnerable to peer pressure, deviant behavior, and alcohol use (Parker and Benson, 2004). High levels of parental support have the opposite effect and have been shown to buffer adolescents from risk factors and enhance the impact of protective factors (Stadler et al., 2010; Wills and Cleary, 1996). Given that the influence of parental factors may be greater among those with high levels of negative affect (Wills et al., 2001), it is possible that the relationship between anxiety disorders and AUDs differs as a function of parental support. In other words, at low levels of parental support, individuals with anxiety disorders may be at risk for AUDs due to reduced self-esteem and vulnerability to affiliate with deviant peers. In contrast, at high levels of parental support, individuals with anxiety disorders may spend more time with parents (and less with peers) and thus, be protected from alcohol use initiation and AUD onset.

Another important factor which may contribute to previous mixed findings is the role of developmental period (Hussong et al., 2013). Research indicates that risk processes can differ as a function of age and environment (Dick et al., 2006; Hussong et al., 2011; Kendler et al., 2008). For example, Sung and colleagues (2004) found that anxiety disorders increased the risk of developing SUDs in girls at age 16, but prior to this, anxiety disorders were unrelated to SUDs. Elkins et al. (2006) also found effects of timing of disorder onset such that the personality trait 'low constraint' was more robustly associated with the development of AUDs prior to age 17 relative to ages 17–20. This suggests that the effects of anxiety disorders and parental support may differ as a function of age/developmental period.

The aim of the current study is to test: (1) whether maternal and/or paternal support moderates the association between anxiety disorders and the development of AUDs and (2) whether the unique and interactive effects of anxiety disorders and parental support differ as a function of age of AUD onset. Data come from the Oregon Adolescent Depression Project (OADP; Lewinsohn et al., 1993) – an extensive 4-wave longitudinal study on adolescent and adult psychopathology. Anxiety disorders included in the study are social phobia, specific phobia, panic disorder, obsessive–compulsive disorder, and separation anxiety disorder. Based on the reviewed literature, we hypothesized that at low maternal support, but not high maternal support, anxiety disorders would increase the risk of AUD onset.

Table 1
Participant demographics, clinical characteristics, and study variables.

	Those without anxiety disorders at T1 ($n = 737$)	Those with anxiety disorders at T1 ($n = 80$)
Demographic variables at T1		
Age (years; *SD*)	16.6 (1.2)$_a$	16.7 (1.1)$_a$
Gender (% female)	56.3%$_a$	70.0%$_b$
Race (% Caucasian)	89.9%$_a$	92.1%$_a$
Average parent education		
Less than high school degree	7.5%$_a$	10.0%$_a$
High school degree or equivalent	62.4%$_a$	66.3%$_a$
Greater than high school degree	30.1%$_a$	23.7%$_a$
Alcohol use disorders T2–T4		
Alcohol abuse	15.7%$_a$	11.3%$_a$
Alcohol dependence	18.5%$_a$	23.8%$_a$
Study variables at T1		
Maternal support	0.2 (4.3)$_a$	−0.3 (3.8)$_a$
Paternal support	−0.1 (4.2)$_a$	0.1 (4.4)$_a$
No. of people in the household	3.1 (1.4)$_a$	3.0 (1.6)$_a$
Mother is biological mother	90.1%$_a$	91.3%$_a$
Father is biological father	67.2%$_a$	57.5%$_a$
Birth order	1.9 (1.1)$_a$	2.0 (1.3)$_a$
Major depressive disorder Through T1	22.5%$_a$	41.3%$_b$
Externalizing disorder through T1	10.3%$_a$	16.3%$_a$
Coping skills	47.5 (7.5)$_a$	45.9 (7.9)$_a$

Note: Means or percentages with different subscripts across rows were significantly different in pairwise comparisons ($p < .05$, Chi-square test for categorical variables and Tukey's honestly significant difference test for continuous variables).

2. Methods

2.1. Participants and procedures

Detailed descriptions of recruitment procedures, participation rates, and sample characteristics have previously been reported (Lewinsohn et al., 1993, 2003; Olino et al., 2008; Rohde et al., 2007). Briefly, participants were randomly selected from nine high schools in western Oregon. A total of 1709 adolescents (mean age 16.6 years [*SD* = 1.2]) completed the initial (T1) assessment between 1987 and 1989. Approximately one year later, 1507 of the adolescents completed the second assessment (T2). Once participants were 24 years old, a third wave (T3) of data collection took place. By design, only a subset of 941 individuals, over-sampled for psychopathology and non-white race, completed the T3 evaluation. Thus, those without a history of psychopathology by T2 were under-sampled. To account for this sampling approach, in each model adolescents were weighted based on their probability of being invited to participate in T3. At age 30, 816 of the participants completed a T4 assessment. Though differences between participants and non-participants at each wave were minor (Lewinsohn et al., 1993; Olino et al., 2008), individuals with a lifetime diagnosis of AUDs evidenced significantly higher attrition rates (17%) at the T4 assessment relative to individuals with no history of AUDs (12%). Higher attrition rates were also found for men versus women (16% versus 11%) and for those with and without a history of cannabis use disorders (18% versus 12%).

Participants were excluded from the current study if they did not participate in the T3 ($n = 566$). An individual was classified as having an anxiety disorder ($n = 80$) if they had a lifetime diagnosis by T1 of social phobia ($n = 12$), specific phobia ($n = 23$), panic disorder ($n = 4$), obsessive–compulsive disorder ($n = 3$), or separation anxiety disorder ($n = 38$). Of note, 11 individuals had more than one anxiety disorder diagnosis at T1. Analyses indicated that there were no differences between individuals with anxiety disorders at T1 that did and did not complete the T3 assessment on any major demographic or diagnostic variables. We excluded adolescents with a T1 lifetime diagnosis of alcohol abuse or dependence ($n = 61$) so that we could test the temporal relation between anxiety disorders and first onset of an AUD. Lastly, a subset of participants were excluded due to missing parental support ($n = 16$) or covariate data ($n = 47$). Our final sample included 817 individuals. Demographic and clinical information from the T1 assessment is provided in Table 1.

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