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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.

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Contributors

Peter Lewinsohn was the principal investigator of the Oregon Adolescent Depression Project. John Seeley contributed to the design of the study and was involved in the collection and management of data. Thomas Olino, Derek Kosty, and Stewart Shankman provided assistance with statistical analyses and made important contributions to the editing of the manuscript. Stephanie Gorka provided the rationale for the paper, conducted the statistical analyses, and wrote the first draft of the manuscript. All authors contributed and have approved the final manuscript.

Conflict of Interest

All authors declare that they have no conflicts of interest.

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.

Keywords

anxiety disorders; alcohol use disorders; maternal support

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 et al., 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 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 (Behrendt et al., 2011; Buckner et al., 2008), 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 to 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.

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 1,709 adolescents (mean age 16.6 years [*SD* = 1.2]) completed the initial (T1) assessment between 1987 and 1989. Approximately one year later, 1,507 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% vs. 11%) and for those with and without a history of cannabis use disorders (18% vs. 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.

2.2 Diagnostic Assessments

Current and lifetime diagnoses were assessed directly at T1 and T2 and by telephone at T3 and T4. Phone interviews and direct interviews yielded high inter-method reliability for diagnoses and symptoms (Rohde et al., 1997). Participants were interviewed at T1 with a modified version of the Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS; Chambers et al., 1985; Orvaschel et al., 1982), which included additional items used to derive *DSM-III-R* diagnoses (APA, 1987). At T2 and T3, participants were interviewed using the K-SADS and the Longitudinal Interval Follow-up Evaluation (LIFE; Keller et al., 1987) to assess psychopathology since the last interview. At T4, participants were administered the LIFE interview and the Structured Clinical Interview for Axis-I *DSM-IV* Disorders Non-Patient Edition (SCID-NP; First et al., 1996). Therefore,

diagnoses were made according to *DSM-III-R* criteria (APA, 1987) for T1 and T2 and *DSM-IV* criteria (APA, 1994) for T3 and T4.

All interviewers were carefully trained and supervised, and completed a 70-hour course in diagnostic interviewing. The inter-rater reliability of lifetime diagnoses for any anxiety disorder (kappa = .94) and AUDs (kappa = .97) were excellent. Individuals were considered to have an anxiety disorder if they had a lifetime (i.e., current or past) diagnosis of an aforementioned anxiety disorder by the T1 assessment. At the T1 evaluation, 29 individuals had a current diagnosis of an anxiety disorder and 51 had a past diagnosis of an anxiety disorder. AUDs were coded as diagnoses of alcohol abuse or alcohol dependence starting after T1 through T4 (yes/no).

2.3 Maternal and Paternal Support

At the T1 evaluation, adolescents completed a battery of self-report measures. The Appraisal of Parents subscale of the Conflict Behavior Questionnaire (CBQ; 11 items; Prinz et al., 1979) and the Parental Attitude Research Instrument (PARI; 6 items; Schaefer, 1965) were combined to create the parental support measures. The CBQ assesses adolescent-parent communication, conflict, and relationship quality, while the PARI assesses emotional support and affection. Paraphrased examples of questionnaire items include: “I enjoy talking with my mother/father; My mother/father and I don’t get along; My mother/father believes in showing me love; My mother/father makes me feel better when I am worried.” For all items, participants based their ratings on the past four weeks. Maternal and paternal support variables were derived by standardizing each item and computing the mean of the standardized item scores. A prorated scale score was computed by multiplying the mean of the standardized item scores by the number of items in the scale (maternal support range: −3.3 to 13.5; paternal support range: −4.3 to 10.8). Analyses indicated acceptable reliability for maternal (α = .76) and paternal support (α = .77).

2.4 Covariates

Several well-established risk factors for AUDs, as well as other demographics, were included as covariates in the analyses - gender, average parental education (proxy for socioeconomic status), number of individuals living in the household at T1, whether or not each parent was the adolescents’ biological mother/father (separate parent variables coded yes/no), adolescent birth order, adolescent lifetime diagnoses of MDD through T1 (yes/no), adolescent lifetime diagnosis of an externalizing disorder (ADHD, CD, ODD, or illicit substance abuse or dependence) through T1 (yes/no), and adolescent self-reported coping skills. Coping skills were included in the model to account for unexplained variance in our dependent variable and increase power, as prior research indicates that maladaptive coping techniques are associated with AUD onset (Berking et al., 2011; Hasking et al., 2011). The coping skills variable was derived by computing the prorated mean of items from the Self-Control Scale (Rosenbaum, 1980), modified Antidepressive Activity Questionnaire (Rippere, 1977; Parker and Brown, 1979), and Ways of Coping Questionnaire (Folkman and Lazarus, 1980). This composite variable had adequate reliability in the current sample (α =. 78). All covariates were assessed at T1 via adolescent self-report questionnaires or diagnostic interview.

2.5 Data Analysis Plan

To test our hypotheses, we used a series of Cox proportional hazards (PH) models. These models are similar to other regression techniques for dichotomous dependent variables (e.g., logistic regression) in that they allow for an examination of whether one or more variables predicts the likelihood of a specified outcome (Cox, 1972). PH models are superior to many of these other techniques, however, in that they allow for the timing of the event to be taken into account, not just whether or not the event occurred. In the current study, the time-to-event dependent variable was time (in years) since study enrollment (T1= 0 years) to the first onset of an AUD, or time of last assessment (i.e., total years enrolled in the study since the T1) for individuals that did not develop an AUD (i.e., censored observations). Separate PH models were used to examine the moderating effect of maternal and paternal support. Hazard ratios (HR) were used as estimates of effect size as they are considered indices of differences in hazards as a function of predictor variables.

A subset of adolescents did not have paternal support data (n = 70) and were therefore excluded from the paternal models. The proportional hazards assumption was tested by including a Time by Predictor interaction term in each Cox model (Singer and Willett, 1991). In all models, the interaction term was not significant (p's > .79) and thus, the proportional hazard assumption was met and the interaction term was removed. Analyses were conducted using SUDAAN 8.0.3 (Shah et al., 1997).

Variables were entered into the models in three steps: all 8 covariates were simultaneously entered in Step1, the main effects of T1 lifetime diagnosis of an anxiety disorder and maternal or paternal support were entered in Step 2, and the interaction term was entered in Step 3. The covariates were identical for both maternal and paternal support models; however, whether or not the adolescents' mother was the biological mother (yes/no) was included in the maternal support model, and whether or not the adolescents' father was the biological father was included in the paternal support model.

To test the effects of developmental period, we first coded individuals that developed an AUD as either early onset (i.e., prior to age 20 years) or late onset (i.e., after age 20 years). Twenty years was chosen as the cut-off as it was the average age of AUD onset within the current sample, and was consistent with prior studies on median AUD age of onset (Kessler et al., 2005). Next, two separate PH models (identical to those described above) were run including either: 1) individuals that had developed an AUD prior to age 20 years and individuals that had never developed an AUD (total N = 681) and 2) individuals that had developed an AUD after age 20 years and individuals that had never developed an AUD (total N = 682). Therefore, the first model excluded individuals that developed an AUD after age 20 whereas the second model excluded individuals that developed an AUD prior to age 20.

3. RESULTS

3.1 Participant Diagnoses and Characteristics

At T1, 80 (9.8%) participants had a lifetime diagnosis of an anxiety disorder with a mean age of onset of 7.4 years (SD = 3.4). After T1, 280 (34.3%) participants developed an AUD.

Of the 80 participants that had an anxiety disorder at T1, 28 (35%) developed an AUD during T2-T4. Average age of onset for an AUD was 20.8 years of age (*SD* = 3.2). Compared with adolescents without a lifetime history of anxiety disorders, participants with a lifetime diagnosis of an anxiety disorder at T1 reported greater levels of state anxiety ($t(786) = 2.70$, $p < 0.05$), worry ($t(783) = 3.25$, $p < 0.05$), and social anxiety ($t(815) = 2.82$, $p < 0.05$) at the T1 evaluation, and greater levels of worry ($t(803) = 2.91$, $p < 0.05$) and social anxiety ($t(803) = 1.91$, $p = 0.05$) at T2 (see Andrews et al., 1993 for detailed descriptions of self-report measures). This highlights that regardless of whether their anxiety diagnosis was current or past, adolescents with a lifetime diagnosis of anxiety disorders were more anxious than adolescents without an anxiety disorder diagnosis at T1 and T2. Individuals with anxiety disorders at T1 did not differ from individuals without anxiety disorders at T1 on maternal or paternal support (both *p's* > 0.42). There were no significant differences between ratings of maternal and paternal support at T1 ($p = 0.32$).

3.2 Anxiety Disorders by Maternal Support Interaction on AUD onset

Results are presented in Table 2. There were no significant main effects of anxiety disorders at T1 or maternal support. However, there was a significant anxiety disorders by maternal support interaction. To follow-up the interaction, maternal support was centered 1 *SD* above and below the mean to probe the simple slopes (Aiken and West, 1991; Holmbeck, 2002). Two *post-hoc* PH models were run incorporating all the aforementioned covariates, anxiety disorders at T1, the moderator, and the interaction term. At low maternal support, individuals with anxiety disorders were 65% more likely to develop an AUD than individuals without anxiety disorders (HR = 1.65, 95% CI = 1.21 – 2.26, $p < 0.01$). At high maternal support, individuals with anxiety disorders were 35% less likely to develop an AUD than individuals without anxiety disorders (HR = 0.74, 95% CI = 0.55 – 1.00, $p = 0.05$).

3.3 Anxiety Disorders by Paternal Support Interaction on AUD onset

Results indicated that there were no significant main effects for anxiety disorders at T1 or paternal support. There was also no significant anxiety disorders at T1 by paternal support interaction (see Table 3).

3.4 Early and Late Onset AUDs

Results from the early onset model indicated that there was an anxiety disorders at T1 by maternal support interaction (HR = 0.92, 95% CI = 0.87 – 0.97, $p < 0.01$). At low maternal support, anxiety disorders were associated with a 50% greater risk of AUD onset (HR = 1.50, 95% CI = 1.06 – 2.13, $p < 0.01$). At high maternal support, anxiety disorders at T1 were associated with a 39% reduced risk of AUD onset (HR = 0.72, 95% CI = 0.52 – 0.98, $p = 0.02$). Results from the late onset model also indicated an anxiety disorders at T1 by maternal support interaction (HR = 0.92, 95% CI = 0.87 – 0.98, $p = 0.01$). At low maternal support, anxiety disorders at T1 were associated with a 73% greater risk of AUD onset (HR = 1.73, 95% CI = 1.19 – 2.52, $p < 0.01$). At high maternal support, anxiety disorders at T1 were not associated with AUD onset (HR = 0.90, 95% CI = 0.64 – 1.25, $p = 0.52$). These effects are displayed in Figure 1.

We also examined the moderating effect of paternal support for early and late onset AUDs. There was no significant interaction for the early onset ($p = 0.13$) or late onset AUD model ($p = 0.19$).

3.5 Additional Post-hoc Analyses

To examine the effects of our covariates, we conducted two PH models with just the main effects and the interaction term (i.e., no covariates). Results were consistent with the aforementioned findings – the anxiety disorders by maternal support interaction was significant (HR = 0.91, 95% CI = 0.87 – 0.96, $p < 0.001$) and the anxiety disorders by paternal support interaction was not significant ($p = 0.17$). We also conducted an additional analysis (with covariates) with both maternal and paternal support included in the model. Results indicated that the anxiety disorders at T1 by maternal support interaction remained significant (HR = 0.92, 95% CI = 0.86 – 0.97, $p < 0.01$), and the pattern of results was unchanged.

4. DISCUSSION

The aim of the current study was to examine whether parental support moderates the association between childhood/adolescent anxiety disorders and AUDs, and whether these effects differ as a function of age of AUD onset. Results indicated that across adolescence and early adulthood there was a significant anxiety disorders at T1 by maternal support interaction. Specifically, at low maternal support, anxiety disorders were associated with an *increased* risk for AUD onset. At high levels of maternal support, anxiety disorders were associated with a *reduced* risk for AUD onset at a trend level.

Interestingly, this pattern of results differed as a function of age of AUD onset. For individuals that developed an AUD prior to age 20, relative to later in life, anxiety disorders were more robustly associated with a reduced risk for AUD onset at high maternal support. The opposite was true at low maternal support. Anxiety disorders increased the risk of AUDs across both developmental periods. Therefore, anxiety disorders functioned as both a risk and protective factor at high versus low levels of support. These findings help to clarify previous mixed findings in the literature and indicate that perceptions of maternal support across early development have an important influence on the likelihood of developing AUDs.

At low levels of maternal support, our findings suggest that anxiety disorders are a risk factor for the development of AUDs across adolescence and early adulthood. This is consistent with the broader internalizing disorder-substance use literature which posits that individuals with anxiety are motivated to engage in alcohol use to reduce or avoid their negative affect (Baker et al., 2004; O'Neil et al., 2011). Specifically, acute alcohol consumption is thought to bring perceived relief from negative affective states (Abrams et al., 2001; Thomas et al., 2003), thereby negatively reinforcing this behavior and increasing the likelihood of continued use (Kassel et al., 2000; Robinson et al., 2011).

Although negative reinforcement processes could apply to all individuals with anxiety disorders, the results suggest that those with perceived low maternal support may be

specifically vulnerable to AUD onset. It is possible that children and adolescents with low maternal support have less physical, emotional, and financial resources to cope with their distress, which makes them vulnerable to lifelong patterns of maladaptive coping. It is also possible that mothers that are perceived as providing low support engage in maladaptive parenting behaviors (and may even have anxiety disorders themselves) that impair the development of self-regulation capabilities in their offspring (Morris et al., 2007). This impairment may have persistent deleterious effects including increased vulnerability for alcohol use. Lastly, low maternal support may be associated with an overall aversive home environment which causes individuals to avoid home, spend more time with peers, and consequently, have more opportunities to initiate alcohol use (Duncan et al., 1994). High levels of anxiety coupled with early exposure to alcohol may ultimately propel individuals along the internalizing pathway to AUDs.

Our results indicate that at high maternal support, anxiety disorders decrease risk for developing AUDs. Notably, this protective effect is slightly weaker than the risk effect at low maternal support. Moreover, individuals with anxiety disorders were ‘protected’ from AUD onset at high maternal support to a greater extent prior to age 20. There are several potential mechanisms that may contribute to this finding. Most notably, children and adolescents with anxiety disorders often exhibit interpersonal deficits which could cause them to withdraw from peers and abstain from social activities (Chansky and Kendall, 1997; Clark et al., 1994). Since experimentation with alcohol frequently occurs in social settings in adolescence and early adulthood (Hussong, 2000) individuals with anxiety disorders may have fewer opportunities to experiment with alcohol and subsequently develop AUDs (Fite et al., 2006).

The findings suggest that the ‘protective power’ of anxiety may only apply to children and adolescents with perceived high maternal support. One possibility for this result is that those with a highly supportive mother may perceive their overall home environment as pleasant and be most reluctant to leave home and engage in social activities, including alcohol use. Alternatively, highly supportive mothers may also exhibit parenting behaviors that could be considered accommodating of the child’s anxiety symptoms which could implicitly or explicitly maintain anxiety symptoms and discourage adolescents to affiliate with peers. Importantly, past age 20, high maternal support no longer had a significant effect on AUDs. Given that after age 20, cohabitation with mothers is less frequent and alcohol is more easily accessible, perceived maternal support may no longer have an effect and these ‘protected’ (or overprotected) individuals may become vulnerable to AUD onset later in life.

Our findings indicated that perceived paternal support did not influence the association between anxiety disorders and AUDs. Research indicates that mothers often spend more time with their offspring than fathers during childhood through adolescence (Craig, 2006). Therefore, maternal support may be a more potent factor to individuals with anxiety disorders than paternal support. Second, evidence has suggested that mothers play more of a role in the emotional developmental of their offspring (Fivush et al., 2000; McDowell et al., 2002), and thus, may also play a larger role in onset of psychopathology. Third, of the individuals with anxiety disorders in the current study, approximately 58% were living with a step-father at T1. However, almost all individuals (91%) were living with their biological

mother. It is therefore possible that the current pattern of results is specific to biological parents and not step-parents. Therefore, future studies are needed to clarify the role of biological fathers' support.

Although these findings address important gaps in the literature, there are several limitations of note. First, the current sample of individuals with anxiety disorders at T1 was relatively small (9.8%) which likely reduced statistical power. Second, we did not include post-traumatic stress disorder (PTSD) or generalized anxiety disorder (GAD) in our baseline anxiety variable, as these disorders were not assessed at T1. It is important to note, however, that PTSD and GAD are both highly comorbid with other anxiety disorders (Kessler et al., 2005) and it is likely that some of these cases were accounted for. Third, our measure of parental support was broad and it is possible that different facets of support (e.g., affection, communication patterns) differentially moderate the association between anxiety disorders and AUD onset. Similarly, due to our small sample size, we were unable to adequately examine alcohol abuse and alcohol dependence as separate outcome variables. Of note, however, in the recent 5th edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM5; APA, 2013), both disorders were consolidated into one condition. Fourth, we did not directly examine peer factors in the current study and it is possible that peer support and/or delinquent peer affiliation impacts the current findings. This is therefore an important area of future research. Lastly, there were a subset of individuals in the present study that did not have parental support data and individuals with AUDs had slightly higher attrition rates than individuals without AUDs. This may have affected the generalizability of the current sample and underscores the need for replication.

With these limitations in mind, there are several important implications of these findings. Most notably, perceived maternal support has a significant impact on the anxiety disorders-AUD pathway, but that the magnitude and direction of effects differs as a function of level of support and developmental period. Alcohol use intervention efforts may therefore benefit by assessing perceived maternal support in adolescents with anxiety disorders. More specifically, among adolescents with anxiety disorders and perceived low maternal support, family-based cognitive behavioral therapy (Creswell and Cartwright-Hatton, 2007) may consider systematically targeting expressions of maternal support to mitigate risk of AUDs. In contrast, adolescents with anxiety disorders that report high maternal support may not be at an immediate risk for AUDs and thus, a focus may be on assessing how other vulnerability and risk processes could contribute to their development of AUDs over time.

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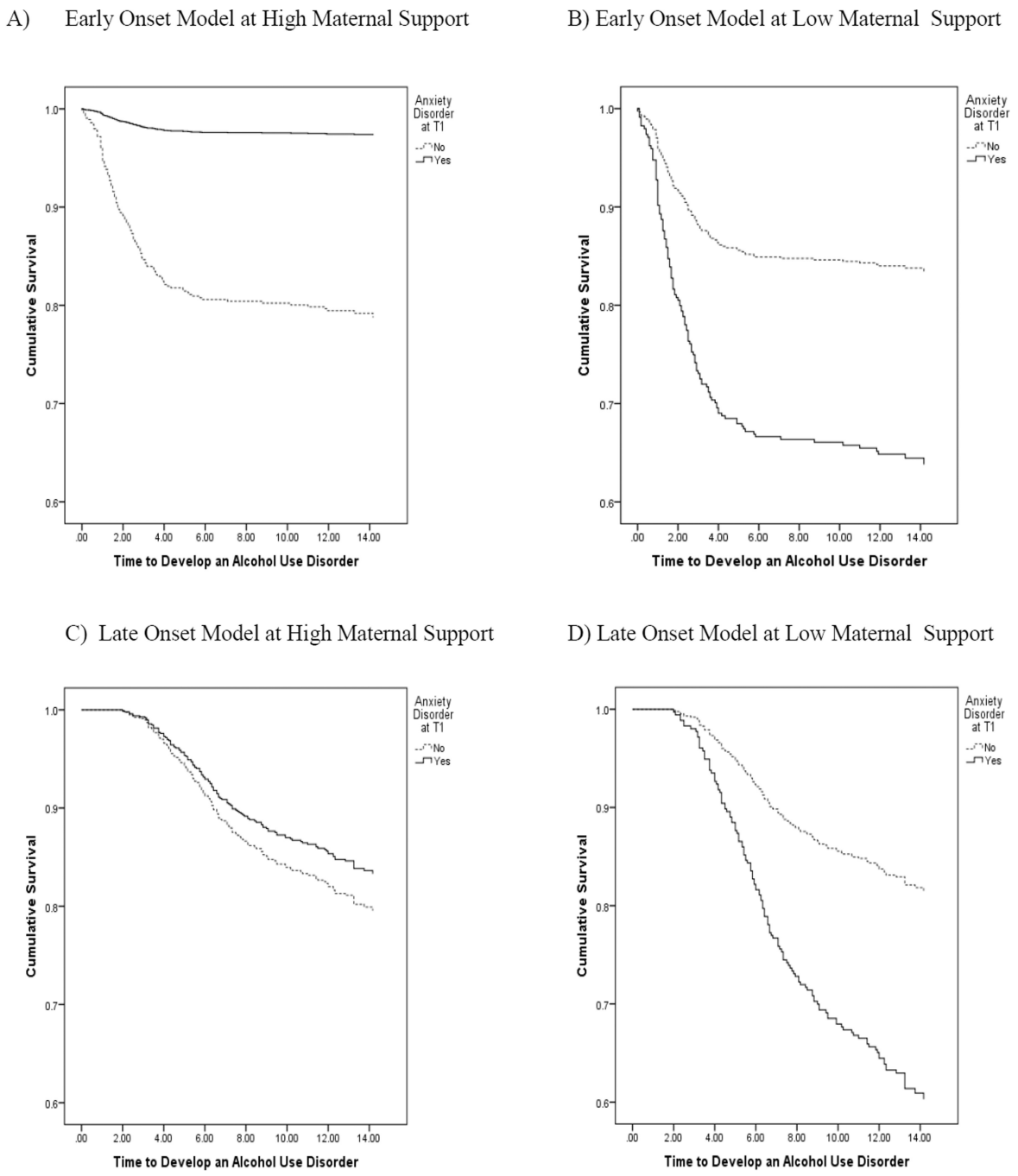

Figure 1.

Survival hazards plot of the relation between anxiety disorders at T1 and time to develop an alcohol use disorder, at high and low maternal support, for early onset and late onset AUDs.

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).

Table 2

Proportional hazards results examining unique and interactive effects of anxiety disorders at T1 and maternal support on time to develop an AUD.

Variable	HR	95% CI	*p*-value
Step 1			
Gender	1.11	0.95 – 1.29	0.17
Average Parent Education	1.01	0.95 – 1.08	0.73
No. of People in Household*	0.94	0.89 – 0.99	0.03
Mother's Status	0.97	0.76 – 1.24	0.81
Birth Order	1.01	0.95 – 1.08	0.66
T1 MDD*	1.23	1.04 – 1.45	0.02
TI Externalizing Disorder+	1.26	0.98 – 1.62	0.07
Coping Skills*	0.99	0.98 – 0.99	0.04
Step 2			
T1 Anxiety Disorders	1.07	0.87 – 1.33	0.51
Maternal Support	1.00	0.98 – 1.02	0.91
Step 3			
T1 Anxiety × Maternal Support**	0.91	0.86 – 0.96	< 0.00

Note.

** $p < 0.01$;

* $p < 0.05$;

+ $p < 0.10$;

MDD = major depressive disorder; Externalizing Disorder = attention deficit hyperactivity disorder, conduct disorder, oppositional defiant disorder, or illicit substance abuse or dependence; Mother's Status = whether or not the mother was the adolescent's biological mother.

Table 3

Proportional hazards results examining unique and interactive effects of anxiety disorders at T1 and paternal support on time to develop an AUD.

Variable	HR	95% CI	*p*-value
Step 1			
Gender	1.09	0.94 – 1.28	0.25
Average Parent Education	1.00	0.93 – 1.07	0.99
No. of People in Household^{+}	0.95	0.91 – 1.00	0.08
Father's Status	0.97	0.82 – 1.14	0.72
Birth Order	1.01	0.95 – 1.07	0.79
T1 MDD*	1.25	1.05 – 1.48	0.01
TI Externalizing Disorder^{+}	1.29	0.98 – 1.70	0.07
Coping Skills^{+}	0.99	0.98 – 1.00	0.09
Step 2			
T1 Anxiety Disorders	1.13	0.89 – 1.44	0.30
Paternal Support	0.99	0.98 – 1.02	0.78
Step 3			
T1 Anxiety × Paternal Support	0.96	0.92 – 1.02	0.18

Note.

$^{*}p < 0.05$;

$^{+}p < 0.10$;

MDD = major depressive disorder; Externalizing Disorder = attention deficit hyperactivity disorder, conduct disorder, oppositional defiant disorder, or illicit substance abuse or dependence; Father's Status = whether or not the mother was the adolescent's biological father.