Drug and Alcohol Dependence 164 (2016) 38–46

Contents lists available at ScienceDirect

Drug and Alcohol Dependence

journal homepage: www.elsevier.com/locate/drugalcdep

Full length article

Internalizing and externalizing disorders as predictors of alcohol use disorder onset during three developmental periods

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ARTICLE INFO

Article history:
Received 29 December 2015
Received in revised form 26 March 2016
Accepted 15 April 2016
Available online 23 April 2016

Keywords:
Alcohol use disorders (AUDs)
Predictors
Psychopathology
Externalizing
Internalizing
Gender

ABSTRACT

Background: The developmental pathways associated with an enhanced risk for future alcohol use disorders (AUDs) continue to be a topic of both interest and debate. In this research, internalizing and externalizing disorders were evaluated as prospective predictors of the index AUD episode onset, separately within three developmental periods: early-to-middle adolescence (age 13.0–17.9), late adolescence (18.0–20.9), and early adulthood (21.0–30.0).
Methods: Participants ($N = 816$) were initially randomly selected from nine high schools in western Oregon and subsequently interviewed on four separate occasions between ages 16 and 30, during which current and past AUDs were assessed as well as a full range of psychiatric disorders associated with internalizing and externalizing psychopathology domains.
Results: In adjusted analyses for each of the three developmental periods investigated, externalizing domain psychopathology from the most proximal adjoining developmental period predicted AUD onset. Distal externalizing psychopathology also predicted AUD onset among early adult onset cases. Proximal or distal internalizing psychopathology, in comparison, was not found to be a significant predictor of AUD onset in adjusted analyses for any of the developmental periods examined.
Conclusions: Findings overall suggest that externalizing developmental histories are robust predictors of AUD onset within the age range during which index episodes are most likely to occur, and that gender does not moderate this association.

1. Introduction

Contemporary epidemiological perspectives suggest that developmental pathways for alcohol use disorders (AUDs) are established well before problematic alcohol use begins (Clark, 2004), and likely causally related to processes that increase vulnerabilities to externalizing and internalizing psychiatric disorders (Hussong et al., 2011; Sher et al., 2005; Vanyukov et al., 2012). The internalizing-externalizing organizational model of psychopathology (Achenbach, 1966; Krueger, 1999) is a statistically-derived framework that accounts well for the covariation among psychiatric symptoms and disorders among children and adults in cross-sectional and longitudinal studies (e.g., Achenbach, 1966; Caspi et al., 2014; Farmer et al., 2013b; Kessler et al., 2011; Krueger and Markon, 2006), and has been suggested as a guiding framework for research on common causal pathways that account for lifetime disorder comorbidity (Kessler et al., 2011; Krueger, 1999), including AUDs (Hussong et al., 2011).

Externalizing disorders and their precursors are associated with oppositional, aggressive, impulsive, disruptive, and rule-breaking behavior. Prospective studies have documented that externalizing tendencies or disorders robustly predict the future onset of alcohol use problems or AUDs (Chassin et al., 2004; Elkins et al., 2006; Englund et al., 2008; Feingold et al., 2015; Fergusson et al., 2007; Grekin et al., 2006). Internalizing disorders and their precursors, in contrast, are associated with depression, anxiety, fear, rumination, and distress. Compared to externalizing developmental pathways, the role of internalizing pathways in the development of AUDs has been little researched (Hussong et al., 2011). Although internalizing symptoms or disorders are often concomitants with alcohol use

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http://dx.doi.org/10.1016/j.drugalcdep.2016.04.021
0376-8716/

problems or disorders in cross-sectional research (e.g., Burns and Teesson, 2002; Hasin et al., 2007; Kessler et al., 2005), prospective studies on the risk posed by internalizing tendencies or disorders on future alcohol use problems or AUDs have produced mixed findings (Boschloo et al., 2013; Buckner et al., 2008; Buckner and Turner, 2009; Crum and Pratt, 2001; Elkins et al., 2006; Gilman and Abraham, 2001; Grekin et al., 2006; Kushner et al., 1999; Trautmann et al., 2015; Zimmermann et al., 2003).

There are three primary aims associated with this study. First, this research evaluated whether risk for index AUD episodes within each of the developmental periods examined was associated with prior externalizing or internalizing disorders. Three developmental periods corresponding to AUD onset were defined: early-to-middle adolescence (age 13.0–17.9), late adolescence (18.0–20.9), and early adulthood (21.0–30.0). Outcomes from the analyses associated with this aim are expected to elucidate the relative contributions of externalizing and internalizing disorders in the prediction of AUD risk as well as the developmental specificity versus generality of these predictors. Second, for the two latter developmental periods studied, we evaluated whether distal externalizing and internalizing disorders from earlier developmental periods also predicted risk for AUDs. Findings from these analyses are expected to highlight whether risk factors that occurred later versus earlier in development are most relevant in the prediction of subsequent AUD risk. Third, important differences in the development and course of AUDs among men and women have been reported (Ammon et al., 2008; Kessler et al., 1994; Nolen-Hoeksema, 2004). Consequently, in the present research, we explored whether gender moderates observed effects.

This study builds on existing studies in the following ways. First, this research was conducted with a large representative community sample. Many earlier studies that have sought to identify externalizing or internalizing predictors of AUDs utilized high-risk, treatment, or convenience samples (e.g., university students). Second, it is often unclear from existing prospective studies if risk-based evaluations of internalizing and externalizing disorders are referenced to the first or subsequent episode of problematic drinking or an AUD. This is an especially important consideration when evaluating risk factors for versus consequences of AUDs, as AUDs are known to increase risk for future internalizing disorders (Fergusson et al., 2011) as well as externalizing disorders, especially other substance use disorders (Kandel et al., 1992). Third, rather than limiting prediction models to single diagnostic categories, externalizing and internalizing disorder domains in the present research are represented by several individual disorders, thus resulting in broader coverage of content domains associated with risk-related pathways. Fourth, studies that have examined internalizing tendencies or disorders as risk factors for future AUDs often do not statistically control for externalizing features or disorders, and vice versa (Hussong et al., 2011). In the present research, we evaluated adjusted prediction models that controlled for demographic and psychosocial variables as well as the disorder domain that was not the primary predictor variable modeled in the analysis.

2. Materials and methods

2.1. Participants

2.1.1. Participant sampling, composition, and retention. At the first wave of data collection (T_1; ~age 16), the sample consisted of 1709 adolescents randomly selected from 9 high schools that were representative of urban and rural districts in western Oregon. About one year later (T_2), 1507 (88%) of these persons were reassessed. At T_3 (~age 24), a sampling stratification procedure was introduced whereby eligible participants included all non-white participants to enhance ethnic diversity and all persons with a positive history of a psychiatric diagnosis by T_2 ($n = 644$) and a randomly selected subset of participants with no history of a psychiatric or substance use disorder by T_2 ($n = 457$ of 863 persons). Of these 1101 eligible persons, 941 (85%) completed T_3. The T_4 assessment period was conducted approximately 6 years after T_3 (~age 30). From the 941 eligible persons who completed T_3, 816 (87%) completed the T_4 assessment. Earlier analyses of participant attrition (Farmer et al., 2013a; Lewinsohn et al., 1993; Rohde et al., 2007) revealed minimal sample bias related to study discontinuation.

By age 30.0, 34.3% of the weighted T_4 panel had a *DSM-III-R* or *DSM-IV* lifetime AUD diagnosis (43.0% male, 95% confidence interval [CI_{95}] = 37.4–48.5; 27.6% female, CI_{95} = 23.4–31.8; $p < 0.05$). For those with a lifetime AUD episode by age 30.0, the mean age at time of first AUD onset was 20.2 years of age ($SD = 3.9$).

2.1.2. Weighting procedures based on stratification implemented at T3. As a result of the unequal stratified sampling strategy implemented at T_3, Caucasian participants without a psychiatric diagnosis by T_2 were under-sampled at T_3 and T_4. To adjust for this sampling procedure, Caucasian participants with no lifetime diagnosis by T_2 were assigned a weight that reflected the probability of this subgroup being sampled during T_3 and T_4 assessments (see Farmer et al., 2013a for details). All findings subsequently presented (e.g., rates, ratios) were based on weighted data, with references to the numbers of cases based on unweighted data.

2.1.3. Reference sample for this research. The reference sample for this study varied according to the research question. Demographic predictors of AUD onset were evaluated using data from the complete T_4 panel ($n = 816$). Psychiatric predictors of early-to-middle adolescent AUD onset (between ages 13.0 and 17.9) were evaluated using data from participants without an incidence of AUD between ages 0 and 12.9 ($n = 810$), with AUD episodes occurring at or after age 18.0 right-censored.[1,2] Psychiatric predictors of late adolescent AUD onset (between ages 18.0 and 20.9) were evaluated with data from participants without an incidence of AUD before age 18.0 ($n = 730$), with AUD episodes occurring at or after 21.0 right-censored. Finally, psychiatric predictors of early adult AUD onset (between ages 21.0 and 30.0) were evaluated with data from participants without an incidence of AUD before age 21.0 ($n = 641$).

2.2. Diagnostic assessments

2.2.1. Definitions of internalizing and externalizing disorder domains and subdomains. When evaluating predictors of AUD onset, internalizing and externalizing disorder domain scores were categorically modeled, whereby a value of 0 was assigned if no disorder associated with a given domain was diagnosed within the timeframe specified and a value of 1 assigned if one or more domain-related disorders was diagnosed at any time within the indicated timeframe. Based on our earlier research with the OADP sample (Farmer et al., 2009, 2013b; Seeley et al., 2011), *DSM-*

[1] Earlier studies with community samples demonstrated that initial AUD onsets before age 13 are rare (Cohen et al., 1993; Clark, 2004). To evaluate the prospective associations between childhood disorders and later AUD onset, 6 cases with initial AUD onsets prior to age 13 (2% of participants with a lifetime AUD) were excluded from analyses involving predictors of onset (i.e., analyses presented in Tables 2–4). These participants, however, were included in analyses of demographic factors related to AUD onset (Table 1).

[2] Right-censoring here refers to an exclusion from consideration any index AUD episode onsets that emerged after the cessation of the developmental period that was the primary focus of the analysis. These cases did not have an observed AUD onset time within the developmental period examined, and were right-censored based on the last known observation time.