Drug and Alcohol Dependence 147 (2015) 235–242

Contents lists available at ScienceDirect

Drug and Alcohol Dependence

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

Family history density predicts long term substance use outcomes in an adolescent treatment sample

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ARTICLE INFO

Article history:
Received 23 May 2014
Received in revised form
12 November 2014
Accepted 13 November 2014
Available online 26 November 2014

Keywords:
Family history
Alcohol and drug dependence
Treatment outcome

ABSTRACT

Aims: This study explored whether the density of family history (FH) of substance use disorders relates to post-treatment substance use outcomes in adolescents, with the primary aim of determining whether FH exerts a relatively stronger influence on longer-term outcomes.
Method: The present investigation examined adolescents (ages 12–18, $n = 366$) from two independent samples who were treated for alcohol/substance use disorder (ASUD) and re-assessed during the eight years following treatment with identical methodology. Primary substance use outcomes were assessed at 1, 2, 4, 6, and 8 years post-treatment and included total drinks, days using marijuana, and days using other drugs.
Results: In hierarchical linear models there were significant FH density × linear time interactions for total drinks ($z = 12.75$, $p < 0.001$) and marijuana use days ($z = 4.39$, $p < 0.001$); greater FH density predicted more total drinks and more marijuana use days, with both associations becoming stronger over time. The increasing linkage between FH and other drug use was not significant over time.
Conclusions: Findings are consistent with previous research indicating that the risk associated with FH increases over time, especially in relation to quantity/frequency measures of alcohol and marijuana use. By extending these findings to an adolescent clinical sample, the current study highlights that FH density of alcohol and drug dependence is a risk factor for poorer long-term outcomes for adolescent-onset ASUD youth as they transition into adulthood. Future work should explore the mechanisms underlying greater post-treatment substance use for adolescents/young adults with greater FH density.

1. Introduction

The development and long-term progression of alcohol and substance use disorders (ASUDs) is a complex process influenced by both biological and environmental variables. The specific contribution of risk conferred by genetic factors and environmental variables is complex (Liu et al., 2004; Prescott and Kendler, 1999), with effects varying over the course of development. In genetically-informative studies of substance use etiology, the role of genetic factors appear to increase over the course of development, while shared environmental factors diminish in importance (Koopmans et al., 1997; White et al., 2003; Viken et al., 1999). More specifically, initiation and early substance use patterns seem to be more strongly influenced by social and familial environments, with progression to more severe levels of use under relatively greater genetic influence (Kendler et al., 2008).

1.1. Family history as a risk factor

While there are many pathways toward developing ASUDs, one long-recognized common risk is a positive family history (FH), such as biological parent alcoholism (Cadoret et al., 1995; Heath et al., 1991; McGue, 1997). Approximately 40–60% of the variance

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http://dx.doi.org/10.1016/j.drugalcdep.2014.11.009
0376-8716/

in alcohol use disorders may be explained by genetic influences (Heath et al., 1997; McGue, 1999; Prescott and Kendler, 1999), and estimates of genetic heritability may be up to 60%-80% for nicotine and cocaine (Kendler and Prescott, 1998; True et al., 1999).

1.2. Long-term influences of family history

Although FH is a commonly employed clinical indicator of genetic risk, its influence also includes environmental and social influences. Several mechanisms may underlay the means whereby risk conferred by FH changes over time. Research to date suggests FH may have a long-term influence on substance use severity and problems, including a more severe course and higher rates of ASUDs at long-term follow-up time points (Dawson et al., 1992; Chassin et al., 2004; Cloninger et al., 1981; Grant, 1998; Worobec et al., 1990). For instance, in community samples, individuals with positive FH consumed greater maximum drinks, met more dependence criteria, and had higher rates of marijuana use at an 8-year follow-up in one investigation (Schuckit and Smith, 1996).

FH impact may become stronger over time through cumulative effects of genetic and environmental components of risk (Jackson et al., 2000; Chassin et al., 2002). These longer-term effects of FH are potentially partially mediated by lower subjective response to alcohol and subsequent consumption of greater quantities of drinks (e.g., Quinn and Fromme, 2011; Schuckit, 2002; Schuckit et al., 2004). Taken together, prior studies suggest positive FH should relate more strongly to long-term measures of substance use, but few studies have utilized frequent, repeated measures of substance use outcomes to closely examine whether the influence of FH on these outcomes changes over time.

1.3. The impact of family history on adolescents

The majority of studies examining the effects of FH on ASUDs have utilized community samples recruited according to FH status and followed over time. Few studies have examined the long-term effects of FH on those youth diagnosed and treated for ASUDs in adolescence. Late adolescence and young adulthood (i.e., age 18–22) is the highest risk developmental period for onset of alcohol and substance use related disorders (Johnston et al., 2011; Substance Abuse and Mental Health Services Administration, 2009). Thus, this period may be particularly impactful for those with a history of early ASUDs. Given that the influence of genetic factors on substance use may increase over time (Kendler et al., 2008; Koopmans et al., 1997; White et al., 2003; Viken et al., 1999) and FH is thought to capture both genetic and environmental aspects of addiction that exert influence on outcomes once use has been initiated (e.g., Chassin et al., 2002; Jackson et al., 2000; Schuckit and Smith, 1996), the influence of FH could be more pronounced among those youth in substance use treatment.

1.4. Family history density

A comprehensive measure of FH computed according to the combination of familial relatedness and ASUD history (i.e. FH density score) may be the most comprehensive measure of FH (Dawson et al., 1992; Harford et al., 1992; Schuckit and Smith, 1996; Stoltenberg et al., 1998). The FH density score considers the contribution of first- and second-degree relatives and has been considered a more appropriate clinical measure of biological risk than a single dichotomous variable (Zucker et al., 1994). Furthermore, a density score accounts for more variance in alcoholism severity and consequences of drinking when compared to a dichotomous FH score of first- and second-degree relatives (Turner et al., 1993), supporting the utility of FH density as a clinically-based measure of potential genetic risk for ASUDs.

1.5. Study aims

The primary aims of the present study were to examine whether FH density predicts post-treatment substance use outcomes in youth diagnosed with and treated for ASUDs in adolescence, and to explore whether FH density exerts relatively stronger influence on longer-term post-treatment outcomes. This is a qualitatively different group than examined in prior community sample studies. Although prior literature has indicated FH impacts drinking outcomes, there are gaps in the literature regarding (1) the differential effects of FH density on short- compared to long-term outcomes and (2) its impact on a high-risk group of treatment-seeking adolescents. We hypothesized that greater FH density would predict greater levels of alcohol, marijuana, and other drug use, and that the effects of FH density would be relatively stronger for longer-term compared to shorter-term outcomes. To examine the independent effects of FH density, we adjusted for other influences on adolescent treatment outcomes, which are associated with FH of ASUD. Specifically, conduct disorder, a risk factor previously associated with both ASUD, FH, and long-term substance use outcomes (Chassin et al., 1999; Chung et al., 2003; Sher, 1991; Zucker et al., 1994), and time-varying levels of depression and anxiety, were covaried to determine whether FH density was independently associated with adolescent ASUD treatment outcomes above and beyond the effects of these common prognostic indicators.

2. Methods

2.1. Participants

The present research was conducted according to the guidelines and under the approval of the University of California, San Diego Human Research Protections Program. The current sample ($n = 366$) included youth selected from two previous studies of long-term alcohol/substance use treatment outcomes for adolescents (ages 12–18 at baseline), who were recruited at the onset of inpatient stays at alcohol and substance use treatment facilities in the San Diego area. The six treatment facilities were abstinence-focused and used a 12-step model of alcohol/substance abuse treatment as well as individual, family, and group psychotherapies drawing from cognitive-behavioral strategies. Length of inpatient treatment ranged from 5 days to 3 weeks. All participants in both studies met Diagnostic and Statistical Manual of Mental Disorders (DSM-III-R; American Psychiatric Association, 1987) criteria for alcohol and/or substance dependence. In the full combined sample utilized in the current study, 159 participants (43.4%) were adolescents who met no DSM-III-R Axis I disorders exclusive of conduct disorder (hereafter referred to as the ASUD-only group). The rest of the sample ($n = 207$, 56.6%) met criteria for an alcohol and/or substance use disorder and an additional non-conduct, DSM-III-R Axis I disorder (Comorbidity group). Axis I disorders were assessed with the Diagnostic Interview Schedule for Children—Computerized Version (DISC—III-R; McCarthy et al., 2005; Piancentini et al., 1993; Ramo et al., 2005; Tomlinson et al., 2004) administered separately with the adolescent and a collateral reporter (parent or custodial guardian). Additional eligibility criteria (across both studies) included 12–18 years of age, residence within 50 miles of the research site, participants' literate in English, and availability for one-year follow-up. Youth who did not have a collateral reporter (i.e., parent or guardian) to corroborate personal and FH information, had current psychotic symptoms, or had physical handicaps prohibiting participation were excluded from the study.