Psychiatric Morbidity is Linked to Problem Drinking in Midlife Among Alcohol-Dependent Men: A Co-Twin Control Study

DANIEL M. BLONIGEN, PH.D.,^{a,*} THOMAS BURROUGHS, M.A.,^b JON RANDOLPH HABER, PH.D.,^c
AND THEODORE JACOB, PH.D.^c

^aCenter for Health Care Evaluation, Veterans Affairs Palo Alto Health Care System, Menlo Park, California

^bPalo Alto University, Palo Alto, California

^cFamily Research Center, Veterans Affairs Palo Alto Health Care System, Palo Alto, California

ABSTRACT. Objective: Prior research on predictors of problem drinking has been limited because of an inability to attribute an unambiguous environmental explanation to observed findings. Using a prospective co-twin control design, we examined the extent to which a history of psychiatric symptoms exerts an environmental influence on problem drinking in midlife that is unconfounded by genetic underpinnings. **Method:** Participants were 367 complete male twin pairs (208 monozygotic, 159 dizygotic) from the Vietnam Era Twin Registry who were assessed in midlife as part of the Family Twin Study ($M_{\text{age}} = 51.4$ years, $SD = 2.8$). Twin pairs who were concordant for a lifetime diagnosis of an alcohol use disorder (AUD) in 1992 were selected for participation and were reinterviewed in 2001 to measure symptoms of AUD (i.e., problem

drinking) since the prior assessment (past 10 years). **Results:** Within-pair differences in lifetime symptom counts of several psychiatric disorders measured in 1992 (i.e., major depression, dysthymia, generalized anxiety disorder, panic disorder, antisocial personality, mania, and posttraumatic stress disorder) were significantly associated with within-pair differences in AUD symptoms in the subsequent 10 years. **Conclusions:** A history of psychiatric problems, particularly one marked by internalizing symptoms, appears to be linked to problem drinking in midlife above and beyond the confounding influence of genetic effects and underscores the potential value of integrated interventions for comorbidity to address problem drinking among individuals during this period of the life course. (*J. Stud. Alcohol Drugs*, 74, 136–140, 2013)

SCHOLARS HAVE LONG SOUGHT to identify predictors of the course of alcohol use disorders (AUDs) across the life span (Finney and Moos, 1992; Sher et al., 2005; Zucker et al., 2006). A crucial limitation of this literature is the inability of most research designs to attribute an unambiguous environmental explanation to observed findings, given that some ostensible sources of environmental influence may be genetic in nature (Plomin and Bergeman, 1991). Another limitation in this literature is that most investigations have focused on the course of AUD from adolescence to young adulthood and neglected to look beyond the third decade of life. This is an important issue, given that mid-late life (i.e., ages 40–60) is marked by continued problem drinking among many young-adult problem drinkers, and it is estimated that 1.4 million individuals in mid-late life will have an AUD diagnosis by 2030 (Molander et al., 2010). Using a sample of twin pairs concordant for a lifetime diagnosis of

AUD but discordant on drinking characteristics at midlife, we sought to identify an environmental link between psychiatric symptoms and problem drinking that is unconfounded by genetic underpinnings.

Untangling genetic and environmental influences in the prediction of problem drinking in midlife

Genetic influences account for significant variation for virtually all behavioral outcomes (Turkheimer and Waldron, 2000), but such influences cannot be modeled without being measured. Modeling these effects is particularly important in the area of evidence-based treatment where the implicit assumption is that targets of clinical interventions are environmental in nature. However, this “environmental assumption” may not be entirely true when genetic effects account for substantial portions of variance. Twin designs are uniquely suited to test the environmental assumption. In particular, co-twin control designs allow for identification of environmental influences that contribute to the course of AUD but are unconfounded by genetic effects (Eisen et al., 1991; Grant et al., 2010; Scherrer et al., 1996). Specifically, if twins are found to differ in regard to both a predictor variable and a drinking outcome, then one would have strong evidence that nonshared environmental influences play a causal role in accounting for the drinking outcome.

In the present study, we selected complete twin pairs from

Received: July 18, 2012. Revision: September 19, 2012.

This research was supported by the Veterans Administration Merit Award and National Institute on Alcohol Abuse and Alcoholism Grant R01-AA016402 (to Theodore Jacob). Daniel M. Blonigen was supported by a Career Development Award-2 from the Veterans Affairs Office of Research and Development (Clinical Sciences Research & Development).

*Correspondence may be addressed to Daniel M. Blonigen at the Center for Health Care Evaluation, Veterans Affairs Palo Alto Health Care System, 795 Willow Road (152-MPD), Menlo Park, CA 94025, or via email at: dmbлонigen@gmail.com.

the Vietnam Era Twin Registry (VETR; Eisen et al., 1987) with a lifetime diagnosis of an AUD and tested whether differences within twin pairs on problem drinking in midlife (defined as number of AUD symptoms) were associated with differences within twin pairs in psychiatric symptoms. A history of psychiatric disorders such as internalizing (mood and anxiety disorders—e.g., Burns and Teesson, 2002) and externalizing (drug use disorders, antisocial personality disorder; Dick et al., 2007), mania (Goodwin and Jamison, 2007), and posttraumatic stress disorder (Bremner et al., 1996) are associated with a chronic course of problem drinking among individuals with AUD (Finney and Moos, 1992; Sher et al., 2005; Zucker et al., 2006).

Furthermore, prior twin research, including prospective studies (e.g., Edwards et al., 2011; Hicks et al., 2007), has found that genetic effects contribute to significant variation in these disorders as well as covariation with AUDs (Krueger et al., 2002; Pickens et al., 1995; Todorov et al., 2006). Thus, there is a need to control for genetic effects when examining associations between psychiatric morbidity and problem drinking among those with an AUD history.

To our knowledge, the co-twin control design has not been used previously to identify factors that differentiate the course of problem drinking in midlife. We hypothesized that, above and beyond the influence of genetic effects, psychiatric symptoms in the domains of internalizing and externalizing would exhibit significant associations with AUD symptoms in midlife. Given the complexity of issues under study, however, it is recognized that co-twin control findings alone are insufficient to demonstrate clearly a genetic or nongenetic interpretation in observed associations.

Method

Participants

Participants were drawn from a subsample of the VETR who participated in both the Harvard Drug Study (HDS; Tsuang, 1996) and the Family Twin Study (FTS; Jacob et al., 2003). The VETR was designed as an epidemiological study such that findings regarding the course of substance use should be comparable to the general population. Supportive of this, sociodemographics and rates of AUDs are comparable across the VETR and various epidemiological studies (e.g., National Epidemiological Survey of Alcoholism). Eligibility for the VETR required both members of a twin pair to have served in active military duty during the Vietnam era (from May 1965 to August 1975).

In 1992, a telephone assessment was administered to twins from the VETR to gather information about lifetime psychiatric diagnoses as part of the HDS. A subsample of the HDS was contacted in 2001 and was asked to complete an assessment on substance use history as part of the FTS. Data collection was performed by trained interviewers from

the Institute for Survey Research using the computer-assisted telephone interview, which allowed for standardization of interview items/prompts and automatic data entry. Procedures for the HDS and FTS were approved by the local institutional review board.

The FTS comprises 1,774 individual twins who were selected from the HDS sample. Eligible cases were identified based on a positive AUD status in one member of a twin pair as assessed in the 1992 HDS. Eighty-three percent of identified cases consented to participate. This response rate yielded a sample that was comparable to other VETR studies in terms of sociodemographics. Of the 1,774 FTS participants, 1,225 received a lifetime diagnosis of an AUD at the 1992 HDS assessment (i.e., alcohol abuse or dependence, *Diagnostic and Statistical Manual of Mental Disorders, Third Edition, Revised* [DSM-III-R]; American Psychiatric Association, 1987). This sample consisted of 367 complete twin pairs who were concordant for a history of AUD (208 monozygotic [MZ], 159 dizygotic [DZ]; $N = 734$) and is the sample reported on in the current study ($M_{\text{age}} = 51.4$ years, $SD = 2.8$).

Measures

Psychiatric symptoms. During the 1992 HDS, lifetime symptoms for several DSM-III-R psychiatric disorders (i.e., major depressive disorder, dysthymia, generalized anxiety disorder, panic disorder, antisocial personality disorder, drug dependence [one or more of the following: marijuana, cocaine, opioids, phencyclidine/lysergic acid diethylamide, sedatives, amphetamines], mania, and posttraumatic stress disorder) were assessed with the Diagnostic Interview Schedule (Robins et al., 1981). Internalizing and externalizing composites were constructed to represent the average of standardized (z) scores for symptom counts of major depressive disorder, dysthymia, generalized anxiety disorder, and panic disorder (internalizing) and antisocial personality disorder and drug dependence (externalizing). Diagnostic frequencies and means/SDs for number of lifetime symptoms endorsed were as follows: major depressive disorder (14.7%; $M = 2.89$, $SD = 2.73$), dysthymia (4.6%; $M = 1.68$, $SD = 1.76$), generalized anxiety disorder (4.2%; $M = 2.28$, $SD = 4.42$), panic disorder (3.4%; $M = 0.89$, $SD = 2.54$), antisocial personality disorder (7.9%; $M = 2.99$, $SD = 2.60$), drug dependence (31.9%; $M = 1.87$, $SD = 2.48$), mania (1.5%; $M = 1.40$, $SD = 1.83$), and posttraumatic stress disorder (14.7%; $M = 3.58$, $SD = 4.79$). The frequencies of having any internalizing and externalizing disorder were 18.9% and 34.9%, respectively.

Alcohol use disorder symptoms. The Lifetime Drinking History (LDH)—a structured interview (Skinner and Sheu, 1982) modified by Jacob (1998)—was administered during the FTS in 2001 to obtain information on participants' lifetime drinking patterns and was used in this study to obtain

participants' maximum number of AUD symptoms during any given year in the time since the HDS assessment (10 years). The LDH uses retrospective questioning to identify distinct phases of drinking across the lifetime. The first phase begins at the age when an individual started drinking regularly (i.e., at least one drink per month). The end of this phase and the beginning of the next phase is defined by a significant change in the frequency or quantity of alcohol consumption. Interviewers direct respondents to consider life changes and their associated drinking patterns to assist with recall. Participants could report up to 11 distinct drinking phases ending with their current age. On average, FTS participants reported 4.1 phases ($SD = 1.4$) in their lifetime ($M_{\text{phase length}} = 7.9$ years, $SD = 4.1$). The LDH is a reliable and valid instrument for assessing drinking patterns over the life span (Gladysjo et al., 1992; Jacob et al., 2006; Koenig et al., 2009). For example, Jacob et al. (2006) reported test-retest reliability over 5 years to be between .57 and .71, and Koenig et al. (2009) reported correlations between .57 and .69 for quantity/frequency measures reported prospectively and retrospectively over 13 years.

Analyses

We conducted co-twin control analyses by calculating within-pair difference scores on each predictor and computing correlations with within-pair difference scores on the maximum number of AUD symptoms reported for any given year over 10 years, measured with the LDH from 1992 to 2001. This approach controls for genetic effects, given differences in genetic relatedness between members of a pair (McCaffery et al., 2001). Because MZ twins share all of their genes, co-twin differences must be attributable to nongenetic influences. Members of DZ twin pairs share 50% of their genes, on average; therefore, co-twin differences provide less stringent control of genetics. However, in separate regression models predicting AUD symptom-count variable, all interactions between the psychiatric symptom-count variables and zygosity were nonsignificant. Therefore, to address the loss of power when using only MZ twins, within-pair difference score correlations were conducted with the combined MZ + DZ sample. For the sake of completeness, results for the MZ subsample also are provided. For each analysis, the psychiatric predictor variables were tested individually, and the number of AUD symptoms reported in the past year in the 1992 HDS was controlled for.

Results

Co-twin control analyses of psychiatric symptoms and alcohol use disorder symptoms

Table 1 provides correlations of within-pair difference scores between the psychiatric symptom counts and AUD

TABLE 1. Correlations between within-pair difference scores for psychiatric symptom counts (lifetime) and AUD symptom counts over 10 years

Within-pair difference scores	Within-pair difference scores (AUD symptoms)	
	MZ + DZ	MZ
Major depressive disorder	.19***	.15*
Dysthymia	.17***	.14*
Generalized anxiety disorder	.16**	.14*
Panic disorder	.16**	.05
Antisocial personality disorder	.14**	.09
Drug dependence	.09	.08
Mania	.15**	.13
Posttraumatic stress disorder	.12*	.04
Internalizing composite	.22***	.16*
Externalizing composite	.15**	.11

Notes: AUD = alcohol use disorder; MZ = monozygotic; DZ = dizygotic; $N_{\text{Twin Pairs (MZ)}} = 208$; $N_{\text{Twin Pairs (DZ)}} = 159$; psychiatric symptom counts were measured in the 1992 Harvard Drug Study (HDS). AUD symptom counts were measured in the 2001 Family Twin Study. Each parameter estimate represents partial correlations, which controlled for past-year AUD symptoms in the 1992 HDS.

* $p < .05$; ** $p < .01$; *** $p < .001$.

symptom counts. These represent partial correlations, which controlled for past-year AUD symptom counts in the 1992 HDS. In the combined sample, significant difference score correlations emerged for symptom counts of all the internalizing disorders (major depression, dysthymia, generalized anxiety disorder, and panic disorder), the internalizing composite, antisocial personality disorder, the externalizing composite, mania, and posttraumatic stress disorder. For the MZ subsample, these correlations remained significant only for major depression, dysthymia, generalized anxiety disorder, and the internalizing composite. However, comparing the magnitude of the correlations for the combined sample versus the MZ subsample (where only the former was statistically significant) indicates that these correlations were not significantly different from one another for most of the assessed variables.

Discussion

Prior research has identified psychiatric disorders as significant predictors of problem drinking (Burns and Teesson, 2002; Finney and Moos, 1992); however, this work has been unable to attribute an unambiguous environmental explanation to observed findings. Our co-twin control analyses revealed that links between psychiatric morbidity and problem drinking in midlife among individuals with a history of AUD do not appear to be confounded by genetic effects. To our knowledge, this represents the first co-twin control study of factors that differentiate the course of problem drinking in midlife. Moreover, by identifying an unambiguous environmental explanation to a risk factor of AUD, this study builds on our program research, which has focused on the course of AUD from drinking onset to midlife (Jacob et al., 2005, 2009, 2010, 2012). Although these findings alone are

insufficient to demonstrate a clear genetic or environmental interpretation regarding correlates of problem drinking, they highlight the potential value of integrated interventions for comorbidity to address problem drinking among individuals in midlife (Drake et al., 2008; Torrens et al., 2012). Specifically, these findings suggest that co-occurring psychiatric symptoms may exert a direct effect on the risk of problem drinking in midlife that is not mediated by shared genetic risk factors. Further, these findings encourage future research to elucidate the components of integrated treatments that are most beneficial for the treatment of problem drinking in midlife (e.g., coping skills training; medication management).

Among the psychiatric variables we examined, symptoms of internalizing disorders exhibited the most consistent and robust link with problem drinking in midlife after genetic influences were accounted for, as evidenced by the significant correlations for these disorders in both the combined sample and the MZ subsample. (Given that the correlations in the combined vs. MZ subsamples were comparable in magnitude for the other disorders, the nonsignificant associations in the MZ subsample may have resulted from the loss of power in this smaller subsample.) Although prior research has identified genetic contributions to the covariance between AUDs and several mental health disorders, including both internalizing and externalizing syndromes (Fu et al., 2002; Pickens et al., 1995), this literature also has reported that nonshared environmental effects make independent contributions to variance in these outcomes (Turkheimer and Waldron, 2000). It is those nonshared environmental influences that are specifically identified with the co-twin control design.

Notably, that the psychiatric symptoms were assessed before the problem drinking outcome supports a causal interpretation of these findings. That is, a history of psychiatric symptomatology may be linked to an increased risk of problem drinking among individuals in midlife above and beyond any shared genetic overlap between these disorders. This possibility, however, must be tempered with acknowledgment of the fact that the assessment of AUD symptoms was retrospective in the present study. Nonetheless, the findings highlight the importance of AUD interventions to monitor and address co-occurring psychiatric disorders for individuals in midlife. Such an integrated treatment approach has received increasing recognition in the substance use disorder treatment community (Drake et al., 2008; Torrens et al., 2012). Moreover, prior research has suggested that for dual-diagnosis patients (i.e., individuals with co-occurring substance use and psychiatric disorders), enhancement of general, as well as substance-specific, coping skills is needed to improve long-term functioning (Moggi et al., 1999).

Some limitations of the present work must be noted. First, although the VETR was designed as an epidemiological study, it remains unclear whether the current findings will generalize to a nonveteran sample or to women, given that

estimates of genetic and nongenetic variance are sample-specific. Second, assessment of problem drinking via the LDH interview was based on retrospective recall. Prospective assessment is recommended in future endeavors. However, as noted earlier, the extensive validation of the LDH as a measure of problem drinking history lends credence to the present findings (Gladsjo et al., 1992; Jacob et al., 2006; Koenig et al., 2009). Third, the assessment of psychiatric symptoms in 1992 was in reference to lifetime symptom counts. Hence, we were not able to determine whether any of these symptoms persisted in the 10-year period between the HDS and FTS assessments. Therefore we cannot infer a direct effect of psychiatric morbidity on the course of problem drinking in midlife. Nevertheless, the fact that a history of psychiatric symptoms measured 10 years earlier could exert an environmental influence on the course of problem drinking is notable and encourages further research to tease apart the interrelations between these constructs during the midlife years. Fourth, although the Diagnostic Interview Schedule has been validated as a psychiatric interview (Robins et al., 1981), interviews that were designed specifically to assess comorbid psychiatric disorders among substance users may be recommended in future work (see Hasin et al., 2006).

In summary, the present study represents the first co-twin control study to examine the link between psychiatric morbidity and problem drinking in midlife. These findings underscore the value and potential of integrated treatment approaches to address comorbid substance abuse and dependence and mental health problems in existing or future evidence-based treatments for AUDs.

Acknowledgments

The United States Department of Veterans Affairs has provided financial support for the development and maintenance of the VETR. Numerous organizations have provided invaluable assistance, including the VA Cooperative Study Program; the Department of Defense; the National Personnel Records Center, National Archives and Records Administration; the Internal Revenue Service; the National Institutes of Health; the National Opinion Research Center; the National Research Council, National Academy of Sciences; and the Institute for Survey Research, Temple University. Most importantly, the authors gratefully acknowledge the continued cooperation and participation of the members of the VETR and their families. Without their contribution, this research would not have been possible.

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