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Religion/Spirituality, Risk, and the Development of Alcohol Dependence in Female Twins

Jon Randolph Haber,
Veterans Affairs Palo Alto Health Care System, Menlo Park, CA

Julia D. Grant,
Department of Psychiatry, Washington University School of Medicine

Carolyn E. Sartor,
Department of Psychiatry, Yale School of Medicine

Laura B. Koenig,
Department of Psychology, Kutztown University

Andrew Heath, and
Department of Psychiatry, Washington University School of Medicine

Theodore Jacob
Veterans Affairs Palo Alto Health Care System, Menlo Park, CA

Abstract

The contention that Religion/Spirituality (R/S) influences the development of alcohol dependence (AD) is increasingly supported, but risk factors have not been adequately examined together with protective R/S factors so as to determine the nature and relative strength of these domains at different stages in the development of alcoholism. Secondary data analysis of a sample of 4,002 young adult female twins used conditional Cox proportional hazards survival models to examine three distinct stages in the development of alcoholism: years to initiation of drinking, years from first drink to at-risk drinking, and years from at-risk drinking to AD. Risk and protective factors from models of alcoholism etiology and studies of R/S dimensionality were modeled simultaneously as predictors of each discrete stage and compared. Findings demonstrated that both risk factors and R/S variables influenced initiation of alcohol use; only R/S variables influenced subsequent progression to at-risk drinking; and risk factors primarily influenced further progression to AD. Protective factors (R/S variables being an exemplar) appeared to be critical determinants of intermediate-stage progression, thus suggesting that R/S factors and other psychosocial interventions might be particularly effective in delaying progression toward AD at this stage. In contrast, after the onset of at-risk drinking, the influence of (genetically based) risk factors appeared to accelerate AD regardless of most other influences. Thus, the timing of psychosocial interventions appears critical to their potency and impact.

Keywords

alcohol dependence; developmental stages; religionspirituality; protective factors; risk factors

Correspondence concerning this article should be addressed to Jon Randolph Haber, VAPAHCS, 795 Willow Road, MC 151J, Menlo Park, CA 94025. randyhaber@gmail.com.

Empirical research increasingly shows a direct relationship between religion/spirituality (R/ S) and health in general (H. G. Koenig, McCullough, & Larson, 2001) and an inverse relationship between R/S and substance dependence in particular. Miller et al. (1997; p.75) concluded that "reviews of the extant literature reveal a striking convergence of findings indicating the reasonably consistent inverse relationship between religion/spirituality and substance abuse and dependence." Empirical evidence is found in both epidemiological and clinical research (Kelly, Magill, & Stout, 2009; Kendler, Gardner, & Prescott, 1997; L. B. Koenig, Haber, & Jacob, 2011), and findings are reasonably stable whether R/S is examined retrospectively or prospectively, and whether R/S is defined as religious affiliation, participation, personal importance, or as part of one's upbringing (H. G. Koenig et al., 2001). As well, these findings have important clinical implications (Kaskutas, Turk, Bond, & Weisner, 2003). For example, Michalak, Trocki, and Bond (2007) used U.S. National Alcohol Survey data to show that religious affiliation, religious importance, and religious proscription were all strongly associated with abstinence from alcohol use. Kendler et al. (2003) used a measurement model to identify seven dimensions of R/S, and found that five of seven were inversely associated with alcohol dependence (AD). Pardini, Plante, Sherman, and Stump (2000) examined individuals in recovery from alcohol or drug addiction, and found that sustained recovery was associated with high levels of religious faith and religious affiliation. Haber and Jacob (2007) found that childhood religious affiliation moderated adolescent alcoholism risk for children of alcoholics. However, the growing literature on R/ S and other positive or protective influences has not been well integrated into the larger alcoholism and substance disorder literature, which to a greater extent focuses on risk factors, etiological models, and developmental pathways that predict disorder. To support the integration of the R/S and alcohol literatures, the current study examined the influence of both protective and risk factors on different developmental stages in the progression toward an AD diagnosis.

The empirical study of alcohol and substance use disorders has produced a well-developed literature and a number of explanatory models. Of current etiological models (Sher, Grekin, & Williams, 2005), the most prominent is the "social deviance" model that focuses on behavioral undercontrol beginning in early childhood. Early externalizing behaviors are seen as progressing to childhood disorders including attention deficit hyperactivity disorder (ADHD), oppositional defiant disorder (ODD), and/or conduct disorder (CD), and subsequently to early onset of alcohol use (McGue, Iacono, Legrand, Malone, & Elkins, 2001), increasing alcohol-related risk (Zucker, Fitzgerald, & Moses, 1995), and early development of an AD diagnosis (Caspi et al., 1997). Another etiological model, the "negative affect regulation" model, focuses on negative emotionality wherein early childhood internalizing behaviors progress to major depression disorder (MDD), social phobia, and other anxiety disorders (Kendler & Prescott, 2006). Behavior genetic research has further substantiated these models by identifying distinct genetic influences that underlie externalizing and internalizing disorders respectively. Environmental factors, specifically family factors such as parent–child conflict and parental divorce (Theodore Jacob et al., 2003) and exposure to negative life events (e.g., traumas) (Dube et al., 2006), have also been implicated as risk factors in both models. It should be noted that gender differences play a role in these models. Males have a higher prevalence for externalizing disorders, and females have a higher prevalence for internalizing disorders (Brady & Randall, 1999), although externalizing factors are important in both genders (Pitkänen, Katja, Anna-Liisa, & Lea, 2008). Gender differences are also evident in the specific risk factors identified within each model (Dawson, Grant, & Ruan, 2005; Lloyd & Turner, 2008). Taken together, these two models summarize the most reliable risk factors associated with the etiology and development of alcohol and substance use disorder.

Even so, a critical limitation of this literature is the lack of knowledge about the interplay between these well-documented risk factors and positive or protective factors that influence the etiology and development of substance disorders, a notable example being R/S influences. It has been argued that the literature shows a preoccupation with variation in risk that has resulted in the understudy of protective factors and the tendency to consider protection as simply the absence of risk (Richard Jessor, Van Den Bos, Vanderryn, Costa, & Turbin, 1995). However, the rise of positive psychology and the study of resilience factors (Keyes & Haidt, 2003; Pardini et al., 2000; Rutter, Clarke-Stewart, & Dunn, 2006; Rutter, 2006) has increasingly supported Rutter's (1987) view that protective factors and risk factors should be treated as conceptually distinct influences, not as opposite ends of a single continuum. To the extent that protective factors are independent variables with their own direct effects (Richard Jessor et al., 1995), protective and risk factors should be empirically distinct as well. Applied to the current study, the implication is that R/S influences, as an exemplar of protective resilience factors, and alcoholism risk factors, as the leading predictors of alcohol use disorders, should be modeled as separate and distinct influences and examined - simultaneously as independent predictors of alcohol outcomes.

To test this contention, this research group previously conducted a cross-sectional, multivariate study of R/S dimensions and risk factors as predictors of alcohol milestones (Haber, Grant, Jacob, Koenig, & Heath, 2012). A female adolescent/young adult twin sample was selected because the age range spanned the period of highest alcohol risk (Galanter, 2006), because risk has been accelerating for the female gender, and because this design could target early etiological factors associated with AD (Holdcraft & Iacono, 2002). Also note that - females report greater R/S involvement compared to males (Hood, Spilka, Hunsberger, & Gorsuch, 1996). This design, therefore, allowed an informative examination of R/S influences and recognized risk factors on key alcohol use milestones in a population where risk was rapidly increasing. In the study, nine R/S dimensions and eight alcoholism risk factors were examined as predictors of five alcohol milestones (initial drink, first intoxication, regular use, heavy consumption, and AD). Results indicated that risk factors and R/S variables were both strong and independent predictors of alcohol use milestones, and showed that neither mediation nor moderation effects contributed to observed effects. These findings confirmed that protective factors and risk factors were conceptually and empirically distinct, and that both were necessary to the prediction of alcohol - outcomes. Even so, results were limited in important ways that now - are addressed in the current study.

Specifically, in the past decade, the alcoholism literature has moved beyond static models of association to the study of factors influencing the developmental progression of alcohol use from onset to an AD diagnosis. Current evidence shows that risk factors accelerate a person's progression (Sartor, Lynskey, Heath, Jacob, & True, 2007), and that protective influences delay progression (Haber et al., 2012; H. G. Koenig et al., 2001). There are, however, differences in which psychiatric and psychosocial risk factors influence what developmental milestones when comparing (a) the initiation of alcohol use (McGue, Iacono, Legrand, & Elkins, 2001; McGue, Iacono, Legrand, Malone et al., 2001), (b) at-risk drinking (Zucker et al., 1995), and (c) AD diagnosis (Cloninger, Sigvardsson, & Bohman, 1996; T Jacob, Koenig, Howell, Wood, & Haber, 2009). Most studies focus on the predictors of specific milestones, but, with this approach, the predictors of any milestone will necessarily include the predictors of all preceding milestones. For example, the predictors of AD will also include the predictors of initiation of drinking since initiation always - precedes AD. A more precise, and perhaps more useful, approach is to conceptualize development as a series of discrete sequential periods rather than overlapping timeframes. This is not often done. As noted by Sartor et al. (2007, p.216), "the majority of studies assessing psychiatric and psychosocial risk factors for AD predict lifetime AD diagnosis without considering the

multistage process involved in the pathway from first use to AD." However, conditional analytic models allow a stage of progression to be defined as beginning at one milestone and ending at another milestone; this allows each developmental stage to be examined independently. In a study of young adults, Sartor et al. (2007) used conditional survival analysis to examine risk factors associated with (a) years to first drink and (b) years from first drink to AD. Results found CD to be a consistent predictor across both initiation and AD, but also found that ADHD, maternal AD, paternal AD, male gender, and parental divorce influenced only the initiation of drinking; and nicotine dependence, cannabis abuse, and generalized anxiety disorder only influenced subsequent progression to AD. Stage analyses proved to be effective in illuminating critical distinctions in the risk profiles at different stages of progression to AD. To date, no studies based in these methods have examined protective factors.

The current study used the same adolescent/young adult female twin sample described above. Both risk and protective factors were modeled as predictors of the duration of each of three nonoverlapping stages in the progression to AD: time (in years) to initiation of drinking, time from initiation to at-risk drinking, and time from at-risk drinking to AD using conditional survival analysis. The predictive covariates were (a) risk factors from the alcoholism etiology literature including childhood psychiatric disorders, familial risk factors, and traumatic events, and (b) protective factors from the R/S domain that were identified in the author's earlier work: religious motivation-devotion, religious involvement, religious prohibition, and religious affiliation types (Haber et al., 2012; Haber, Jacob, & Spangler, 2007). The aim of this study was to differentiate predictors during the developmental course to AD. Although the literature in this area is limited, it is sufficient to propose the following hypotheses:

1: Consistent with Haber et al. (2012), both risk and protective factors will simultaneously, independently, and differentially explain significant variation in the progression of alcohol use disorder;

1. Consistent with Sartor et al. (2007), factors known to influence the development of AD will be found to exert their influence at a specific stage of progression rather than continuously throughout the developmental period (Sartor et al., 2007; Zucker et al., 1995);

2. R/S variables known to reduce the prevalence of alcohol milestone endorsements (i.e., "personal" R/S variables; see Haber et al., 2012) will be found to delay the progression toward AD (at specific stages), and risk factors known to increase the prevalence of alcohol milestone endorsements (Haber et al., 2012; Sartor et al., 2007) will be found to accelerate progression toward AD (at specific stages);

3. The strongest identified R/S variables will have a similar effect size as the strongest identified risk factors in predicting the progression of AD (Haber et al., 2012; Richard Jessor et al., 1995).

Method

Sample

This secondary data analysis was based on data obtained from the adolescent/young adult female twin participants and their parents as part of the Missouri Adolescent Female Twin Study (MOAFTS; NIAAA AA09022, PI Heath). The study targeted all twin pairs live-born in Missouri between 1975 and 1987. Of those families identified, an 87% participation rate was achieved for the initial interview, thus providing a large representative sample of the population of female twins in Missouri (Heath et al., 1999). Baseline assessments included

an initial parental interview and an expanded diagnostic interview obtained from the parents of 2,369 families and from each adolescent twin girl between the ages of 13 to 19 years (N = 3,582) together with an adolescent questionnaire (N = 2,080). There were 434 Black adolescent girls in this sample; other adolescents were almost entirely of European ancestry. Five years later, all originally targeted adolescent, now young adult, women were again contacted. For those completing wave 1, the participation rate was 84% at wave 2, and some earlier nonparticipants were added. Median age at baseline and follow-up was 15.8 and 21.8 years, respectively. Comprehensive data profiles on alcohol use, alcoholism risk factors, and religious variables were obtained. When compiled as a cross-sectional data set, N = 4,002 female offspring cases were available; when both waves of data were available (true for 84% of cases), the later data were used. Previous attrition analyses of this sample were based on geosocial information derived from census block data. Results indicated some minor demographic differences between participants and nonparticipants in the initial data collection, whereas those lost to follow-up were found to have lower income, non-White race, and more paternal history of alcoholism. Attrition bias on outcomes, however, was found to be not significant.

To the extent that this adolescent/young adult female sample exhibited lower rates of alcohol use (Brady & Randall, 1999) and somewhat greater religiousness (Hood et al., 1996) as compared to male samples, conservative estimates of alcohol use and some additional sensitivity in the detection of the R/S effects would be expected. Concerning use of a twin sample, since twins have not been found to be significantly different from nontwins on a variety of characteristics including alcohol abuse and alcohol dependence (Johnson, Krueger, Bouchard, & McGue, 2002), these findings can be considered generalizable to nontwin populations. As well, since the target sample was all live-born twin pairs in Missouri, this sample can be considered representative of the larger population from which they were drawn. Note that the twin feature of this dataset was not used in current analyses, but the issue of possible nonindependence of observations was addressed (see Data Analysis).

Assessment, Selection, and Form of Variables

Table 1 lists variables drawn from four domains of specific relevance to the present analyses: demographics, risk factors, R/S variables, and alcohol milestones. Variables were constructed (a) consistent with their original form as continuous, rank ordered, or dichotomous variables, and (b) after transformation to dichotomous form (to correct for normality and linearity violations observed in the original variables). Both formats were analyzed, and highly similar results were obtained. This was because the standard procedures for correcting violations of the Proportional Hazards (PH) assumption in Cox PH survival analyses (see Data Analysis) adjusted similar features of the data as are addressed with dichotomous transformation. Thus, both approaches yielded essentially equivalent results. The results reported here are from analyses based on dichotomous variables since these results were slightly more conservative.

Self-reported demographic variables were obtained to adjust for potential confounds. Parental education was a dichotomous indicator of fathers and mothers who attended at least some college. Because education data were partially missing, a binary variable was constructed to identify missing data for both father and mother; this allowed a zero to be entered (rather than a missing indicator) so as to include the case in most computations, and permitted assessment of the relationship between missing education data and alcohol outcome. Mother's report of family income was obtained and transformed into two binary income variables that represented the higher 20% and lower 20% income brackets. Offspring age identified those 21 years of age or older.

The assessed risk factors included comorbid psychiatric disorders and stressful life events known to be linked to alcohol outcomes. The psychiatric variables were obtained by self-report from each family member using an adapted Semi-Structured Assessment of the Genetics of Alcoholism-II (SSAGA-II) interview and its companion child (C-SSAGA-C) and adolescent (C-SSAGA-A) interviews. The SSAGA was developed from validated items used in other psychiatric research interviews (see Kathleen K. Bucholz, Cadoret, Cloninger, & Dinwiddie, 1994). Six *Diagnostic and Statistical Manual–Fourth Edition* (*DSM–IV*) (American Psychiatric Association, 1994) disorders were included: ADHD (mother's report on child), ODD, CD, MDD, PTSD, and Panic Disorder (child's report). The reliability of SSAGA diagnoses has been assessed, and test–retest reliabilities with kappas ranging from .70 to .90 have been reported (Hesselbrock, Easton, Bucholz, Schuckit, & Hesselbrock, 1999). Stressful life events were selected from SSAGA-II self-report items on trauma and adverse family experiences, factors identified in current literature as associated with AD outcomes.

Religion/spirituality (R/S) variables included three "personal R/S" variables and six religious affiliation variables, each having acceptable reliability (test–retest [t/rt] and/or internal consistency [Cronbach's Alpha] ratings). *Personal R/S variables*: Based on the investigators' factor analysis of items within the R/S domain (Haber et al., 2007), three personal R/S variables were identified: Religious Motivation-Devotion, Religious Attendance, and Existential Well Being. Religious Motivation-Devotion included four religious importance items (see R. Jessor & Jessor, 1977) (= .86); four religious well-being items [from the Spiritual Well-being Scale (see Ellison, 1983) (t/rt = .94; = .82)]; and one R/S self-rating item; the resulting factor score was dichotomized by median split. Religious attendance was a binary indicator of attending religious services one or more times per week. Existential Well-Being [four existential items from the Spiritual Well-being Scale (Ellison, 1983) (t/rt = .90; = .81)] was a nontheistic measure of spiritual well-being dichotomized by median split.

R/S affiliation types variables—Current affiliations were drawn from a respondent list of 20 choices including "no religious affiliation" that were categorized into four types: Differentiating, Accommodating, Catholic, and No Religious Affiliation. These types were described and validated in two previous studies (Haber & Jacob, 2007, 2009). The typology reflects differences in the degree to which the beliefs and behaviors espoused by the affiliation were similar to or different from those espoused by the general culture. Differences in beliefs (such as the return of Jesus Christ and healing through prayer) and in behavioral norms (such as prohibiting gambling and dancing) typify Differentiating affiliations. Similarities in beliefs and behaviors typify Accommodating affiliations. Differentiating affiliations included Baptist, Church of Christ, and other Protestant church affiliations ($n = 1,683$). Accommodating affiliations included Methodist, Lutheran, and Presbyterian churches ($n = 436$). The Catholic Church ($n = 797$) included both attributes and was examined separately. "No Religion" endorsements formed the reference group ($n = 625$ cases). [Note that 460 cases (11.5%) were unclassified due to minimal representation.] As well, across these types, religious proscription of alcohol was assessed by endorsement that a family's religious affiliation had rules against all alcohol use (a "Religious Rules" indicator). Substantial within-group heterogeneity was evident within these categories, but between-groups main effects have been sufficiently robust as to validate these constructs. Finally, religious affiliation during childhood was obtained from mother's report of family affiliation during child's ages 6–13, and was considered a family-level characteristic.

The outcome variables in this survival analysis was the time (in years) between a conditional starting event and an ending event; this provided a measure of the duration of a given stage. Censoring indicator variables were constructed to identify cases not reaching the ending

event by the close of data collection. Three stages in the progression to AD were examined: time to initiation of drinking (age at first full drink), time from initiation to at-risk drinking (years from first drink to either the first intoxication or the first period of regular drinking), and time from at-risk drinking to AD (in years). All age-of-onset variables were obtained by retrospective self-reports. The reliability of retrospective reports of alcohol use has been demonstrated (K. K. Bucholz, Heath, & Madden, 2000; L. B. Koenig, Jacob, & Haber, 2009).

Data Analysis

Survival analysis was used to model different stages of alcohol progression using time-to-event data to examine the impact of psychosocial and psychiatric risk and protective factors on the rate of progression; that is, the duration of each of the three stages defined above. Specifically, three Cox proportional hazards regression models were constructed to predict (a) time to first drink, (b) time from first drink to onset of at-risk drinking, and (c) time from at-risk drinking to onset of AD. Models included psychosocial factors, notably R/S, and psychiatric risk factors as covariates influencing rate of progression through each stage. The dependent variable was the cumulative survival function; that is, the proportion of cases that had not reached a given alcohol transition, thus "surviving" to that point in time. With this method, all data up to the time of censorship (i.e., time of interview) were used in calculating hazard ratios. Basic univariate analyses were conducted with PASW Statistics Software, Release 18.0.0 (IBM-PASW, 2009). Advanced analyses were conducted with STATA, version 8.2 (StataCorp, 2001). Standard deviations of means and confidence intervals for advanced Cox regression models were adjusted for family clustering using Huber-White robust standard errors. The Efron approximation (Hosmer & Lemeshow, 1989) was used to handle ties. Missing data were deleted listwise in Cox regression analyses.

Initial analyses involved a series of univariate Cox proportional hazards regressions used to characterize the relationship between individual psychiatric and psychosocial predictors and the rate of progression at a particular stage of AD development. Nine R/S variables and eight etiological risk factors were modeled as binary contrast covariates (without adjustment for demographic covariates). A general profile of effects was obtained.

Subsequently, three multivariate Cox proportional hazards regression models were constructed to identify the strongest predictors after accounting for all other variables. The starting model adjusted for eight demographic variables and included 11 risk factors and eight R/S variables. Since an early onset drinker generally progresses more slowly through these stages than later onset drinkers, this difference was controlled by an indicator of drinking before age 15. Each model was reduced by manual backward deletion to identify a final model of significant predictors. ODD, CD, MDD, and Trauma were modeled as time-dependent covariates; this eliminated cases where risk occurred after (rather than before) a given alcohol transition (causality can only be inferred if a predictor precedes transition). The approach was to construct "Person year data" using SAS, version 9.1 (SAS Institute Inc., 2006) consisting of one line of data for each person for each year of life. For cases positive for ODD, CD, MDD, or Trauma, a time-dependent variable was computed as 0 (absent) for each year up to the age of onset of the risk factor, and then 1 (present) from that year onward. Time invariant binary endorsements were used for (a) early age behaviors (e.g., under the age of 7 or 13), (b) when data were lifetime endorsements (e.g., have you ever had event X), or (c) when data reflected current status (e.g., family income).

The proportional hazards assumption (PHA) underlying Cox proportional hazards analyses requires that risk remain constant over time. The PHA was tested by the Grambsch and Therneau test of the Schoenfeld residuals (Grambsch & Threneau, 1994). Violations indicated uneven change across time (such as a large effect in early years and little effect in

later years). In the first model (age to first full drink), 12 variables violated the PHA, and the model was adjusted by splitting the age distribution into ages 1–11 years, 12–14 years, 15–17 years, 18–20 years, and 21 years and up. Interaction terms then represented the original variables as two (or more) age-specific sub-variables that covered the entire period and met the PHA (e.g., CD required two interaction terms, one modeling ages 0 to 14 and the other modeling ages 15 and up). The same procedure was used with the second model except that the time measure was the duration in years from first drink and onset of at-risk drinking. In this case, the distribution of years of duration was split into four divisions: 0–2 years, 3–5 years, 6–8, and 9 or more years. Two covariates, Black Race and Catholic Affiliation, required three-level interaction terms to address the PHA violation. In the model reduction phase, decisions about retaining or removing interaction variables were made for all components together, not separately, until the final step. For the third model, all variables met the PHA.

Results

Sample Description

As seen in Table 1, the majority of both fathers and mothers had attended some college; approximately 20% of households had an income under $25,000 and 15% had incomes over $60,000; and about half of these twin girls were over 21 years of age. A history of AD was reported by 20.1% of fathers and 4.8% of mothers (not shown). Mothers reported that 82% of offspring had some religious background, and 27% of these young adults said that they currently attended religious services at least weekly. Many offspring (85%) reported having had at least one full drink; of those, 79% reported at-risk drinking (getting drunk or drinking regularly); and 15% of at-risk drinkers had progressed to AD. Offspring were found to underreport childhood disorder, so parent reports were used for ADHD and ODD. Fifteen percent of offspring met criteria for ADHD (under age 7); 16% of offspring met criteria for ODD [mean age 11.3 (3.5) years]; 8% met criteria for CD [mean age 12.9 (2.6) years]; 31% met criteria for MDD [mean age 15.1 (3.8) years]; and 47.3% had a traumatic experience [mean age 11.5 (5.8) years]. Mean age of measured risk factors were found to occur earlier than mean age of alcohol milestones: Onset of drinking [mean age 15.8 (2.6) years]; onset of at-risk drinking [mean age 17.0 (2.5) years]; and onset of AD [mean age 18.1 (2.3) years].

Univariate Tests

Results of the univariate Cox proportional hazards analyses are seen in Table 2, indicating significant individual associations between each covariate and the survival curve: (a) Stage I shows time to first drink; (b) Stage II shows time from first drink to at-risk drinking; and (c) Stage III shows time from at-risk drinking to AD (without adjusting for demographics). In Stage I, every risk factor and every religion variable was found to influence time to initiation of drinking. The sign of the regression coefficient, if negative, indicated a protective effect (defined as a delay in progression) and, if positive, indicated an increase in risk (defined as accelerated progression). All risk factors accelerated initiation of drinking; the "personal R/S" measures (motivation/devotion, weekly attendance, and existential well-being) delayed initiation. Concerning R/S affiliation types, three affiliation variables delayed initiation: affiliations with rules against alcohol use, and both childhood and current Differentiating religious affiliation. In contrast, three affiliation categories had positive coefficients indicating accelerated initiation of drinking: Accommodating, Catholic, and endorsing no religious affiliation.

Results for the subsequent two periods were more discriminating. In Stage II, no risk factors influenced the progression of alcohol use from initiation to at-risk drinking. In contrast, six of nine R/S variables continued influencing progression. Two of the three personal R/S

measures (motivation-devotion and weekly attendance) and the same three affiliation variables (affiliations with rules against alcohol use, and both childhood or current Differentiating affiliation) delayed transition to at-risk drinking. (Existential well-being was not significant). Catholic affiliation was the only R/S affiliation variable that increased the rate of progression to at-risk drinking during Stage II.

In Stage III, the pattern was the reverse of that seen in Stage II. All alcohol risk factors robustly accelerated progression whereas R/S variables had little influence. EWB was the only R/S variable to delay transition to AD. (Note that the effect for Differentiating religious rearing was no longer a significant predictor when adjusted for demographics).

Multivariate Tests

Multivariate tests controlled for demographics, eliminated redundant variance, identified which factors were most strongly associated with progression, identified which stages were most influenced by each predictor, and indicated the direction of each predictor's effect after accounting for all other variables (see Table 3). In all three time periods, 8 to 12 independent predictors explained significant variation in the survival curve, and the pattern of effects within each period was distinct from the pattern of effects in the other periods. Use of z-scores allowed straightforward comparison and interpretation. In general, results indicated that the multivariate pattern of findings was consistent with the univariate pattern.

Concerning years to initiation of drinking (Table 3, Stage I), 10 independent predictor variables were identified after addressing violations of the PHA (see Method) and controlling for demographics. Considering risk factors, parental divorce/separation strongly accelerated drinking onset at all ages, but especially in younger offspring (age 1–14; $z = 6.30$). Offspring with a history of PTSD, ADHD, MDD, Trauma, or CD exhibited more rapid onset of drinking in childhood and early adolescence ($z = 4.07$, $z = 4.08$, $z = 3.97$, $z = 2.94$, and $z = 2.70$, respectively), but this acceleration was not evident after midadolescence. Concerning R/S variables, the strongest R/S effect was on initiation, and the strongest predictor across all variables and all stages, was religious attendance ($z = -12.45$). Concerning the initiation of drinking (age of first drink), R/S attendance was associated with delayed progression, a protective effect. Also delaying onset were Differentiating affiliation and motivation/devotion ($z = -4.91$ and -2.48, respectively). In contrast, Catholic affiliation accelerated onset of drinking ($z = 4.04$) in those who were age 12 and older.

Results for Stage II, from drinking initiation to at-risk drinking (defined as either first intoxication or first regular use of alcohol), are seen in Table 3, Stage II. Four independent predictors were identified after controlling for demographics, two of which needed to be partitioned to meet the PH assumption. All four predictors were R/S variables; no alcohol risk factor was significantly related to stage 2 drinking, results that were consistent with univariate findings (see Table 2). Two R/S variables were found to be protective (in that they delayed onset of at-risk drinking: current weekly religious attendance ($z = -3.66$), and those affiliations with rules against alcohol use ($z = -2.17$). In contrast, Accommodating religious affiliation ($z = 2.54$) and Catholic religious affiliation ($z = 3.69$ to 4.57 depending on age) accelerated onset of at-risk drinking.

The third model, seen in Table 3, Stage III examined predictors of time from at-risk drinking to meeting criteria for an AD diagnosis. All variables met the PH assumption, seven variables were significant, and six of these variables were risk variables that accelerated transition to AD. These included five psychiatric conditions: ODD, CD, MDD, Trauma, and Phobia ($z = 2.26$, 2.54, 2.44, 2.44, and 2.16, respectively) and a variable indicating an early initiation of drinking (at age 14 or younger). The only variable that delayed progression through Stage III to AD was the R/S variable of Existential Well-being ($z = -2.68$).

Discussion

In using conditional survival analysis to examine three distinct stages in the development of alcoholism (years to initiation of drinking, progression from initiation to at-risk drinking, and then progression from at-risk drinking to an AD diagnosis), the current study identified unique patterns of stage-specific influences for both risk and protective factors. Most important, protective R/S variables were found to be critical determinants of intermediate-stage progression, thus suggesting a specific timeframe within which psychosocial influences are more likely to be influential in delaying progression to AD. After the onset of at-risk drinking, however, the influence of protective factors appeared to be much reduced while the influence of risk factors became prominent in accelerating progression to AD. These findings suggest that the timing of protective influences is critical to their potency and impact. It may be that protective factors are "gatekeepers" that might inhibit progression of drinking into the at-risk stage. However, those with greater risk due to their psychiatric histories appear to be more likely to progress into at-risk drinking. Such a pattern of findings, if replicated, has important implications for the prevention of alcohol and substance use disorders, for understanding of the natural course of substance disorders, and for treatment efforts as well.

The findings of the current study need to be understood in their epidemiological context. This was a sample of adolescent/young adult women. Recent studies indicate that over half of U.S. adolescents have used alcohol, and females currently have about half the prevalence rate of alcohol and drug use disorders as seen in males (Merikangas & McClair, 2012). However, the rates of increase over recent decades have been 117% for females compared to 21% for males (Holdcraft & Iacono, 2002), thus indicating that risk for female substance use disorders (SUD) has been accelerating, and gender differences have been declining. This is critical because adolescence is the key developmental period for SUDs since there are striking increases in prevalence rates well into young adulthood (Merikangas & McClair, 2012), and because those who develop an SUD in adolescence are at increased risk for persistent symptoms well into adulthood (Rohde, Lewinsohn, Kahler, Seeley, & Brown, 2001) and into middle age (Pitkänen et al., 2008). Therefore, knowledge of the nature of risk and protective factors on this developmental period is critical, and especially for females.

The findings of this study are also informative. Whether examined at the univariate or multivariate levels of analysis, findings reveal different predictive patterns for risk and protective factors at each stage of development (see Table 2). Both risk factors and R/S variables were significant predictors of the first stage, years to initiation of drinking. However, no risk factors were significant at the second stage of development (onset to at-risk drinking) even though many R/S variables continued to be significant. This pattern was reversed in the final stage when the effect of R/S variables was minimal and every risk factor accelerated development of AD. Considerable variation was evident across these stages for both risk and protective variables. Most notable was the strength of R/S factors at Stage II and their role in delaying progression while, at the same time, there was an absence of influence from the risk factors. Thus, Stage II appeared to provide an important window of opportunity for intervention in the natural course of AD disorder.

Univariate analyses were consistent with previous findings regarding the direction of effects for different dimensions of R/S influence. Where significant, personal R/S variables (Religious Attendance, Motivation/Devotion, and Existential Well-being) consistently delayed progression (H. G. Koenig et al., 2001; Miller et al., 1997). Religious affiliation, however, was mixed in its pattern of effects, this pattern replicating that found in four earlier studies (Haber et al., 2012; Haber & Jacob, 2007, 2009; L. B. Koenig et al., 2011). That is, delayed progression was observed for Differentiating affiliations (e.g., conservative

Protestant, evangelical, and fundamental affiliations) and for affiliations with reported rules against all alcohol use. Religion-based differentiation appears to protect adherents in an alcohol-permissive majority culture. In contrast, Accommodating and Catholic religious affiliations were usually positively associated with AD, thus accelerating progression and acting as risk factors. This latter result, however, may be confounded with another characteristic. Both Accommodating (Lutheran, Presbyterian, and Methodist) and Catholic churches have longstanding traditions in American culture, and some adherents might endorse these religions simply because they were raised with that identification even if not practicing the religion per se. Nominal endorsements could inflate estimates of risk.

These univariate findings were consistent with and further elaborated in the comprehensive multivariate models. These analyses accounted for redundant variation and identified the stronger predictors of each stage. For initiation of drinking (see Table 3, Stage I), both risk and protective factors played important roles (though usually with opposite directions of effect). All significant risk factors accelerated onset; these included parental divorce or separation, psychiatric disorders (ADHD, CD, MDD, PTSD), and trauma. These findings are consistent with the general alcoholism literature (Zucker et al., 1995), and are specifically documented in the alcohol use onset literature (Maggs & Schulenberg, 2006; McGue, Iacono, Legrand, & Elkins, 2001; McGue, Iacono, Legrand, Malone et al., 2001), and the alcoholism etiology literature (Sher, Slutske, Stricker, Widiger, & Weiner, 2003; Zucker & Gomberg, 1986). Concerning R/S, the strongest effect was for religious attendance, which robustly delayed the age of first drink. Since this protective effect was considerably stronger than any risk factor's effect, it underscored the strength of R/S influences in early stages of alcoholism development. Other protective R/S influences were motivation-devotion and current Differentiating religious affiliation. These findings are consistent with the extant literature on R/S and age of onset (Gorsuch & Butler, 1976; H. G. Koenig et al., 2001). As can be seen, modeling both risk factors and protective R/S factors together confirmed the critical role of both domains on initiation of drinking.

Following initiation, progression to at-risk drinking (intoxication or regular drinking) was influenced by R/S factors but not by risk factors (see Table 3, Stage II). This finding is new to the literature and implies that progression may not be subject to the influence of recognized risk factors at this stage, but may be strongly influenced by protective factors including R/S variables. Religious attendance and religious affiliations having rules against all alcohol use delayed progression at this stage. Religious attendance was previously shown to encompass social factors (Haber et al., 2007) including religious social support, social norms, and social role models that discourage alcohol involvement (Edlund et al., 2010), and may inhibit drinking in ways that parallel Alcoholics Anonymous (Kelly et al., 2009). Thus, this intermediate stage of progression to AD might be a window of opportunity where the influence of risk factors is minimal and the influence of protective environmental factors (including R/S factors) may be high. Since females tend to be more religious and more carefully supervised than their male counterparts, these influences may be stronger in female than male samples. Even so, the observed pattern is robust and illuminates an important opening for intervention that may warrant further examination.

The final stage involves at-risk drinkers who are progressing toward an AD diagnosis (see Table 3, Stage III). Risk factors accelerating progression to AD were ODD, CD, MDD, Phobia, and Traumatic life events. These findings that are often reported in the literatures on the epidemiology or the comorbidity of AD diagnosis (Merikangas et al., 2010; Sher et al., 2005; Zucker et al., 1995). The frequent comorbidity observed between psychiatric disorders and AD diagnosis has also been the subject of behavioral genetic examination. The consistent finding is that common genetic variation explains much of the observed comorbidity between psychiatric risk factors and AD (Kendler & Prescott, 2006; Krueger,

Markon, Patrick, Benning, & Kramer, 2007; Slutske et al., 1998). Furthermore, according to Merikangas and McClair (2012), genetic epidemiological studies have consistently demonstrated that environmental factors play an important role in exposure to and initial use of substances, but that genetic factors have a major influence on progression of use to dependence. To the extent this is true, the expression of genetic risk would be more closely associated with the final stage of progression to an AD diagnosis than with earlier stages. One could speculate that it is the common genetic variance shared by psychiatric disorders and AD that influences the final stage of progression to AD, and that the earlier stages are less genetically and more environmentally determined.

Taken together, these results support the study hypotheses. Concerning hypothesis 1, study findings demonstrated that both risk and protective factors were important predictors of the progression of alcohol use disorder within its larger developmental course, and exerted their influence independently and differentially. Concerning hypothesis 2, both risk and protective factors known to influence the development of AD were found to exert their influence at specific stages of progression rather than continuously throughout the developmental period. No predictors influenced all three stages, and the majority of predictors only influenced one stage. Concerning hypothesis 3, personal R/S variables (known to reduce the prevalence of alcohol milestone endorsements) did delay the progression toward AD at specific stages, and risk factors (known to increase the prevalence of alcohol milestone endorsements) were found to accelerate progression toward AD at specific stages (Haber et al., 2012; Sartor et al., 2007). Concerning hypothesis 4, the strongest identified R/S variable (religious attendance; $z = -12.45$) did have a comparable (or stronger) effect size as the strongest identified risk factor (parental divorce/separation; $z = +6.30$) in predicting progression in Stage I. Generally, most effect sizes for both risk and protective factors were comparable and of similar and moderate size.

Limitations

This study was based on a sample of young adult women; thus, findings may not generalize to male samples or older age groups. As well, these results reflect only the specific risk factors and R/S variables selected; there are many other risk and R/S factors that should be examined. Religion and spirituality were not distinguished in this study; however, it appears that religion might be more influential in earlier stages and spirituality (notably, Existential Well-being) in later stages of alcohol progression. Methodologically, results could have been influenced by common method variance since most measures were derived from offspring self-reports. As well, since R/S is not time-invariant, religious change over time could alter these conclusions.

Conclusion

Risk and protective factors are empirically distinct influences, and both must be modeled in any comprehensive examination of the development of alcohol (or substance) use disorders, and will vary in their respective influence according to the developmental stage.

Acknowledgments

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Table 1

Prevalence of Endorsements for All Variables

	Operationalization	Prevalence
Demographic characteristics		
Dad's education	>12 years	54.6%
Dad's education	—	41.7%
Mom's education	>12 years	75.6%
Mom's education	—	16.2%
Higher income	$62,500	14.8%
Lower income	$25,500	19.7%
Offspring age	21 years	55.8%
Offspring race	African American	14.4%
Risk factors		
Early onset	14 years	23.7%
Late onset	18 years	19.6%
ADHD	6+ inattention sx or 6+ hyperactive sx for 6+ months	15.2%
ODD	4+ oppositional-defiant sx for 6 + months	15.6%
CD	3+ conduct disorder sx for 12+ months	7.5%
MDD	5+ major depression sx for 2+ weeks	30.9%
PTSD	PTSD criteria 1+ month	7.1%
Phobia	Unreasonable fear (with embarrassment) for 6+ months	14.3%
Trauma	Life at risk in an accident, disaster, witnessed a killing, raped, molested, physically attacked, abused, neglected, threat with weapon or kidnapped	47.3%
Inconsistent rules when parented	1-item	42.6%
Parent-child arguments (often)	1-item	21.1%
Parental divorce or separation	1-item	33.3%
Religion/spiritual variables		
Motivation-devotion	Sum of ratings on 4 items dichotomized by median split	58.9%
Attendance	Once per week+	27.1%
Existential well-being	Sum of ratings on 4 items dichotomized by median split	56.7%
Rules against any alcohol use	1-item	41.1%
Raised with a differentiating affiliation	Family affiliation at age 6–13	53.4%
Differentiating affiliation	Current (see Methods)	47.5%
Accommodating affiliation	Current (see Methods)	12.3%
Catholic affiliation	Current (see Methods)	22.5%
No affiliation	Current (see Methods)	17.7%
Alcohol progression	*n*	%
1st drink endorsed	3389 of 4001	84.7%
"At-risk" drinking endorsed (given 1st drink)	2675 of 3389	78.9%
AD dx endorsed (given at-risk drinking)	401 of 2675	15.0%

Table 2

Univariate Associations Using Cox Proportional Hazards Survival Models

	Stage 1 Years to onset		Stage 2 Onset to at-risk drinking		Stage 3 At-risk drinking to AD	
	B	*p*-value	B	*p*-value	B	*p*-value
2a. Risk factors						
ADHD	0.28	0.000			0.47	0.000
ODD	0.31	0.000			0.53	0.000
CD	0.82	0.000			0.93	0.000
MDD	0.25	0.000			0.70	0.000
PTSD	0.20	0.002			0.88	0.000
Phobia					0.71	0.000
Trauma	0.18	0.000			0.58	0.000
Inconsistent rules when parented	0.23	0.000			0.26	0.01
Parent–child arguments	0.23	0.000			0.34	0.002
Parental divorce or separation	0.31	0.000			0.32	0.002
2b. Religion/spirituality variables						
Attendance (current)	−0.71	0.000	−0.34	0.000		
Motivation-devotion	−0.42	0.000	−0.22	0.000		
Existential well-being	−0.19	0.000			−0.48	0.000
Rules against any alcohol use	−0.19	0.000	−0.19	0.000		
Raised with a differentiating type of affiliation	−0.37	0.000	−0.28	0.000	0.26	0.01
Differentiating affiliation (current)	−0.43	0.000	−0.24	0.000		
Accommodating affiliation (current)	0.15	0.007				
Catholic affiliation (current)	0.32	0.000	0.25	0.000		
No affiliation (current)	0.26	0.000				

Note. Positive B scores reflect a higher hazard ratio, an increase in risk, an accelerated transition; negative B scores reflect lower hazard ratios, a decrease in risk, a delayed transition (a protective effect). Significant scores are shown; protective effects (delayed transition) are underlined.

Table 3

Significant R/S and Risk Factors Across 3 Stages of Progression to Alcoholism

	Stage 1 Time to drink 1		Stage 2 Drink 1 to at-risk use		Stage 3 At-risk use to AD	
	Age	z-score	Period	z-score	Period	z-score
Demographic characteristics						
Dad's education > 12 years						
Dad's education —						
Mom's education > 12 years					all	+2.29
Mom's education —	1–14 / 15–17	−3.28 / −2.95	all	+2.86	all	+2.56
Higher income $62,500	1–11	−2.36	all	+2.65		
Lower income $25,500	12–30	−3.07				
Offspring age 21 years			all	−2.58		
Offspring race = African American			0–2 yrs / 3–5 yrs	−7.40 / −2.93	all	−3.57
Risk factors						
Early onset 14 years	—	—				
Late onset 18 years	—	—				
ADHD	1–17	+4.07			all	+2.66
ODD [Time-variant]	1–14	+2.70			all	+2.26
CD [Time-variant]	1–14	+3.97			all	+2.54
MDD [Time-variant]	1–14	+4.08			all	+2.44
PTSD	1–14	+4.08				
Phobia					all	+2.16
Traumatic event (life at risk in an accident, disaster, witnessed a killing, raped, molested, physically attacked, abused, neglected, threat with weapon or kidnapped) [Time-variant]	1–17	+2.94			all	+2.44
Inconsistent rules when parented						
Parent–child arguments (often)						
Parental divorce or separation	1–14 / 15–30	+6.30 / +4.48				

| | Stage 1 | | Stage 2 | | Stage 3 | |
| | Time to drink 1 | | Drink 1 to at-risk use | | At-risk use to AD | |
	Age	z-score	Period	z-score	Period	z-score
Religion/spiritual variables						
Attendance (current)	all	−12.45	all	−3.66		
Motivation-devotion	all	−2.48				
Existential well-being					all	−2.68
Rules against any alcohol use			all	−2.17		
Raised with a differentiating type of affiliation						
Differentiating affiliation (current)	all	−4.91				
Accommodating affiliation (current)			all	+2.54		
Catholic affiliation (current)	12–30	+4.04	0–2 yrs	+3.69		
			6+ yrs	+4.57		
No affiliation (current)– reference group	—	—	—	—	—	—

Note. Positive *z*-scores reflect a higher hazard ratio, an increase in risk, an accelerated transition; negative *z*-scores reflect lower hazard ratios, a decrease in risk, a delayed transition (a protective effect). Positive *z*-scores are set in italic. Negative *z*-scores are underlined.