Mediation of Family Alcoholism Risk by Religious Affiliation Types*

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ABSTRACT. Objective: Religious affiliation is inversely associated with alcohol dependence (AD). Our previous findings indicated that when a religious affiliation differentiated itself from cultural norms, then high-risk adolescents (those having parents with alcoholism history) raised with these affiliations exhibited fewer AD symptoms compared with adolescents of other religious affiliations and nonreligious adolescents. The first of two studies reported here provides a needed replication of our previous findings for childhood religious affiliation using a different sample, and the second study extends examination to current religious affiliation. **Method:** A national sample of male and female adolescents/young adults ($N = 1,329$; mean age = 19.6 years) was selected who were the offspring of members of the Vietnam Era Twin Registry. Parental alcoholism, religious affiliation types, and their interactions were examined as predictors of offspring AD symptoms. **Results:** (1) Offspring reared with a differentiating religious affiliation during child-

hood exhibited significantly fewer AD symptoms as young adults; (2) offspring with current differentiating religious affiliation also exhibited fewer AD symptoms; this main effect was not weakened by adding other measures of religiousness to the model; (3) differentiating religious affiliation was correlated with both family alcoholism risk and offspring outcome, and removed the association between family alcoholism risk and offspring outcome, thus indicating that differentiating religious affiliation was at least a partial mediator of the association between family AD history risk and offspring AD outcome. **Conclusions:** Current results indicate that religious differentiation is an inverse mediator of alcoholism risk for offspring with or without parental AD history and regardless of the influence of other religion variables. Results replicated our previous report on religious upbringing between ages 6 and 13 years and indicated an even stronger effect when current differentiating affiliation was examined. (*J. Stud. Alcohol Drugs* **70**: 877-889, 2009)

ALCOHOLISM RISK IS ASSOCIATED WITH specific family lineages (Cotton, 1979), and thus family studies have become an important component of alcoholism research (Hartman et al., 2006). These studies have shown that more than half of the variance in adolescent alcohol dependence (AD) is accounted for by factors transmitted from parents to children (Hartman et al., 2006). Further, the familial nature of alcohol-use disorders has been shown to involve both genes (inherited from parents; Heath et al., 1997; McGue, 1997; Prescott and Kendler, 1999) and environmental factors (usually provided by parents; Hartman et al., 2006; Hopfer et al., 2003; Jacob et al., 2003). One psychosocial factor that is reliably and inversely associated with alcoholism risk

and that appears to modify the transmission of alcoholism risk from parents to children is family religion. This inverse relationship is well documented in the psychosocial literature and includes support from both epidemiologic and clinical studies (Kendler et al., 1997) using both retrospective and prospective designs and is observed with various religious variables including membership, participation, personal importance, and upbringing (Gorsuch and Butler, 1976). In addition to basic research, religion/spirituality (R/S) also has a history of relevance to alcoholism treatment, and programs based on R/S approaches have been shown to be as effective as other evidence-based approaches in the treatment of AD (Project Match Research Group, 1997). This report presents two similar studies. The first provides a needed replication of our earlier findings on the role of childhood religious affiliation (ages 6-13 years) on subsequent adolescent alcohol outcomes using a different sample. The second extends examination to consideration of young-adult religious affiliation and alcohol outcomes.

Of particular interest to the current study were findings regarding particular types of religious affiliation. For example, from a nationally representative sample of adolescents who participated in the National Comorbidity Survey, results indicated that those in fundamentalist religious denominations exhibited significantly lower rates of alcohol abuse

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(AA) and AD than those in other denominations (Miller et al., 2000). These findings were consistent with earlier reports suggesting that groups having strong rules against alcohol consumption (Beeghley et al., 1990; Bock et al., 1987) or strong doctrinal positions on sin and redemption (Francis, 1994) were associated with lower rates of alcohol problems. As well, in a comprehensive study of Australian adult twins that included an alcohol challenge procedure, *ADH2* genotyping, broad psychiatric assessment, and demographic data (Heath et al., 2003), "other Protestant" religious affiliation (a group including all smaller Protestant denominations) was significantly and inversely associated with AD even after controlling for the most well-recognized predictors of alcohol outcomes (including history of childhood conduct disorder, major depression, regular smoking, high alcohol sensitivity, and possession of the *ADH2*2* allele). Also, ethnic groups may be associated with a particular religion that influences alcohol use in that community. In a study of African-American adolescent female twins and their white counterparts conducted in Missouri, African-American girls exhibited a later age at onset of alcohol use. However, when religion was controlled, the effect was removed, thus indicating that religion mediated the ethnic effect (Heath et al., 1999b). Clarification of the underlying mechanisms by which religious affiliation exerts its influence would be beneficial.

In the first of two studies reported here, we examined differences in childhood religious affiliation as a predictor of subsequent young-adult AD symptoms and as a mediator or moderator of the transmission of alcoholism risk from parents to children. This provided a needed replication of our previous study (Haber and Jacob, 2007) that found that offspring at high risk (by virtue of their parent's history of alcoholism) who were raised with any religious affiliation (vs no religious affiliation) subsequently exhibited fewer AD symptoms in adolescence (at a mean age 15.8 years). Findings also indicated that this moderation effect was accounted for by a specific subset of religious affiliations that could be characterized as "differentiating" affiliations. The religious differentiation construct has considerable history but was more recently operationalized by other researchers (Johnson, 1963; Stark, 1985) into a typology based on the observation that religious groups differed on a continuum from highly accommodating ("Type A") to highly differentiating ("Type D") with respect to their support of general cultural norms. As described below, Type A affiliations referred to mainline churches that were inclusive in membership, accommodating of cultural norms, and seldom at odds with major cultural values. In contrast, Type D affiliations referred to smaller church groups who were more exclusive in membership and more aligned with notions of religious purity and religious values that could be considerably at odds with majority cultural ideas and values. The hypothesis was that, given permissive cultural norms about alcohol use in the United States, if a religious affiliation modeled differentiation from

cultural norms, then high-risk youth raised with this affiliation would follow the model and differentiate themselves from cultural pressures promoting alcohol use. Results in our previous study confirmed fewer AD symptoms for high-risk youth with differentiating affiliations compared with high-risk youth of other religious affiliations and nonreligious youth. However, the original study's sample was a young female adolescent community sample of twins and their parents from Missouri. Hence, replication using a more generalizable sample was needed. To address this need, the current replication used a sample that was national in scope and involved families with adolescent and young-adult offspring (mean age = 19.6 years) who were of both genders and were not twins (as in the original study).

Four basic questions/hypotheses guided our approach: (1) Does religious affiliation (broadly defined) predict alcohol outcomes? (2) Does religious affiliation (broadly defined) mediate and/or moderate the association between family history of alcoholism and offspring alcohol behavior? (3) Do specific affiliation types differentially mediate and/or moderate the association between family history of alcoholism and offspring alcohol behavior? and (4) Do any other religion variables account for the observed affiliation effect? These four questions were examined for religious affiliation at two different periods. Study 1 examined the impact of childhood religious affiliation on subsequent alcohol outcomes. Study 2 examined the impact of young-adult religious affiliation on young-adult alcohol outcomes.

Method

Subjects

This family study focused on a national sample of 1,329 male and female adolescents and young adults (mean age 19.6 years) and their biological parents. These data were collected for the Children of Alcoholics (COA) project (Jacob et al., 2003), where one major aim was to examine the mediators and moderators of alcoholism risk within the context of family alcoholism risk. The fathers of this study were adult twin members of the Vietnam Era Twin Registry. The Vietnam Era Twin Registry is a national military twin registry that is comprised of male-male twin pairs born between 1939 and 1957 where both twins served in the U.S. military between May 1965 and August 1975 (see Eisen et al., 1987, 1989; Henderson et al., 1990, for a detailed description). In 1987, Registry members completed a mailed questionnaire on their general health and military experience (Eisen et al., 1991). In 1992, Registry members were administered a telephone psychiatric interview covering a range of psychiatric disorders that included alcohol-use diagnoses (Harvard Drug Study; Tsuang et al., 1996). About a decade later, the current COA study was conducted, which focused on the effects of paternal AD on offspring outcomes. The COA data collection

supplemented earlier data on these fathers and also collected data on the father's offspring and on the biological mothers of these offspring. Of the eligible twins, 1,295 (83%) participated in the COA father's telephone interview. Participating fathers identified and gave consent to contact 1,113 mothers; 904 mothers (81%) participated in the mother's telephone interview. Consent from both parents regarding contact with their children was obtained, for a total of 1,564 offspring. Of these, 1,329 offspring (85%) participated in an offspring telephone interview. Data collection for the COA study was conducted in 2000-2001, and the procedures by which the sample was drawn from the Vietnam Era Twin Registry, by which the Institute for Survey Research at Temple University in Philadelphia conducted data collection activities, and the content and sources of the comprehensive assessment battery of the larger study have been extensively documented elsewhere (Haber et al., 2005; Jacob et al., 2003).

Assessment

Data for the variables used in the current study were derived from several sources. The Harvard Drug Study (Tsuang et al., 1996) obtained psychiatric and substance abuse and dependence diagnostic data on all fathers (lifetime diagnoses according to the Diagnostic and Statistical Manual of Mental Disorders, Third Edition, Revised (DSM-III-R; American Psychiatric Association, 1987). The father's telephone interview in the COA study obtained updated demographic data on fathers (e.g., age and employment status). As well, both mothers and offspring were administered diagnostic interviews adapted from the Semi-Structured Assessment on the Genetics of Alcoholism, Version II, assessment battery (Bucholz et al., 1994), which provided lifetime diagnoses according to the DSM, Fourth Edition (DSM-IV; American Psychiatric Association, 1994). The mother's telephone interview in the current COA study provided basic demographic data on mother and family (e.g., age and household income), self-reported psychiatric and substance abuse and dependence diagnoses (notably, AA and AD diagnoses), and major depression diagnoses, as well as childrearing reports for each offspring. Mothers reported on the religious rearing of each child when he or she was 6-13 years of age that included his or her religious affiliation and his or her frequency of attendance. The offspring's telephone interview in the current COA study provided demographic data (e.g., age and gender) and extensive self-report data on alcohol use, abuse, and dependence (including lifetime diagnoses), and on current religious characteristics, in particular, current religious affiliation, current frequency of attendance, and all other current religion variables.

Major variables used in the present article, and their operationalization, were as follows.

Family alcohol history. Paternal DSM-III-R diagnoses for lifetime AD or AA were obtained from Harvard Drug Study

data and were combined with maternal DSM-IV diagnoses for lifetime AD or AA obtained in mother's COA interview to create a binary indicator for any alcohol-use diagnosis in the history of either parent.

Paternal psychopathology. Paternal DSM-III-R diagnoses for lifetime antisocial personality, major depression, generalized anxiety, social phobia, and posttraumatic stress disorder were obtained from Harvard Drug Study data and used to create a binary indicator of any psychiatric diagnosis in father's history.

Maternal depression. In the mother's telephone interview, a structured lifetime major depression diagnostic assessment was administered that provided a DSM-IV diagnosis for major depression, which was included as a binary variable.

Offspring alcohol-dependence symptom count. In the offspring telephone interview, a structured lifetime alcoholism diagnostic assessment was administered that included assessment of each of the seven DSM-IV AD criterion; the endorsed diagnostic criteria were summed to produce an AD symptom count.

Religious affiliation. For childhood affiliation, mothers were asked to identify the religious affiliation of each of her children during their ages 6-13 years by selecting the primary affiliation from a list of 20 affiliations including "no religious affiliation." For current affiliation, offspring were asked to identify their current religious affiliation from the same list of 20 choices.

Other childhood religious variables. The COA mother's telephone interview included the mother's report on the child's religious activity during the ages 6-13 years. In addition to affiliation, questions included (1) the child's rate of religious attendance (reduced to binary form indicating attendance weekly or more) and (2) religious rules against all alcohol use by the child's affiliation (binary yes or no).

Other current religious variables. In addition, the COA offspring's telephone interview included this child's self-report of his or her current religious activity. The items used in the two current studies included (1) offspring's current rate of religious attendance (binary indicating weekly or more); (2) religious rules against all alcohol use at the current affiliation (binary yes or no); (3) single item: reliance on religious teaching when you have a problem (binary indicator of the highest of three levels: "very important"); (4) single item: importance of belief in God (binary indicator of the highest of three levels: "very important"); (5) single item: reliance on religious beliefs as a guide for day-to-day living (binary indicator of the highest of three levels: "very important"); (6) single item: importance of turning to prayer when you have a problem (binary indicator of the highest of three levels: "very important"); (7) religious/spiritual self-rating on a scale of 0 to 100 (binary indicator of the highest quartile); and (8) time spent on religious or spiritual practices (binary indicator of the top two of four categories).

Demographic variables. Based on questions asked of each respective family member during their telephone interview, dichotomous indicators were constructed to adjust analyses for variation arising from key demographic differences as follows: (1) offspring age of 18 years or older (examining differences between adolescent and young-adult offspring), (2) offspring gender is male (examining differences between male and female behaviors), (3) father's education includes some college (examining socioeconomic-status differences between more and less paternal education), (4) mother's education includes some college (examining socioeconomic-status differences between more and less maternal education), and (5) family income exceeds \$65,000, the third and fourth quartile of income (examining socioeconomic-status differences between higher and lower family income).

Development of religious affiliation typology

Early research on religious groups by key scholars (e.g., Max Weber; Reinhold Niebuhr) (Hood et al., 1996) suggested that religious affiliation groups differed in the degree to which their beliefs, values, and behaviors corresponded with those of the larger host culture in which the group was embedded. Further theoretical development by Johnson (1963, 1971) and more recent empirical work by Bainbridge and Stark (1980a, 1980b) resulted in a survey of 2,326 Protestant denomination members that assessed (a) behaviors such as gambling, dancing, and censorship, and (b) beliefs such as evolution, the return of Jesus Christ, and healing through prayer. These efforts operationalized a hypothesized accommodation-differentiation dimension reflecting the degree of correspondence between the beliefs and behaviors of a given religious affiliation and U.S. culture in general. Results supported the typology (Hood et al., 1996) and distinguished mainstream Protestant churches and smaller Protestant "sectarian" groups. This work, and the seven criteria described by Oden (1994), directed the categorization used previously and in the current study as described above (Type A for accommodation and Type D for differentiation). Development of the childhood model for Study 1 is illustrative. To avoid extreme means associated with small *n*'s and to improve interpretability, several affiliations were excluded (those with fewer than 25 member endorsements): Jehovah's Witnesses (*n* = 12), Orthodox (*n* = 6), Jews (*n* = 3), and Seventh-Day Adventists (*n* = 3). Also excluded was an undefined "other" group that could not be categorized (*n* = 62). Thus, the Type A affiliation group (*n* = 351) included Methodist (*n* = 103), Lutheran (*n* = 148), Presbyterian (*n* = 41), and Church of Christ (*n* = 33) affiliations. The Type D affiliation group (*n* = 378) included Baptist (*n* = 242), other Protestant (*n* = 95), and Mormon (*n* = 41) affiliations. The other Protestant group encompassed the many smaller Protestant denominations (see Bainbridge and Stark, 1980b). As in any categorization scheme, some groups were near the center, and judgment

was involved in classifying these groups. Further, there was notable within-group heterogeneity in most affiliations. Lutheran affiliations are classified as accommodating, although some Lutheran denominations are relatively differentiating groups; Baptist affiliations are classified as differentiating, although some Baptist denominations are relatively accommodating groups. Nonetheless, between-group main effects have been reliably observed in previous research (Bainbridge and Stark, 1980b) and our own research (Haber and Jacob, 2007), and as will be seen, appear to represent an important explanatory construct. Earlier research did not consider Roman Catholicism. The worldwide nature of Catholicism appeared consistent with accommodation characteristics; however, differentiation characteristics can be observed in Catholic opposition to current social trends (e.g., abortion, eliminating school prayer). Given that this affiliation was of sufficient size (*n* = 993), a separate group was included (Type C for Catholic). Finally, a "no religion" reference group (*n* = 113 cases) was assembled from those who endorsed "no religion" on the affiliation question. Total *N* for both studies was 1,215 cases and included four nominal predictive categories.

Data analysis

To characterize the sample on demographics (age, gender, race, parental education, father's employment, family income) and parental psychopathology (father's psychiatric history, mother's depression, and both father's and mother's alcohol history) according to the four religious affiliation groups, simple analysis of variance procedures were used.

Hypothesis testing focused on the prediction of offspring AD symptoms as the outcome of interest, a quasicontinuous dependent variable. Linear regression analyses were employed to test models of increasing complexity. All models included covariates to control for demographic variation (offspring age, offspring gender, father and mother's educational level, and family income) and parental psychopathology (father's psychiatric history and mother's depression). Study 1 began with a simple examination of childhood religious affiliation (any affiliation between ages 6 and 13 years compared with no affiliation) as a predictor of offspring AD symptoms (Model 1). Model 2a was a straightforward test of family history of alcoholism as a predictor of offspring AD symptoms. (Note that, because there was no religion term in Model 2a, this model became the basic comparison with which all subsequent "religion" models were compared [Models 3a to 9a were identical.]) Model 2b examined both family history of alcoholism and childhood religious affiliation and their interaction as predictors of offspring AD symptoms. Model 3 expanded Model 2 by simultaneously considering family history of alcoholism and the three affiliation types (accommodating, differentiating, and Catholic compared with the nonreligious type) and their interactions

as predictors of offspring AD symptoms. Finally, Model 4 expanded Model 3 by adding additional religious variables to the model (one at a time) to determine if other religious measures explained all or part of the observed affiliation effect. These four models address the four questions posed above. For each multifactorial model, bivariate correlations were computed to determine whether an affiliation variable was correlated with both the predictor (family alcoholism risk) and the outcome (offspring AD symptoms), an essential criterion in evaluating mediation effects.

Study 2 also included four models (Models 5, 6, 7, and 8) comparable to the four models for Study 1, but instead of childhood affiliation, current religious affiliation was examined. Model 8 examined 10 additional religion variables in individual tests in a comprehensive examination of whether other religious variables accounted for observed affiliation effects. Further, Model 9 combined these tests into an all-inclusive multivariate model to determine which religion variables most greatly influenced observed affiliation effects while accounting for all other factors.

Models were also constructed that were stratified by race to test hypotheses about African-American churches. Although African Americans were underrepresented in this sample, resulting in statistical power that was insufficient for conclusive results, it was still desirable to examine mean patterns of effects for comparison with the original study's findings.

All basic statistics, linear regression models, confirmatory transformation models, and graphical comparisons were completed with SPSS software (SPSS for Windows, Release 14.0.1; SPSS Inc., Chicago, IL). Replication of results while controlling for the nonindependence of observations given the similarity of offspring within the same families was conducted using STATA software (STATA Release 7; StataCorp LP, College Station, TX).

Results

Sample description

As seen in Table 1, sample demographics and parental psychiatric status were examined for 1,215 qualifying families. Offspring had a mean age of 19.6 years and were 51% male. Fathers had a mean educational level of 13.7 years, and mothers averaged 14.2 years of education in this national sample. Fathers were 92% employed, and family income averaged about \$71,000. Several significant differences were identified by analyses of variance across the four religious affiliation groups. First, although father's education did not vary across groups, mother's education was higher in accommodating affiliations and lower in differentiating affiliations. Second, consistent with lower education, families with differentiating affiliations reported lower annual family incomes (more than \$10,000 lower). Concerning psychiatric

characteristics, fathers with accommodating affiliations reported fewer psychiatric disorders than other groups, and those with no religious affiliation reported more psychiatric disorders than others. Because this was a high-risk sample designed to overselect for paternal AD, prevalence of AD was higher than the general population. In this sample, about 55% of fathers with differentiating affiliations met AD or AA criteria during their lifetime, and 74% of fathers in the "no religious affiliation" group met AD or AA criteria. Mother's alcohol disorders did not differ significantly across groups.

Hypothesis testing

Study 1 tested four hypotheses regarding childhood religious affiliations. Model 1 (see Table 2) tested an all-inclusive religious affiliation variable ("any religious affiliation" vs none) as a predictor of offspring AD symptoms and found a trend toward significance ($t = -1.8$, 1 df, $p = .07$). Model 2 examined family history of alcoholism as a predictor of offspring AD symptoms (Model 2a) and then added "any religious affiliation" as a second predictor and an interaction term (Model 2b). The baseline test (Model 2a) found a highly significant family history effect ($t = 5.1$, 1 df, $p < .001$) that confirmed strong parent to child transmission of alcoholism risk. This baseline model excluded religion variables and was the basis of comparison for subsequent tests of religious affiliation from Model 2 through Model 9). Model 2b provided a test of religious affiliation as a potential mediator (main effect) or moderator (interaction effect) of the association between family history of alcoholism and offspring AD symptoms. Results indicated a trend toward a main effect for the "any religious affiliation" variable ($t = -1.63$, 1 df, $p = .10$); the interaction was nonsignificant. Although short of the significance threshold, the borderline main effect almost entirely accounted for the family history effect, as shown by the decrease of this effect to nonsignificance ($t = 0.26$, 1 df, $p = .79$).

Model 3 provided more precise characterization by examining religious affiliation types—accommodating, differentiating, and Catholic types—and their interactions with family history. Again, Model 3a referred to the baseline model. Adding affiliation variables, Model 3b again indicated that the family history of alcoholism effect was removed ($t = 0.26$, 1 df, $p = .81$), and the differentiating affiliation type significantly predicted offspring AD symptoms ($t = -2.1$, 1 df, $p = .04$). As seen in Figure 1, offspring raised in differentiating affiliations, both with and without family history risk, exhibited lower mean rates of AD symptoms in contrast to all other groups. As well, the correlations between the differentiating affiliation variable and both the predictor and outcome variables were significant (correlations: family alcohol risk with differentiation, $r = -.11$, $p < .001$; offspring AD symptom count with differentiation, $r = -.11$, $p < .001$). These criteria established that the differentiating affiliation

TABLE 1. Demographics and psychiatric status by affiliation types and entire sample

Variable	<i>p</i>	Type A Accommodating (<i>n</i> = 351) (28.9%)	Type C Roman Catholic (<i>n</i> = 373) (30.7%)	Type D Differentiating (<i>n</i> = 378) (31.1%)	Type N No religion (<i>n</i> = 113) (9.3%)	Total (<i>n</i> = 1,215) (100%) (reference)
Age of offspring, in years mean (SD)	NS	19.3 (4.2)	20.0 (3.9)	19.6 (4.2)	19.4 (4.2)	19.6 (4.1)
Gender of offspring % male	NS	51%	53%	49%	58%	51%
Father's education, in years mean (SD)	NS	13.8 (1.9)	13.8 (1.9)	13.6 (1.9)	13.8 (1.9)	13.7 (1.9)
Mother's education, in years mean (SD)	≤.001	14.6 (2.1)	14.3 (2.1)	13.9 (2.1)	14.1 (1.9)	14.2 (2.1)
Father's employment % employed	NS	94%	94%	91%	88%	92%
Family income mean (SD)	≤.001	74.0K (35.4K)	74.8K (36.1K)	62.9K (32.9K)	73.5K (39.3K)	70.7K (35.6K)
Father's psychiatric history % positive	≤.05	18%	25%	25%	28%	23%
Mother's depression history % positive	NS	21%	16%	18%	22%	19%
Father's alcohol history % AD or AA	≤.001	59%	65%	55%	74%	61%
Mother's alcohol history % AD or AA	NS	12%	13%	18%	19%	14%

Notes: NS = not significantly different across groups; K = \$1,000; AD = alcohol dependence; AA = alcohol abuse.

FIGURE 1. Offspring at low risk (neither parent has a history of alcohol dependence [AD]) have fewer AD symptoms compared with high-risk offspring; in addition, offspring raised with a differentiating religious affiliation have significantly fewer AD symptoms than all other groups regardless of risk status. Ref = reference group; NEG = negative; POS = positive; Hx = history; relig affil = religious affiliation.

type met criteria as an inverse mediator of the relationship between family alcoholism history risk and offspring AD symptoms.

Model 4 examined whether the mediating effect of differentiating affiliation on family history of alcoholism risk would be accounted for by other childhood religion variables (ages 6-13 years). Results indicated that the differentiating affiliation effect was adjusted only a minor amount by two childhood religion variables, but this small change reduced the marginal significant effect from slightly below ($p = .04$) to slightly above ($t = -1.8$, 1 df, $p = .08$) the significance threshold ($p = .05$). Nonetheless, the effect of family history

of alcoholism was again removed by religious affiliation across these models.

Study 2 turned to the influence of current religious affiliation on young-adult AD symptoms and followed four parallel hypotheses. As seen in Table 3, in Model 5 (similar to Model 1), "any current religious affiliation" (vs none) was examined as a predictor of offspring AD symptoms. Results indicated a highly significant effect ($t = -4.7$, 1 df, $p < .001$).

Model 6 returned to the baseline model of family history of alcoholism as a robust predictor of offspring AD symptoms ($t = 5.1$, 1 df, $p < .001$). Again, Model 6b added "any current religious affiliation" and an interaction term to test a potential mediator-moderator relationship. Results indicated a highly significant main effect for (any) religious affiliation ($t = -4.1$, 1 df, $p = .000$) and a significant interaction with family history of alcoholism ($t = 2.2$, 1 df, $p = .03$). As well, religious affiliation again almost entirely accounted for the family history effect, as shown by its decrease to nonsignificance ($t = 0.15$, 1 df, $p = .88$), and the correlations between the general affiliation variable and both the predictor and outcome variables were significant (correlations: family alcohol risk with any affiliation, $r = -.09$, $p = .002$; offspring AD symptom count with any affiliation, $r = -.16$, $p < .001$). These criteria established that the general affiliation variable, when assessed currently, met criteria as an inverse mediator variable.

Model 7 examined accommodating, differentiating, and Catholic types of affiliation and their interaction with family history. Inconsistent with the above findings, family history risk was not removed, main effects were not significant, and there were two significant interaction effects, one for differentiation affiliations ($t = -2.9$, 1 df, $p = .004$), and the other for Catholic affiliation ($t = -2.1$, 1 df, $p = .04$). Further examination revealed that mean scores for low-risk and high-risk religious affiliation groups were relatively consistent between childhood and current affiliations. However, as seen in Figure 2, additional interaction variance clearly arose from a difference in the reference group, the nonreligious offspring. The inconsistency between Figures 1 and 2 was in the low-risk reference group, where a sharp increase in AD symptoms was exhibited (see Discussion for post hoc analyses). Obviously a change in reference group characteristics will affect all statistics. In this one instance, the sum of affiliation-related interaction variance and the exaggeration of interaction variance due to the reference group difference led to a statistical repartitioning of variance as being primarily interactive, with main effects computed as a residual. Fortunately, this computational solution was unstable, and as will be seen, adding any additional control variables into the model restored the main-effects pattern of results. Therefore, these results were dismissed as an anomaly.

Model 8 examined whether the observed pattern of effects for the differentiating affiliation variable would be accounted for by any other religion variable (examined individually).

TABLE 2. Childhood religious affiliation as a predictor of young adult offspring AD symptoms and as a mediator/moderator of family alcoholism history risk

Model	Variables	<i>p</i>
Model 1		
1a	Any childhood religious affiliation	.07
Model 2		
2a	Family alcohol history	.000*
2b	Family alcohol history	.79
	Any childhood religious affiliation	.10
	Family History $\times$ Religious Affiliation	.27
Model 3		
3a	Family alcohol history	.000*
3b	Family alcohol history	.81
	Accommodating religious affiliation	.23
	Catholic religious affiliation	.22
	Differentiating religious affiliation	.04*
	Family History $\times$ Accommodating Affiliation	.21
	Family History $\times$ Catholic Affiliation	.44
	Family History $\times$ Differentiating Affiliation	.33
Model 4		
4a	Family alcohol history	.000*
4b	Family alcohol history	.83
	Accommodating religious affiliation	.35
	Catholic religious affiliation	.33
	Differentiating religious affiliation	.08
	Family History $\times$ Accommodating Affiliation	.25
	Family History $\times$ Catholic Affiliation	.48
	Family History $\times$ Differentiating Affiliation	.40
	Religious attendance (age 6-13)	.21
4c	Family alcohol history	.85
	Accommodating religious affiliation	.32
	Catholic religious affiliation	.26
	Differentiating religious affiliation	.08
	Family History $\times$ Accommodating Affiliation	.26
	Family History $\times$ Catholic Affiliation	.47
	Family History $\times$ Differentiating Affiliation	.40
	Religious rules (age 6-13)	.17

Notes: Covariates are modeled in all analyses and include offspring gender, offspring age ≥ 18 , father's and mother's education ($\geq$ some college), family income in the top quartile, father's history of other psychiatric disorder, and mother's history of depression.

Bold indicates $p \leq .10$; * $p \leq .05$; † $p \leq .001$.

FIGURE 2. When examining *current* religious affiliation groups, offspring exhibit about the same rates of alcohol dependence [AD] symptom as seen for childhood affiliation groups. However, for nonreligious offspring (the reference group), offspring in the low-risk group exhibited a strong increase in mean AD symptoms. The largest contributors to the increase were those changing their affiliation from religious to nonreligious. Ref = reference group; NEG = negative; POS = positive; Hx = history; relig affil = religious affiliation.

This was tested by considering each of 10 additional religion variables. Furthermore, in adding additional control to the variance structure, these models more precisely repartitioned variance. Across all 10 tests, results indicated a main effects solution (see Table 3; Model 8b is shown as an example of findings obtained for all 10 tests, Models 8b through 8k). In every solution, results indicated that the differentiating affiliation main effect was robustly significant, even after controlling for each additional religious variable, some of which were also robustly (but independently) associated with offspring AD symptoms. The significant main effect for differentiating affiliations ($t < 0.52$, 1 df, $p > .60$), together with the removal of the family history of alcoholism effect in this model ($t > -2.5$, 1 df, $p < .01$), and the correlation between the differentiating affiliations variable and the predictor variable (family history risk: $r = -.12$, $p < .001$) and outcome variable (offspring AD symptoms: $r = -.17$, $p < .001$) indi-

cated that differentiating affiliation met criteria as a mediator of the association between family history of alcoholism risk and offspring AD symptoms. It should be noted that, in some models, accommodating and Catholic affiliations also exhibited significant main effects in predicting offspring AD symptoms. However, closer evaluation indicated that these affiliation types were not correlated with both the predictor and outcome variables and, therefore, were not functioning as mediating variables but as risk indicators. Also noteworthy is that accommodating affiliations showed a significant (or near significant) interaction effect across models; this effect reflected the exceptionally high rates of AD symptoms in the high-risk offspring group.

The last model in this series attempted to identify the strongest predictors among the full range of available religion variables; therefore, Model 9 provided an all-inclusive multivariate model that simultaneously partitioned variance

across all factors. Results confirmed that current differentiating religious affiliation was the strongest predictor of offspring AD symptoms. Further, given the significant correlations between the differentiating affiliation variable and both predictor (family history of alcoholism) and outcome (offspring AD symptoms) variables (see above), and the elimination of the family history of alcoholism effect in this model ($t = 0.05$, 1 df, $p = .96$), the mediation effect was also confirmed.

Because the dependent variable used here, offspring AD symptom count, had a distribution with elevated skew and kurtosis, additional analyses were conducted using corrective transformations of the dependent variable: a square root transformation and a natural log transformation. Analyses were rerun with both transformations and produced the same patterns of significant effects in each case, thus indicating that current findings were not the result of violations of the normality assumption. Untransformed means and statistics are reported here for ease of interpretation. As well, given that current data include multiple offspring from the same family, analyses were rerun while controlling for the non-independence of family members using STATA software (Release 7; StataCorp LP, College Station, TX). The pattern of findings were essentially identical to those reported here, thus indicating that observed effects were not the product of familial similarity among offspring.

Finally, although this national sample underrepresented those of African-American ancestry ($n = 63$ offspring, 4.2% of the sample) and conclusive results were not probable, it remained important to determine if data patterns in the current sample were consistent with those of the previous sample, specifically, that African-American church affiliations would most frequently be of the differentiating type and that the offspring of African Americans would exhibit a greater protective effect than the offspring of European ancestry ("white/other"). Examination of affiliation types by race showed that 66.7% of African Americans attended a differentiating religious affiliation (compared with 29.6% of the white/other sample). Concerning alcohol symptoms across religious and nonreligious groups, results showed that the AD symptom rates of African-American offspring were generally higher than white/other offspring. However, African-American offspring who endorsed any religious affiliation exhibited mean AD symptom rates that were about one third as high as their white/other counterparts in both the low-risk ($n = 24$) and high-risk ($n = 25$) groups. The highest rates were exhibited by white/other offspring who were nonreligious, both for the low-risk ($n = 351$) and high-risk ($n = 702$) groups. When differentiated by religious affiliation types, those having differentiating affiliations exhibited lower mean rates of AD symptoms compared with other affiliation types, and offspring AD symptom rates exhibited by African-American offspring were lower than white/other offspring rates for differentiating (low risk: $n = 14$; high risk:

TABLE 3. Current religious affiliation as a predictor of current offspring alcohol-dependence symptoms (AD sx) and as a mediator/moderator of family alcoholism history risk

Model	Variables	<i>p</i>
Model 5		
5a	Any current religious affiliation	.000‡
Model 6		
6a	Family alcohol history	.000‡
6b	Family alcohol history	.88
	Any current religious affiliation	.000‡
	Family History × Religious Affiliation	.03*
Model 7		
7a	Family alcohol history	.000‡
7b	Family alcohol history	.000‡
	Accommodating religious affiliation	.90
	Catholic religious affiliation	.90
	Differentiating Religious Affiliation	.28
	Family History × Accommodating Affiliation	.82
	Family History × Catholic Affiliation	.04
	Family History × Differentiating Affiliation	.004
Model 8 ^a		
8a	Family alcohol history	.000‡
8b	Family alcohol history	.93
	Accommodating religious affiliation	.02*
	Catholic religious affiliation	.06
	Differentiating religious affiliation	.000‡
	Family History × Accommodating Affiliation	.02*
	Family History × Catholic Affiliation	.37
	Family History × Differentiating Affiliation	.11
	Current religious attendance	.000‡
Model 9		
9a	Family alcohol history	.000‡
9b	Family alcohol history	.96
	Accommodating religious affiliation	.03*
	Catholic religious affiliation	.08
	Differentiating religious affiliation	.005†
	Family History × Accommodating Affiliation	.06
	Family History × Catholic Affiliation	.57
	Family History × Differentiating Affiliation	.34
	Religious attendance (age 6-13)	.05*
	Religious rules (age 6-13)	.03*
	Current religious attendance	.03*
	Religious rules (current)	.12
	Rely on religious teaching	.35
	Believe in God	.99
	Religious beliefs are a guide	.91
	Turn to prayer	.16
	Religious/spiritual self-rating	.15
	Time spent in religious activities	.18

Notes: Covariates are modeled in all analyses and include offspring gender, offspring age ≥ 18 , father's and mother's education ($\geq$ some college), family income in the top quartile, father's history of other psychiatric disorder, and mother's history of depression. ^aEach of 10 models, Models 8b through 8f, indicated a similar pattern of effects emphasizing a robust main differentiating effect.

Bold indicates $p \leq .10$; * $p \leq .05$; † $p \leq .01$; ‡ $p \leq .001$.

$n = 20$), accommodating (low risk: $n = 5$; high risk: $n = 4$), and Catholic (low risk: $n = 14$, high risk: $n = 1$) affiliation types. These findings were consistent with those of our previous study.

Discussion

Overview

This examination of the impact of religious affiliation on emerging alcoholism found that both low-risk and high-risk offspring (by virtue of parental history of alcoholism) who were raised with differentiating religious affiliations during childhood (ages 6-13 years) exhibited significantly lower AD symptoms in young adulthood, their period of highest risk. Further, and replicating our previous finding, childhood differentiating affiliation was shown to account for most of the affiliation effect that accounted for the relationship between family history of alcoholism and subsequent offspring AD symptoms. Our ability to draw causal inferences is strengthened by the temporal ordering of events in Study 1; that is, childhood religion may well influence subsequent young-adult alcohol behavior, but it is implausible that young-adult self-reported alcohol use will cause another reporter, their mother, to state that her child was raised with a certain religious affiliation. Thus, the direction of effects is from religious affiliation to alcohol behavior. Study 2 examined "current" young-adult religious affiliation and found a robust association between differentiating affiliation and offspring AD symptoms while also demonstrating that other religion variables did not account for the affiliation effect. In both studies, the main effect for differentiating affiliations together with the removal of the family history of alcoholism effect and the correlation between the differentiating affiliations variable and both family history risk and offspring AD symptoms established that differentiating religious affiliation was at least a partial mediator of the association between family history of alcoholism risk and offspring AD symptoms.

Concerning Study 1, results found that general childhood religious affiliation ("any religious affiliation") only produced a trend toward significance when examining young-adult behaviors. General affiliation appeared to have only a small influence on offspring AD symptoms during their period of highest risk (Model 1: $p = .07$) and only a modest influence as a mediator-moderator of the relationship between family history of alcoholism and offspring AD outcomes (Model 2: $p = .10$). Even so, these findings were noteworthy because (1) the impact on family history of alcoholism risk was large, in that a robust family history effect ($p = .000$) was reduced to nonsignificance ($p = .79$); (2) this was a very general affiliation predictor, and large averages tend toward nonsignificance; and (3) it was anticipated that childhood influences would be stronger in adolescence, as seen in our previous report (Haber and Jacob, 2007), and decline as children mature into young adulthood, as seen in the borderline effect sizes here. More important, when affiliation was more precisely defined as accommodating, differentiating, and Catholic affiliations (Model 3), dif-

ferentiating affiliation emerged as a significant mediator of family history risk as shown by (1) a significant main effect with offspring AD symptoms, (2) the removal of the family history of alcoholism effect, and (3) significant correlations with both predictor and outcome variables. Thus, differentiating religious affiliation appeared to be part of the causal pathway from parental history of alcoholism through family religious history, notably differentiating affiliation history to young-adult alcohol behavior. Other religion variables, when added to the model (Model 4), did not account for much of the variance associated with religious affiliation (although the modest effect size of the differentiating affiliation effect fell slightly below the significance threshold).

These findings need to be considered in the context of our earlier study (Haber and Jacob, 2007). Using an adolescent sample of female twins (mean age = 15.5 years), an "any affiliation" childhood variable was robustly associated with lower rates of AD symptoms. When examined by affiliation type, both differentiating and Catholic affiliations were significantly protective. However, rather than being main effects, these influences were significant interaction effects with family history of alcoholism. This is because most offspring of low-risk families (having parents with no alcoholism history) exhibited the same low-risk alcohol behavior, and therefore religion did not differentiate between these groups. Only high-risk offspring (those whose parents did have an alcoholism history) exhibited a broader range of AD symptoms, and in this group religious upbringing did differentiate offspring AD outcomes. Given the findings in the current study, it appears that the uniform low levels of offspring AD symptoms in all low-risk groups was the result of the young age of this sample. As children mature, increasing exposure and experimentation with alcohol occurs, and even low-risk offspring exhibit increases in alcohol use and AD symptoms. In the current older sample (mean age = 19.6 years), there is significant variation in both low-risk and high-risk offspring, indicating main effects (instead of interaction effects). The most important main effect was the consistent influence of differentiating religious affiliation on offspring outcomes.

Concerning Study 2 (see Table 3), current religious affiliation was examined as a proximal influence using this same sequence of models. The general "any religious affiliation" variable was associated with a markedly lower mean rate of AD symptoms in young adulthood (Model 5: $p = .000$) and was a robustly significant mediator of the relationship between family history of alcoholism and offspring AD outcomes (Model 6: $p = .000$). A significant interaction effect was also found, but this reflected an unanticipated increase in AD symptoms among low-risk nonreligious offspring, the reference group, rather than a religious group (see Figure 2). This change in the reference group was not problematic except in the next model (Model 7). In this case, when analyses characterized effects in terms of accommodating, dif-

ferentiating, and Catholic affiliation types (but without any extra religion variables to help control for excess interaction variance), this reference group elevation appeared as interaction variance that combined with accommodating affiliation interaction variance to overwhelm main effect variance and produce anomalous results. Although the anomaly was easily resolved (see Model 8) and was inconsistent with all other indicators, post hoc analyses revealed an unanticipated and important finding. Between childhood and young adulthood, a change occurred in the reference group (all nonreligious offspring). Although about 80% of offspring maintained the same religious (or nonreligious) affiliation from childhood to young adulthood, about 20% changed from religious to nonreligious (and *visa versa*). Although stable religious offspring exhibited significantly lower mean AD symptom rates compared with nonreligious groups, religious offspring who changed to nonreligious status were associated with high mean rates of AD symptoms, even compared with nonreligious offspring. Most important, among low-risk offspring (whose parents have no history of alcoholism), those raised with a religious affiliation who now self-report as nonreligious exhibit the highest AD symptom rates of all groups, and these additions to the stable, low-risk, nonreligious offspring group raised the low-risk reference group to the same elevation as the high-risk reference group (see Figure 2). The implication was that, for religious offspring, greater alcohol use was associated with a change to a nonreligious status. The reference group change, of course, did not imply the absence of main effects for affiliation groups but did explain what contributed to Model 7 results becoming anomalous.

Model 8 both corrected for the anomaly in Model 7 and confirmed the differentiating affiliation effect. When each of 10 explanatory religion variables were examined, main effects were confirmed, and the differentiating affiliation effect was again confirmed as a robust mediator of the association between family history of alcoholism and offspring AD symptoms because of (1) a robustly main effect across all models (from $p = .01$ to $p < .001$), (2) the removal of the family history effect across all models (from $p = .60$ to $p = .97$), and (3) robustly significant correlations with both predictor ($p = .001$) and outcome ($p < .001$) variables. Model 9, an all-inclusive multivariate model, confirmed that differentiating religious affiliation is a principal mediator in the pathway from family history of alcoholism risk to offspring alcohol outcomes.

It is important to keep the meaning of the differentiating construct in mind when considering these results. Following Johnson (1971) and Bainbridge and Stark (1980b), the differentiating construct was based on beliefs and behaviors held by a religious group that were in tension with generally accepted cultural norms (e.g., doctrines like the second coming of Christ, beliefs such as divine healing, and behaviors such as gambling). The implication was that if someone affiliates with a religious group that holds beliefs that are considerably

different from the beliefs held by others in the same culture, then they must be willing to differentiate themselves from cultural ideas and norms. Because the general culture of the United States is permissive or encouraging of alcohol use, groups of persons who are taught to differentiate themselves from cultural norms appear to be less likely to follow cultural norms and, in turn, are less likely to experience the associated effects. The current study and its predecessor examined the effect of religious differentiation (vs religious accommodation) on alcoholism behavior. It appears reasonable to conclude that religious differentiation may be a protective influence with respect to cultural pressures on adolescents and young adults to engage in alcohol-use behaviors. It appears that both high-risk youth (with parents having a history of alcohol-use disorder) and lower risk youth (those without family risk for alcoholism) are at reduced risk if they were raised with a differentiating religious affiliation or currently endorsed a differentiating religious affiliation compared with youth raised with or currently affiliated with another affiliation type or not religiously affiliated.

These two studies contribute to the alcoholism literature in several ways. Current findings robustly confirmed reports in the COA literature that reliably indicate that COAs are at increased risk for alcoholism (Russell, 1990; Windle, 1997), and that alcoholism risk is transmitted from parents to children (Hartman et al., 2006). Further, current findings support the finding that religious affiliation is a key inverse influence on the transmission of alcoholism risk from parents to children (Haber and Jacob, 2007). Current findings indicate that all offspring, both low and high risk, had significantly lower AD symptom counts when endorsing a differentiating affiliation, and that this factor met criteria as an inverse mediator variable, a finding strengthened by the fact that this family research design permitted stronger inferences regarding the causal direction of effects than other cross-sectional models. The conclusion here is that religious differentiating affiliation exerts a reliable, inverse, mediating, causal influence on the transmission of alcoholism risk from parents to children.

This finding also helped explain findings from previous studies of fundamentalist churches (Francis, 1994; Miller et al., 2000), "other Protestant" churches (Heath et al., 2003), and black churches (Brown et al., 2001) that have identified an inverse association between alcoholism and religious affiliation. Initial characterization of group demographics distinguished the differentiating affiliation group of this sample as having the lowest mean family income of all groups. As well, mother's (but not father's) education was significantly lower in the differentiating affiliations. Even so, despite the additional stress that would be associated with socioeconomic-status disadvantage and associated adverse conditions, families with differentiating religious affiliations had the lowest prevalence of paternal and maternal alcohol-use histories, and the offspring of these families had the lowest rates of reported AD symptoms. This effect was most clearly

illustrated by our examination of African-American families. Although our national sample underrepresented African-Americans ancestry and precluded conclusive statistical results, the findings here were entirely consistent with those of our previous report. African Americans in the current sample were much more likely to be affiliated with a differentiating affiliation, as was found before. As well, mean AD symptom rates of these religious African-American youth were consistently lower than religious white/other youth, as was found before. These results suggest that African-American youth experience a greater protective effect from their religion than is experienced by youth of European ancestry ("white/other"). The consistency of findings provides a convincing explanation of the previous finding that African-American adolescent girls have a later onset of alcohol use as a result of their religious affiliation (Heath et al., 1999a). As well, the increased endorsement of differentiating affiliation by lower-socioeconomic-status families in general suggests that differentiating affiliations are important and effective influences in the community toward positive health-promoting behavior, even in adverse and difficult circumstances.

Future research

Not only are these findings important to understanding the alcoholism-religion relationship per se, but they are important to the larger question of genetic risk factors that underlie alcoholism risk and development as well as environmental factors that mediate this genetic risk. Differentiating religious affiliation may represent an important instance of gene-environment interplay. Because parents transmit both genes and family environmental influences to their offspring, the study of twins and their families has the potential to clarify the role of genetic and environmental factors in the relationship between parental alcoholism and subsequent offspring alcoholism (Heath et al., 1997; McGue, 1997; Prescott and Kendler, 1999). As described by Heath et al. (2003): "the question of...genetic effects on risk of substance use disorders has been answered convincingly, ...[but] behavioral geneticists have been less successful in addressing the mediating-variable questions" (p. 325). The contention here is that differentiating religious affiliation may be an important exemplar in the quest to identify key mediators of genetic risk for alcoholism and the mechanisms that underlie this mediation. Identification of inverse mediators is all the more important because of their potential value to the treatment of this disorder, and thus, the study of the alcoholism-religion relationship is an important component of current alcoholism research.

Limitations

Although this replication raises confidence in the explanatory value of the differentiating religious affiliation

construct in accounting for alcoholism outcomes, there are several limitations in this inquiry that should be noted. Perhaps most important is that the differentiation construct is not measured directly but is inferred from an operationalization based on religious affiliations. It is well known that any single denomination (e.g., Baptist) may have branches that are more differentiating (e.g., Southern Baptist) and more accommodating (e.g., American Baptist). Despite sizable within-group variation, there are important social and psychological differences at the between-affiliation level that are captured in this generalized construct. Especially because variation in this socioreligious dimension contributes to the mediation of alcoholism risk, it is important to attempt direct measurement of this dimension to more precisely understand the nature of the effects being observed.

It should be noted that our analyses were limited to a single dependent variable, offspring AD symptoms. Differentiation effects may vary in relation to other measures of alcohol use, and more so for other substance and psychiatric disorders. These are all interesting and important future directions.

The most interesting future direction for this line of inquiry is toward genetic analyses that would examine the underlying genetic structure of alcoholism-affiliation covariation. It will be important to determine, for instance, whether this differentiating affiliation variable mediated genetic risk for alcoholism per se or mediated a correlated environmental risk factor. Consideration of such behavioral genetic questions is part of our current research plan.

By identifying this socioreligious dimension and its impact on alcohol behavior, a clearer understanding of this aspect of religious influence has emerged. Simply put, if a religious group accommodates cultural norms, and if those norms are permissive of alcohol use, then both the cultural milieu and the religious affiliation become unintentional risk factors for those susceptible to alcoholism risk. On the other hand, if a religious group differentiates itself from cultural norms (in the name of higher values, religious purity, or for other reasons), these alternative values may be protective for those susceptible to alcoholism risk and may inhibit development of alcohol-dependence outcomes. Clearly, this socioreligious variable warrants additional study.

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