A Multivariate Twin Study of the Dimensions of Religiosity and Common Psychiatric and Substance Use Disorders

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Abstract: The authors sought to decompose the covariance between seven dimensions of religiosity and two internalizing psychiatric disorders (major depression and phobia) and two externalizing substance use disorders (alcohol dependence and nicotine dependence). Significant negative correlations, accounted for by shared additive genetic effects, were found between alcohol dependence and six of the seven religiosity factors. Additive genetic effects accounted for significant negative correlations between nicotine dependence and one religiosity factor, social religiosity, and between phobia and unvengefulness. Common environmental effects accounted for a significant positive correlation between phobia and the factor God as judge. No statistically significant covariance due to genetic or environmental effects was found for major depression and any of the seven religiosity factors. Overall, although several statistically significant bivariate relationships were found, the estimates of covariance due to additive genetic effects were modest.

Key Words: Religiosity, twin studies, psychiatric disorders

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In recent years, there has been a renewed interest in the relationships between religiosity and mental and physical health. It remains largely unclear whether being religious influences health outcomes, whether certain health-related situations (*e.g.*, a diagnosis of terminal illness) lead persons to become more religious, or whether a third set of factors (such as genetic effects) is related to both religiosity and health (Smith et al., 2003). Further, as noted by Harden (2010), religiosity and health-related variables (*e.g.*, age of first alcohol use) are "… embedded in a complex matrix of genetic and environmental background factors" (p. 764).

Relationships Between Religiosity and Disorders

Relationships between religiosity and depression have been observed for many years, with associations between religious affiliation and depression reported as far back as the 1880s (Koenig et al., 2001). Smith et al. (2003) conducted a meta-analysis of 147 studies with more than 98,000 subjects. They found a small but robust negative association between religiousness and symptoms of depression, with moderating effects observed for stressful life events (there were stronger religiousness-depression associations in people who were undergoing stress) and type of religiosity measures used (Smith et al., 2003).

Although phobias are among the most common psychiatric disorders, relatively little has been written about associations between religiosity and phobia. Several studies have examined religiosity's relationship with broadly defined "anxiety," but few have studied clinically defined anxiety disorders, and fewer, still, have examined phobias specifically (Koenig et al., 1993; Shreve-Neiger and Edelstein, 2004). Results from these studies have been mixed, with most finding negative relationships between religiosity and anxiety, whereas some have found positive relationships or no relationship between religiosity and anxiety.

The relationship between religiosity and alcohol abuse has one of the longest histories of any health-related research on religiosity. In 1902, William James wrote in his famous book *The Varieties of Religious Experience*, "The sway of alcohol … is unquestionably due to its power to stimulate the mystical faculties of human nature, usually crushed to earth by the cold facts and dry criticisms of the sober hour. Sobriety diminishes, discriminates, and says no; drunkenness expands, unites, and says yes … It makes him for the moment one with truth. Not through mere perversity do men run after it" (James, 1902/1997, pp. 304–305). Certain aspects of religiosity have had a long history of being used in interventions to treat alcohol use problems, with the implicit idea that religiosity may influence alcohol use (Harden, 2010). For example, Alcoholics Anonymous, which was founded in the 1930s, has helped millions of people in their struggle with alcoholism and is, at its core, a spiritual program (Horstmann and Tonigan, 2000).

Studies have found negative associations between religiosity and alcohol use and abuse, with findings specific to the aspect of alcohol use measured and the dimensions of religiosity measured. For example, in a large, representative cross-sectional study of almost 3000 adults aged 18 to 97 years in the Piedmont region of North Carolina, Koenig (1999) found that recent (defined as "in the last 6 months") alcohol abuse and dependence were significantly lower among those who frequently engaged in prayer and scriptural study; recent and lifetime alcohol problems were lower among those who attended worship services; and those who attended religious services at least once a week had less than one third the rate of alcohol abuse compared with those who attended less frequently. Survey literature has reported hundreds of studies of religiosity and substance use, with most of these studies reporting inverse correlations between religiosity and various aspects of alcohol use (Geppert et al., 2007).

Religiosity has also been found to have inverse associations with cigarette smoking. However, results depend on the population studied and the specific aspects of smoking and religiosity examined. For example, a study of older adults in North Carolina found that those who frequently studied the Bible, attended religious services, or prayed privately were much less likely to smoke, and if they did smoke, they smoked fewer cigarettes than their less religious peers (Koenig, 1999). Hestick et al. (2001) found that, among African-American college students, those who regarded spirituality as important were less likely to have ever been lifetime regular smokers, but the importance of spirituality was not significantly related to having ever tried smoking.

Genetic and Environmental Effects on Religiosity

Behavior genetics methodology has been used to examine the heritability of various expressions of religiosity. As with nearly all psychological traits, at least a modest genetic influence on individual differences in various expressions of religiosity has been found, as

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well as common and unique environmental effects (Bouchard and McGue, 2003; Kendler et al., 1997; Koenig et al., 2005, 2007; Tsuang et al., 2002). Depending on what aspect of religiosity is being studied, genetic contributions have been found to range from very little, for example, in religious affiliation (D'Onofrio et al., 1999; Eaves et al., 1990), to moderate, in the case of the belief in God being directly involved in human affairs (Vance et al., 2010).

Genetic and Environmental Effects on Disorders

Past behavior genetics research has shown significant additive genetic and unique environmental effects on major depression (Sullivan et al., 2000). In a twin study using a sample from the Virginia Twin Registry, the heritability of major depression was identical in men and women, with additive genetic effects accounting for 39% of the variance and unique environmental effects accounting for the remaining 61% of the variance (Kendler and Prescott, 1999). A longitudinal study of female twins by Kendler et al. (1993) found that additive genetic effects on the liability for depression were stable over time, whereas environmental effects, which also play a significant role in liability for major depression, were occasion specific and transitory. A study of more than 42,000 twins from the Swedish Twin Registry found that additive genetic and unique environmental effects accounted for the variance in lifetime major depression, with significant differences in heritability in women compared with men (Kendler et al., 2006).

There have been relatively few behavior genetics studies of phobia. Overall, findings indicate that additive genetic effects account for approximately one third to two thirds of individual differences in phobias, with unique environmental effects accounting for the remaining portions of the variance (Hettema et al., 2001; Kendler and Eaves, 2005; Kendler et al., 1992, 2001, 1999).

Additive genetic effects have been found to play a significant role in liability for alcohol dependence (World Health Organization [WHO], 2004). In a population-based study of adult male twins, Prescott and Kendler (1999) found that additive genetic effects accounted for 48% to 58% of the variation in liability for alcohol abuse or dependence using *Diagnostic and Statistical Manual of Mental Disorders, Third Edition* (*DSM-III*), and *DSM-IV* criteria. It is less clear whether additive genetic effects have a strong influence on initiation of drinking and early alcohol use (Maes et al., 1999; Rose et al., 2001; WHO, 2004).

There are many behavioral aspects of smoking and stages of smoking progression that may be considered when examining the genetic and environmental influences on smoking. Among these are the behaviors of trying smoking at least once, becoming a regular smoker, and developing nicotine dependence. Additive genetic effects have been reported for each of these smoking behaviors (Maes et al., 2004; WHO, 2004). Studies have found that there are different genetic contributions to different smoking behaviors and only partial overlap in the sets of genetic factors that contribute to these behaviors (Kendler et al., 1999).

Genetic and Environmental Relationships Between Religiosity and Disorders

Several behavior genetics studies have examined the relationships between religiosity, psychiatric disorders, and substance use (D'Onofrio et al., 1999; Kendler et al., 1999; Maes et al., 1999; Tsuang et al., 2002). In a study of adolescent twins, D'Onofrio et al. (1999) found that belief that drug use is sinful was largely accounted for by environmental influences, although genetic influences could not be ruled out, and there were modest negative relationships between dimensions of adolescent religiosity and substance use. Kendler et al. (1997) studied relationships between religiosity, psychopathology, and substance use in a sample of female twins. The authors found three religiosity factors they called personal devotion, personal conservatism, and institutional conservatism. The religiosity factors had stronger relationships with substance use and dependence than with psychiatric symptoms and disorders. In a follow-up study using the same sample of female twins, Kendler et al. (1999) examined relationships between religiosity, psychiatric illness, and substance use and found that religiosity was related to low risk for symptoms of depression and substance use. Maes et al. (1999) examined religious attendance and frequency of alcohol use. A significant negative association was observed between frequency of church attendance and alcohol use. The authors found that genetic factors primarily accounted for the relationship between alcohol use and church attendance in males, whereas shared environmental factors more strongly accounted for this relationship in females. Tsuang et al. (2002) studied adult male twins and found that existential well-being and spiritual well-being were negatively associated with alcohol abuse and dependence.

Behavior genetics methods can also be used to examine shared genetic and environmental effects on religiosity and various aspects of mental health. For example, Koenig et al. (2007) found that the relationship between religiousness and antisocial behavior was due to genetic effects and common environmental effects shared by religiousness and antisocial behavior. However, the same study found that although the relationship between religiousness and altruistic behavior was largely due to shared genetic effects, only approximately half of common environmental effects were shared by religiousness and altruistic behavior (Koenig et al., 2007).

In a previous article by the authors (Vance et al., 2010), genetic and environmental influences on multiple dimensions of religiosity were reported. We reported seven religiosity factors identified as general religiosity, social religiosity, involved God, forgiveness, God as judge, unvengefulness, and thankfulness. Genetic and unique environmental effects largely accounted for the variance observed across the religiosity construct in the population studied. Two exceptions were found for the factors social religiosity and God as judge, in which modest contributions from shared environmental effects were observed.

Multivariate genetic analyses found that the seven religiosity factors were influenced by one common additive genetic factor, three common unique environmental factors, and unique environmental effects specific to each religiosity factor. We interpreted these results to mean that, for the population studied, a common genetic effect influenced the predisposition to become religious, whereas unique environmental effects shaped the specific expression of religiosity.

In the present, related study, we sought to decompose the covariance between different dimensions of religiosity and internalizing and externalizing psychiatric disorders. Specifically, we examined genetic and environmental effects on seven previously identified religiosity factors (Vance et al., 2010) as they related to two internalizing disorders, major depression and phobia, and two externalizing disorders, alcohol dependence and nicotine dependence.

Given the presence of genetic and environmental influences on psychiatric disorders, substance use disorders, and religiosity, decomposing the covariance among these should yield insight into shared genetic and environmental factors. In the present study we sought to examine the sources of covariation between different seven dimensions of religiosity previously identified by the authors (Vance et al., 2010) and risk for major depression, phobia, alcohol dependence and nicotine dependence.

METHODS

Participants and Sample Characteristics

The sample for the present study comes from two related projects that recruited participants from the population-based Virginia Twin Registry. Female-female twin pairs born between 1934 and 1974 were initially interviewed beginning in 1988. Male-male and

male-female twin pairs born between 1940 and 1974 were initially interviewed beginning in 1993. In 1999, prior participants in these two studies (N = 7230) were mailed questionnaires that contained the religiosity measures included in the present study. Of those who were mailed the questionnaires, 2621 were returned, for a 36.3% response rate. Resources for follow-up were limited. Data collection was approved by Virginia Commonwealth University's Institutional Review Board, and informed consent was obtained before receiving the mailed questionnaires. Zygosity was determined by standardized techniques that have been shown to have an accuracy rate of greater than 95% and were validated using molecular methods (Kendler et al., 2003).

All participants in the present study were white, and 58% were women. The mean age of the participants was 43.06 years; SD, 8.61; range, 27 to 63 years. More than three quarters (77%) of the participants were Protestant (see Table 1), and the most common denominational preference was Baptist. Table 2 shows the 10 most frequently endorsed denominations, which account for more than 88% of all responses.

Twin Study Methodology

Twin studies are designed to take advantage of the fact that monozygotic (MZ) twins share 100% of the genes, whereas dizygotic (DZ) twins share, on average, 50% of their genes identical by descent and are no more alike genetically than nontwin siblings. Therefore, differences in MZ twins provide evidence of environmental effects, whereas differences in DZ twins can result from the effects of genes and/or the environment (Bulik et al., 2000). Sources of variance identified in the present twin study include additive genetic effects (A), which reflect the cumulative effect of many individual genes; common environmental effects (C), which are environmental effects shared by twins and which make twins more alike; and unique environmental effects (E), which reflect either environmental effects that affect twins differently and make them different from one another and/or errors of measurement (Bulik et al., 2000; Loehlin, 1989; Scarr, 1997).

Measures

Religiosity

As discussed previously (Kendler et al., 2003; Vance et al., 2010), because the constructs of religiosity and spirituality are ill defined, a broad selection of items measuring religiosity, spirituality, and related attitudes such as forgiveness and gratitude were used in the present study. The questionnaires included 78 total items measuring various aspects of religiosity. These items were taken from various sources and submitted to factor analysis to yield seven religiosity factors (Vance et al., 2010), which were used in the present study.

Internalizing Psychiatric Disorders

The common internalizing disorders of major depression and phobia were examined in the present study. Major depression was assessed using an adaptation of the Structured Clinical Interview for *DSM-III-R*. Phobia was defined following *DSM-III* criteria as an irrational fear with objective behavioral impact on the respondent's behavior, as judged by a trained interviewer (Kendler et al., 2003).

TABLE 1. Religious Preference, n = 2621

Religious Preference	%	*n*
Protestant	77.44	1988
No preference	9.35	240
Catholic	9.23	237
Other	2.88	74
Jewish	1.09	28
Missing data		54

TABLE 2. Protestant Denominational Preference, n = 2621

Denominational Preference	%	*n*
Baptist	36.29	732
Methodist	21.02	424
Presbyterian	9.12	184
Episcopal, Anglican, Church of England	6.94	140
Pentecostal, Assemblies of God	3.47	70
Lutheran	3.27	66
Nondenominational Protestant	2.97	60
"Christian"	2.93	59
Church of God, Holiness	2.23	45
Other Protestant denominations	11.76	237
Missing, no preference, or non-Protestant		604

Substance Abuse Disorders

Nicotine dependence and alcohol dependence were examined in the present study. Nicotine dependence was defined as a score of 7 or greater on the Fagerstrom Tolerance Questionnaire (Fagerstrom and Schneider, 1989) as assessed during the heaviest period of lifetime use. Alcohol dependence was assessed using an adaptation of the Structured Clinical Interview for *DSM-III-R* (Kendler et al., 2003).

Statistical Analysis

Factor Analysis and Univariate Analysis

Briefly, the authors re-created seven religiosity factors previously reported by Kendler et al. (2003) using confirmatory factor analysis techniques (see Vance et al., 2010). The seven factors were identified as general religiosity, social religiosity, involved God, forgiveness, God as judge, unvengefulness, and thankfulness (Kendler et al., 2003; Vance et al., 2010). The general religiosity factor included 30 items reflecting concern and involvement with spiritual issues and with God. Social religiosity was composed of 12 items tapping the social aspects of religiosity. Involved God included 6 items reflecting belief in God and in God's active involvement in human affairs. Forgiveness was made up of seven items reflecting a loving, caring, and forgiving approach to the world. God as judge included seven items indicating a view of a judgmental and punitive deity. The sixth factor, called unvengefulness, was composed of eight items reflecting an attitude opposed to taking personal retaliation. The final factor, called thankfulness, included four items reflecting an attitude of thankfulness.

Genetic and environmental contributions to variance were estimated for religiosity factor scores (see Vance et al., 2010). Univariate analyses were also conducted on the four psychiatric disorders of interest in the present study (see Vance et al., 2010, for detailed description of univariate analyses).

Bivariate Analysis

The goal of bivariate analysis is to decompose the covariance between two traits of interest (Kendler et al., 1992). Bivariate analyses were conducted to decompose the covariance between seven different dimensions of religiosity and psychiatric and substance use disorders into genetic and environmental effects. Likelihood ratio tests, the chi-square difference test, and Akaike's information criterion (AIC) were used to determine the best-fitting models, with lower values of the AIC indicating models with a better balance of explanatory power and parsimony (Kendler et al., 1993; Kendler and Meyers, 2009). Parameter estimates (a^2, c^2, and e^2) and 95% confidence intervals (CIs) were estimated. Where appropriate, both full ACE models and best-fitting submodels were reported, along with 95% CIs for each parameter in each model.

All bivariate analyses were conducted using the Mx program (Neale et al., 2005). Excessive computer run times made simultaneous, multivariate analysis of all seven religiosity factors and each psychiatric disorder untenable (Hettema et al., 2006; Neale, 2003). Bivariate analyses made computer run times manageable and yielded results that were readily interpretable. Factor scores for the religiosity variables were converted into deciles so that ordinal-level data could be used for analysis of both variables. Cholesky's decompositions were used to estimate the covariance between the religiosity factors and the psychiatric disorders. For each factor and disorder, analysis began with a full Cholesky's model including the latent variables *A*, *C*, and *E*. A graphic representation of a bivariate Cholesky's decomposition is shown in Figure 1.

RESULTS

Representativeness of Sample

As previously reported by Kendler et al. (2003), participation in this study was predicted by female sex, increasing age, increasing education, and monozygosity. These predictors of participation are similar to those reported by others who have examined nonresponse in twin studies using questionnaires (*e.g.*, Heath et al., 2001). Even with poor response rates, previous empirical studies of this problem have reported that response bias is minimal (Heath et al., 2001; Vink et al., 2004). Participation was not predicted by level of religiosity. The generalized linear model procedure in statistical analysis system (SAS Software, Version 9, SAS Institute, Inc, Cary, NC) was used to determine whether any psychiatric disorders predicted participation in the sample used in the present study (Kendler et al., 2003). Logistic regression showed that, of the four psychiatric disorders examined in the present study, only nicotine dependence was a significant predictor of participation. Although those who were nicotine dependent were less likely to participate in this study, the overall size of this effect was quite modest (odds ratio, 0.85; 95% CI, 0.74–0.99; $p = 0.0448$). Extensive analyses were conducted by the authors, who concluded that the sample is probably representative of twins who participated in earlier interview waves from which the data for this study were collected (Kendler et al., 2003).

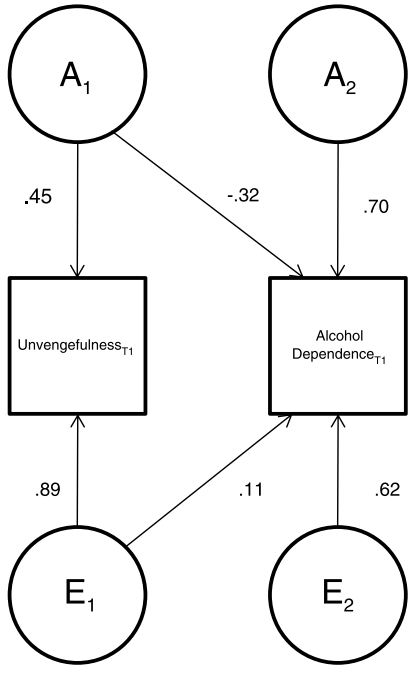

FIGURE 1. Bivariate Cholesky's decomposition with two phenotypes (unvengefulness and alcohol dependence) and additive genetic factors (*A*) and unique environmental factors (*E*). Phenotypes from the first twin (T1) are correlated with phenotypes of the second twin (not shown).

Bivariate Analysis

Bivariate analyses were conducted to estimate the covariance between the seven religiosity factors and two psychiatric disorders, major depression and phobia, and two substance use disorders, alcohol dependence and nicotine dependence. With one exception, the results showed that the latent variable *C* (common environmental effects) was not significant and could be dropped from the models (see Table 3). Subsequent analyses included only an AE model for each religiosity factor and psychiatric disorder. The one exception to this finding was for the relationship between phobia and the religiosity factor God as judge. In this case, bivariate analyses were conducted with an ACE model.

Bivariate analyses of the seven religiosity factors and internalizing and externalizing psychiatric disorders found significant negative correlations between alcohol dependence and six of the seven religiosity factors. These correlations could be accounted for by shared additive genetic factors. Similarly, additive genetic factors accounted for the significant negative correlation between nicotine dependence and one religiosity factor, social religiosity, and between the religiosity factor unvengefulness and phobia. Unique environmental effects were not a significant source of covariance for any of the religiosity factors and psychiatric and substance use disorders. There was no statistically significant covariance due to additive genetic effects or unique environmental effects for major depression and the seven religiosity factors. Correlations for additive genetic effects are summarized in Table 4. Correlations for unique environmental effects are summarized in Table 5.

A portion of the covariance between the religiosity factor God as judge and phobia could be accounted for by common environmental effects. Model comparisons showed that the latent variable *C* (common environmental effects) could not be dropped without a significant loss in model fit (see Table 3). Submodels were tested to determine the significance of correlations due to additive genetic effects, common environmental effects, and unique environmental effects, and 95% CIs were estimated for each parameter. As shown in Table 6, both additive genetic effects and common environmental effects accounted significantly for the correlation. The correlation due to unique environmental effects was not statistically significant. Parameter estimates and 95% CIs are given in Table 7, and the model is represented graphically in Figure 2.

DISCUSSION

The results from the present study were in some ways consistent with what would be expected from previous literature, in that inverse relationships were observed between alcohol dependence and nicotine dependence and several of the religiosity factors (Kendler et al., 1997, 2003). For six of the seven religiosity factors, there were statistically significant negative correlations between religiosity and alcohol dependence, accounted for by additive genetic factors. Similarly, additive genetic factors accounted for the negative correlation between the factor social religiosity and nicotine dependence. Unique environmental factors did not account significantly for any of the relationships between alcohol dependence and nicotine dependence and the seven religiosity factors.

These results indicate that, genetically, the predisposition to become religious is inversely related to a predisposition toward alcohol dependence. In the most conservative interpretation, we can say that common factors, most likely additive genetic factors, account for the association between the religiosity factors and alcohol

TABLE 3. Comparison of Full ACE Model and AE Model, Bivariate Analysis of Seven Religiosity Factors and Four Psychiatric Disorders

Factor	−2LL Full ACE Model	−2LL AE Submodel	$\Delta\chi^2$ (*df*)	AIC	*p*
General religiosity					
Major depression	14998.8	14999.38	0.58 (3)	−5.41	0.90
Phobia	14681.84	14682.44	0.60 (3)	−5.40	0.90
Alcohol dependence	13963.41	13963.41	0 (3)	−6.00	Incalc
Nicotine dependence	13687.8	13687.8	0 (3)	−6.00	Incalc
Social religiosity					
Major depression	14971.04	14973.77	2.73 (3)	−3.27	0.43
Phobia	14658.3	14660.24	1.94 (3)	−4.06	0.58
Alcohol dependence	13920.28	13921.9	1.62 (3)	−4.37	0.65
Nicotine dependence	13659.94	13661.56	1.62 (3)	−4.37	0.65
Involved God					
Major depression	15020.58	15020.58	0 (3)	−6.00	1.00
Phobia	14703.39	14703.39	0 (3)	−6.00	1.00
Alcohol dependence	13984.14	13984.14	0 (3)	−6.00	Incalc
Nicotine dependence	13718.84	13718.84	0 (3)	−6.00	Incalc
Forgiveness					
Major depression	15118.32	15118.32	0 (3)	−6.00	Incalc
Phobia	14801.22	14801.32	10 (3)	−5.90	0.99
Alcohol dependence	14107.39	14107.39	0 (3)	−6.00	Incalc
Nicotine dependence	13817.59	13817.59	0 (3)	−6.00	Incalc
God as Judge					
Major depression	15093.51	15101.11	7.59 (3)	1.59	0.06
Phobia	14697.48	14708.91	11.43 (3)	5.43	0.01*
Alcohol dependence	14014.88	14019.67	4.79 (3)	−1.21	0.19
Nicotine dependence	13733.76	13738.97	5.21 (3)	−0.79	0.16
Unvengefulness					
Major depression	15207.98	15207.98	.001 (3)	−5.99	1.00
Phobia	14886.36	14886.36	0 (3)	−6.00	Incalc
Alcohol dependence	14208.76	14208.76	0 (3)	−6.00	Incalc
Nicotine dependence	13921.53	13921.63	10 (3)	−5.90	0.99
Thankfulness					
Major depression	15116.93	15116.93	0 (3)	−6.00	Incalc
Phobia	14847.88	14847.88	0 (3)	−6.00	Incalc
Alcohol dependence	14132.43	14132.43	0 (3)	−6.00	Incalc
Nicotine dependence	13855.47	13855.47	0 (3)	−6.00	Incalc

*$p < 0.05$.
–2LL indicates mins 2 log-likelihood; Incalc, incalculable.

dependence in the present study. These results suggest that the relationship between alcohol dependence and religiosity is not causal but due to an underlying set of genetic factors that predispose one to both low religiosity and increased risk for alcohol dependence. As Harden (2010) has noted, genes for religiosity are highly unlikely, and what makes an activity religious is defined by culture, not biology.

More speculatively, it may be surprising that environmental effects do not account significantly for the association between

TABLE 4. Additive Genetic Correlations, Religiosity Factors, and Psychiatric Disorders

	Major Depression	Phobia	Alcohol Dependence	Nicotine Dependence
General religiosity	0 (−0.09 to 0.09)	0.02 (−0.08 to 0.12)	−0.21*** (−0.25 to −0.10)	−0.10 (−0.20 to 0.02)
Social religiosity	−0.06 (−0.15 to 0)	−0.02 (−0.05 to 0.07)	−0.27*** (−0.37 to −0.24)	−0.14* (−0.24 to −0.03)
Involved God	−0.04 (−0.13 to 0.05)	0.04 (0.04 to 0.14)	−0.19** (−0.29 to −0.18)	−0.08 (−0.20 to 0.03)
Forgiveness	−0.01 (−0.03 to −0.01)	−0.01 (−0.14 to 0.10)	−0.18* (−0.32 to −0.04)	−0.10 (−0.23 to −0.02)
God as Judge	0 (−0.09 to 0.09)	0.12* (0.01 to 0.18)	−0.11 (−0.22 to 0)	−0.02 (−0.13 to −0.02)
Unvengefulness	0 (−0.19 to 0.20)	−0.33** (−0.59 to −0.12)	−0.32** (−0.61 to −0.09)	−0.05 (−0.30 to 0.19)
Thankfulness	−0.04 (−0.17 to −0.01)	−0.04 (−0.06 to 0.10)	−0.31*** (−0.46 to −0.15)	−0.14 (−0.28 to 0.02)

*$p < 0.05$; **$p < 0.01$; ***$p < 0.0001$.

TABLE 5. Unique Environmental Correlations, Religiosity Factors, and Psychiatric Disorders

	Major Depression	Phobia	Alcohol Dependence	Nicotine Dependence
General religiosity	0.04 (−0.07 to 0.15)	0.01 (−0.11 to 0.13)	−0.03 (−0.16 to 0.02)	−0.11 (−0.24 to 0.01)
Social religiosity	−0.02 (−0.13 to 0.09)	−0.03 (−0.15 to 0.09)	0 (−0.11 to 0.03)	−0.09 (−0.21 to 0.04)
Involved God	0.05 (−0.06 to 0.16)	−0.04 (−0.16 to 0.08)	−0.06 (−0.19 to −0.02)	−0.06 (−0.18 to 0.07)
Forgiveness	0.04 (−0.03 to 0.07)	−0.01 (−0.12 to −0.01)	0.04 (0.01 to 0.13)	−0.04 (−0.16 to 0.05)
God as Judge	0 (−0.11 to 0.11)	−0.06 (−0.18 to 0.06)	−0.04 (−0.12 to 0.04)	0 (−0.04 to 0.12)
Unvengefulness	−0.08 (−0.18 to 0.02)	0.10 (0.01 to 0.20)	0.11 (0 to 0.23)	−0.02 (−0.13 to 0.09)
Thankfulness	0 (0 to 0.11)	−0.03 (−0.04 to 0.05)	0.02 (−0.10 to 0.13)	−0.07 (−0.17 to 0.04)

All covariances are nonsignificant.

religiosity and alcohol dependence, given widely held beliefs about the life-changing effects of becoming religious either through mystical religious experiences (James, 1902/1997) or through spiritually oriented programmatic experiences such as Alcoholics Anonymous (Royce and Scratchley, 1996). Further, others have noted that the perceived beneficial influence of religiosity on alcohol and substance use is reflected in current government policy (Harden, 2010).

Somewhat surprisingly, no statistically significant relationships were observed in the covariance between major depression and the seven religiosity factors. Modest inverse relationships between major depression and religiosity have been observed in past studies (Kendler et al., 2003; Smith et al., 2003). In a previous study using the present sample, Kendler et al. (2003) found overall phenotypic associations between several religiosity factors and major depression. In the present study, the correlations between religiosity factors and additive genetic and unique environmental effects for major depression were essentially zero, showing that, although overall phenotypic associations may be significant, when the variance is decomposed into additive genetic, common environmental, and unique environmental effects, none are significant by themselves.

Interestingly, two religiosity factors had significant correlations with phobia. The correlation due to additive genetic effects between the religiosity factor unvengefulness and phobia was negative, suggesting that the genes that predispose an individual not to seek revenge are also protective against developing phobia. The relationship between the religiosity factor God as judge and phobia was the only bivariate relationship in which common environmental effects accounted significantly for the observed covariance. Previous literature has found that negative religious coping, including appraisals of a punishing God (*e.g.*, "Wondering what I did for God to punish me"), is related to negative psychological health outcomes, including increased anxiety and phobic anxiety (McConnell et al., 2006; Miller, 1998; Pargament, 2002; Pargament et al., 2004). However, some authors of studies that have observed relationships between negative religious coping and anxiety have suggested that other variables, such as cultural and ethnic factors (*e.g.*, religious preference, ethnicity, nationality), may play a role in these relationships (Chapman and Steger, 2010; Zwingmann et al., 2008).

For the factor God as judge, the covariances due to additive genetic effects and common environmental effects were in opposite directions. The covariance due to additive genetic effects was positive, whereas the covariance due to common environmental effects was negative. This suggests that common environmental effects shared by God as judge and phobia may attenuate or cancel out shared additive genetic effects that predispose one to both believe in a punitive and judgmental deity and to develop phobia.

The findings from this study must be considered within the context of methodological limitations. First, as discussed by the authors in a previous, related study (Vance et al., 2010), results should be interpreted in the context of the limitations of classic twin studies. These include power limitations, which typically affect twin studies' ability to detect common environmental effects. A second limitation concerns the response rate for the questionnaires from which this study's data were obtained. As described previously in a related study (Kendler et al., 2003), given the modest response rate, the representativeness of our sample is questionable. Age, sex, years of education, and zygosity had strong effects on participation. Kendler et al. (2003) conducted extensive analyses and concluded that, regarding the relationship between religiosity and psychopathology, the sample is probably representative of twins who participated in earlier interview waves from which this study's data were obtained (see Kendler et al., 2003, for detailed description).

Further studies of shared genetic and environmental effects on different dimensions of religiosity and psychopathology are needed to increase confidence in this study's findings. We used seven factors

TABLE 6. Model Comparisons, Bivariate Analysis of God as Judge, and Phobia

	−2LL	χ^2 (*df*)	AIC	*p*
Full ACE model	14697.48			
Drop COV_A	14707.60	10.12 (1)	8.12	0.001**
Drop COV_C	14703.71	6.23 (1)	4.23	0.01*
Drop COV_E	14700.04	2.56 (1)	0.56	0.11
Drop $COV_{A,C}$	14708.40	10.92 (2)	6.92	0.004**

*$p < 0.05$; **$p < 0.01$.
COV indicates covariance.

TABLE 7. Bivariate Analysis of God as Judge and Phobia

	God as Judge	Correlation	Phobia
Additive genetic effects	0.60 (0.43 to 0.73)	0.49** (0.19 to 0.63)	0 (−0.61 to 0.61)
Common environmental factors	0.50 (0.31 to 0.58)	−0.35* (−0.49 to −0.10)	0 (−0.41 to 0.18)
Unique environmental effects	0.62 (0.57 to 0.66)	−0.10 (−0.11 to 0.02)	0.79 (0.69 to 0.88)

*$p < 0.05$; **$p < 0.01$.

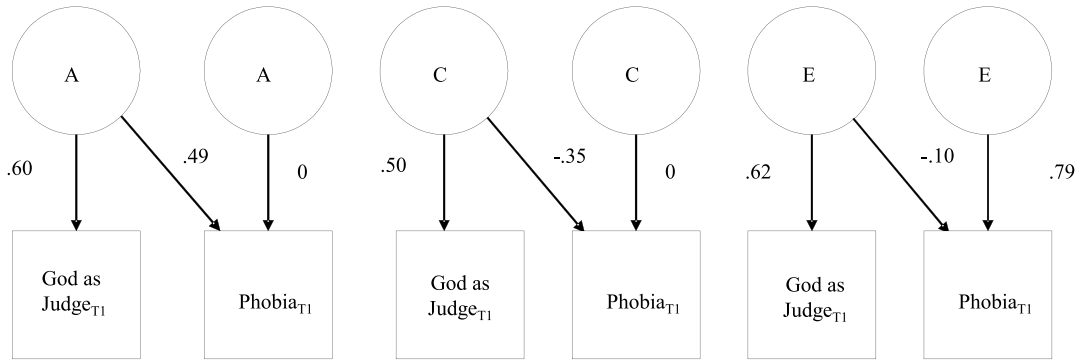

FIGURE 2. Bivariate relationships God as judge and phobia. Bivariate Cholesky's decomposition with two phenotypes (God as judge and phobia) and additive genetic factors (*A*), common environmental factors (*C*), and unique environmental factors (*E*). Phenotypes from the first twin (T1) are correlated with phenotypes of the second twin (not shown).

tapping many behavioral and attitudinal aspects of religiosity. In some cases, our results indicate that there may be other genetic factors that influence the variables we studied, and phenotypic relationships do not necessarily indicate shared covariance when genetic and environmental effects are examined. Additional multivariate studies with other samples would help elucidate the reasons for these findings.

CONCLUSIONS

In conclusion, this study sought to decompose the covariance between seven religiosity factors and internalizing and externalizing psychiatric disorders. Using seven previously identified religiosity factors (see Vance et al., 2010), we found statistically significant, negative correlations between alcohol dependence and six of the seven religiosity factors. These correlations were accounted for by additive genetic effects. Additive genetic effects also accounted for a significant negative correlation between nicotine dependence and the factor social religiosity. In contrast to previous studies, no significant correlations were observed for major depression and any of the seven religiosity factors. In addition to additive genetic effects, common environmental effects accounted for a significant correlation between phobia and the factor God as judge.

If there were causal relationships between religiosity and psychiatric and substance use disorders, the covariance would expectedly be largely environmental. The results of the present study do not support causal relationships between religiosity and psychiatric and substance use disorders but rather suggest a genetically influenced shared temperamental process. This finding is in some ways inconsistent with results reported in other studies that have found correlations between religiosity and substance use behaviors to be largely due to shared environmental factors (Harden, 2010; Kendler and Meyers, 2009). However, these previous results were specific to adolescents. In adulthood, genetic factors have been reported to account for the correlation between religiosity and substance use (Kendler and Meyers, 2009).

Overall, although statistically significant in several bivariate relationships, the estimates of covariance due to additive genetic effects were modest. Therefore, although there are genetic effects shared by the religiosity factors and psychiatric disorders examined in the present study, there are many other variables that have stronger bivariate relationships with these psychiatric disorders (*e.g.*, Kendler et al., 1993). The present findings also support previous conclusions by authors who have noted that the relationships between religiosity and health are complex and modest (Thoresen and Harris, 2002). Further behavior genetics studies examining genetic and environmental covariance between religiosity, psychiatric disorders, and substance use disorders are needed to increase confidence in the findings of this study.

DISCLOSURE

The authors declare no conflict of interest.

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