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ORIGINAL RESEARCH

The Etiology of Stability and Change in Religious Values and Religious Attendance

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Abstract Studies have demonstrated little to no heritability for adolescent religiosity but moderate genetic, shared environmental, and nonshared environmental influences on adult religiosity. Only one longitudinal study of religiosity in female twins has been conducted (Koenig et al., Dev Psychol 44:532–543, 2008), and reported that persistence from mid to late adolescence is due to shared environmental factors, but persistence from late adolescence to early adulthood was due to genetic and shared environmental factors. We examined the etiology of stability and change in religious values and religious attendance in males and females during adolescence and early adulthood. The heritability of both religious values and religious attendance increased from adolescence to early adulthood, although the increase was greater for religious attendance. Both genetic and shared environmental influences contributed to the stability of religious values and religious attendance across adolescence and young adulthood. Change in religious values was due to both genetic and nonshared environmental influences specific to early adulthood, whereas change in religious attendance was due in similar proportions to genetic, shared environmental, and non-shared environmental influences.

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Religiosity (a heterogeneous term which includes, but is not limited to, chosen religion, religious beliefs, religious behavior, and attitudes towards religion) is related to a range of behavioral and psychiatric outcomes. For instance, studies consistently show that religiosity is a protective factor for antisocial behavior (Herrenkohl et al. 2005; Koenig et al. 2007) and alcohol and drug use (D'Onofrio et al. 1999b; Heath et al. 1999; Kendler et al. 2003), as well as predictive of more prosocial behavior (Norenzayan and Shariff 2008; Koenig et al. 2007). Furthermore, religiosity or, more specifically, religious attendance, has also been shown to be associated with lower rates of premature mortality (Gillum et al. 2008; McCullough et al. 2000; Musick et al. 2004) and a positive general well-being (Maselko and Kubzansky 2006; Moreira-Almeida et al. 2006).

Religiosity generally decreases from adolescence to adulthood (e.g. Stolzenberg et al. 1995; Ueker et al. 2007), and then increases slightly throughout adulthood (Argue et al., 1999). The decline in religiosity from adolescence to emerging adulthood appears to be phenotype specific; for example, Ueker et al. (2007) found that a greater decline of approximately 70% for religious attendance compared with only approximately 20% for importance of religion. Contributing factors for the large decline in religious attendance include cohabitation outside of marriage, disrupted cohabitation, and drinking and drug taking, whereas persistence (and even an increase in religious attendance) is found in those who become married and those who have children (Stolzenberg et al. 1995; Ueker et al. 2007). Similarly, family factors are associated with a decline in

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the importance of religion (e.g. cohabitation is a risk for decline whereas marriage and children may even increase importance of religion), but behavioral factors (e.g. drug use) do not appear to have a robust impact on the importance of religion from adolescence to adulthood.

Studies pertaining to the heritability of religiosity in adolescents show little evidence for a genetic contribution, with environmental influences accounting for the majority of the observed variance in religiosity. For instance, a study of 4776 Finnish adolescents reported a small and non-significant heritability of 11% for 16-year-old females, and a heritability of 22% for 16-year-old males, for religious fundamentalism (Winter et al. 1999), whereas shared environment explained 60% of the variance in females and 45% of the variance in males. Given that the participants in these studies are at an age where parents are likely to encourage their own religious activities and attitudes in their children, this finding is unsurprising. This is further evident in a study of adolescent Dutch twins, which showed that correlations for three religiosity measures (religious upbringing, current religiosity, and participation in religious activities) were almost 1.0 for both identical and fraternal twin pairs (Boomsma et al. 1999), indicating that almost all of the variance in these three religious phenotypes was due to shared environmental influences. Low heritability estimates were further confirmed in a study of religiousness (consisting of both behavioral items and items assessing the importance of religion in decision making), assessed in adult American males reporting about religiosity in adolescence, which yielded a non-significant heritability estimate of 12% (Koenig et al. 2005), and a study of religiousness in American females at ages 14 and 18, which reported heritability estimates of 2% (non-significant) and 21%, respectively (Koenig et al. 2008).

Studies of adult religiosity generally report higher heritability estimates for religious attitudes, behavior, and religious attendance, although not for affiliation, which appears to be due largely to cultural factors (D'Onofrio et al. 1999a). A number of studies of religiosity have utilized the Virginia 30000 dataset, using complex genetic models that test for additive and non-additive genetic effects, assortative mating, and environmental transmission. These consistently demonstrate a moderate to high heritability for whatever religious phenotype they are investigating (Maes et al. 1999; D'Onofrio et al. 1999a; Truett et al. 1994; Eaves et al. 1999). In general, heritability estimates of religiosity scores in adults range from approximately 27% (Koenig et al. 2008) to 62% (Eaves et al. 1999), depending on age, sex, and specific phenotype. The extent to which the magnitudes of genetic and environmental influences differ across sex varies across study. Although many studies report a higher heritability for females (Truett et al. 1992, 1994; Kirk et al. 1999), others report a higher heritability for males (Winter et al. 1999), while others report no significant difference (D'Onofrio et al. 1999b).

Despite a number of studies on the etiology of religiosity, there are still few studies of the genetic and environmental influences on the development of religiosity; however, two studies have examined the etiology of change in religiosity from adolescence to adulthood utilizing genetically informative data. The first of these studies assessed religiousness in an adult male twin sample, where religiousness in adolescents was collected retrospectively and current scores were collected for religiousness in adulthood (Koenig et al. 2005). The second study examined current religiousness at two time points in two separate female twin cohorts (the first cohort assessed at ages 14 and again at age 18, and a second cohort assessed at ages 20 and 25 (Koenig et al. 2008). Results from both studies showed an increase in heritability from adolescence to adulthood. The longitudinal study (Koenig et al. 2008) also examined the nature of stability and change in religiousness across age. For the 14- to 18-year-old cohort, i.e., from early adolescence to later adolescence, the stability in religiosity was due to shared environment influences, whereas change in religiousness was due to genetic and nonshared environmental influences. In contrast, in the 20- to 25-year-old cohort, stability in religiousness was due to genetic and shared environmental influences. Very little change in the variance of religiousness was noted across the age assessments, and the small amount of change observed was due to non-shared environmental influences.

The aim of the present study was to examine the changing etiology of religious values and religious attendance from early adolescence to late adolescence or young adulthood. To address these aims, we posed five questions:

1. Do levels of religious values and religious attendance change as a function of age in the available dataset? Based on results from epidemiological studies, we expect a decline in both religious attitudes and religious attendance.
2. Does the heritability of religious values and religious attendance change from adolescence to adulthood? Based on results of previous studies examining the heritability of religiosity in adolescents and adults separately, and a single longitudinal study examining change in religiousness from adolescence to adulthood, we expect the heritability of both phenotypes to increase from adolescence to adulthood.
3. What factors are responsible for the stability of religious values and religious attendance? As the participants in this study are only just becoming adults, and as such are unlikely to have left the family home, we predict that genetic factors remain relatively

constant across waves. Consequently, we expect stability of religiosity to be the result of both genetic and shared environmental effects.

4. What factors account for the change in religious values and religious attendance? Since there is likely some change in genotypic expression from early adolescence to early adulthood, and as the participants gain increasing independence from their families (associated with increased age) they are more likely to have more experiences outside of the family home that influence their attitudes and behavior, we anticipate that change in religiosity will be the result of both genetic and nonshared environmental effects.
5. Are there sex differences in change and stability, and the etiology of change and stability? Only a small number of studies have considered male and females separately, and those that have frequently found differences in heritability across sex, although the reports are inconsistent, with some studies reporting a higher heritability in males with others reporting a higher heritability in females. Consequently, it is difficult to predict whether there will be sex differences in this sample.

Methods

Participants

Participants were from the Community Twin Sample (CTS) and the Longitudinal Twin Sample (LTS) participating in a NIDA-funded Center on Antisocial Drug Dependence (CADD; DA-11015) at the University of Colorado. Detailed information regarding collection of these data is available elsewhere (Rhea et al. 2006). Briefly, inclusion in the CTS was open to all twins born in Colorado and twins attending Colorado primary or secondary schools. These twins were recruited through the Colorado Department of Health and 170 (of 176 eligible) school districts in Colorado. LTS twins were recruited at birth through the Colorado Department of Vital Statistics, and were added to the CADD sample as they reached their 12th birthday. The twins participated in two waves of assessment approximately 5 years apart. Religiosity data were available for 2749 individual twins at wave 1 (male = 1308, female = 1441), and 2478 individual twins at wave 2 (male = 1149, female = 1329). This sample comprised 685 MZ twin pairs (male = 310, female = 375), and 739 DZ twin pairs (male = 230, female = 234, opposite sex = 275). Twins were age 12–18 (M = 14.5, SD = 2.1) at wave 1 and 17–29 (M = 19.6, SD = 2.5) at wave 2. Although the age range at wave 2 is large, only 25.7% were older than 21 (an age at which it would be expected that most adults would have left their parental home), and only 6.3% were older than 23 years (5 years older that the 18 year upper age limit in wave 1). The mean age difference across waves is 5.3 years (SD = 1.0). Twins were included even if they were missing a score at either wave, as described below.

Twin zygosity was determined using a 9-item assessment of physical characteristics (Nichols and Bilbro 1966), completed by the interviewers and by using genotyping results from a minimum of 11 informative short tandem repeat polymorphisms (STRPs) using DNA from cheek swabs or saliva samples to confirm the initial zygosity determination. Twin pairs were categorized as identical, or monozygotic (MZ), if they had similar characteristics and were concordant for all markers, and fraternal, or dizygotic (DZ), if they presented with dissimilar characteristics and were discordant for any of the markers. Any discrepancies between the zygosity determination by the interviewer rating and genotyping were re-evaluated and resolved in a number of ways. Genotyping was initially repeated in the original laboratory and a second laboratory; if required, a new sample was collected and evaluated; and finally, interviewers reviewed photographs of the participating twins.

Measures

Religious values were assessed using five items from Jessor's Adolescent Health and Behavior Questionnaire (Jessor and Jessor 1977) that assessed how important different aspects of religion are to the individual. Participants were asked "How important is it to you":

1. To be able to rely on religious counsel or teaching when you have a problem?
2. To believe in God?
3. To rely on your religious beliefs as a guide for day-to-day living?
4. To be able to turn to prayer when you're facing a personal problem?
5. To attend religious services regularly?

Responses were scored as "Not important at all" (1), "Somewhat important" (2), "Important" (3), and "Very important" (4). These five items were highly correlated and the Cronbach's alpha was 0.92 at wave 1 and 0.95 at wave 2. The total score for religious values was the average score across all four or five items. We allowed for one missing item (if more than one item was missing the participant was given a missing value for the religious values score). Only 6 people (0.2%) at wave 1 and 2 (0.1%) at wave 2 were scored as missing using this criterion). The religious values score is similar to the concept of intrinsic religion (Bouchard Jr. et al., 1999).

Religious values at wave 1 correlated −0.19 (significant at the 0.01 level) with age at wave 1. However, the correlation between religious values and age at wave 2 was −0.01 and non-significant at the 0.01 level. To address the initial effect of age on the mean level of religious values, the religious values scores were regressed on age separately within sex, and the non-standardized residuals were used for analyses. The age regression was conducted for both waves of data, even though there was not a significant correlation with age at wave 2, to maintain consistency across waves.

Religious attendance, an example of religious behavior, was assessed using the question "How many times have you attended religious services during the past year?" from Jessor's Adolescent Health and Behavior Questionnaire (Jessor and Jessor 1977). Responses were: "more than once a week", "about once a week", "2–3 times a month", "about once a month", "about every month", "once or twice", and "none in the past year". The answer "none in the past year" was scored as 0, and "more than once a week" was scored as 6, and data were treated as quasi-continuous. The correlation between religious attendance and age was −0.13 at wave 1 and wave 2, and both correlations were significant at the 0.01 level. To account for these mean-level age differences, we also regressed out the effects of age on religious attendance scores as described above for religious values. All transformed scores were approximately normally distributed. The religious attendance score correlated 0.70 at wave 1 and 0.81 at wave 2 with item 5 from the religious values variable (How important is it to you to attend religious services regularly?), indicating that the religious attendance variable is a correlated but distinct phenotype.

Analyses

Model fitting analyses were conducted using the Mx statistical model-fitting program (Neale 2004). Models were fit to the raw data, modeling both first and second degree statistics, and allowing all twin pairs, including those with missing data, to be included in the analysis. Model fitting to twin data utilizes information about the similarities between MZ twin pairs and DZ twin pairs to estimate the contribution of genetic (A), shared environmental (C; environmental variance that contributes to the similarity between family members), and nonshared environmental (E; environmental variance that contributes to differences between family members) influences on the variance of a phenotype. We fit bivariate (wave 1, wave 2) Cholesky decomposition models separately to the religious values and religious attendance scores. The Cholesky decomposition model (Fig. 1) utilizes information about the phenotypic variances at each wave, the MZ and DZ covariances at each wave, and the cross-twin–cross-wave covariances for MZ and DZ twin pairs to partition the variance and the covariance across waves into genetic and environmental variance. This model decomposes the variance of wave 2 data into components common to time 1 and time 2 (A_1, C_1, and E_1), and that specific to time 2 (A_2, C_2, and E_2). When considering longitudinal data such as those used here, the Cholesky decomposition model can also inform of the nature of the stability of the phenotype, represented by the pathways a_{21}, c_{21}, and e_{21}, and change, represented by the residual variance components specific to time 2, pathways a_{22}, c_{22}, and e_{22}. To estimate how well the Cholesky decomposition model fits our data, we first tested a saturated model, a descriptive model with no constraints, and in which all possible parameters (means, variances, and covariances) are estimated freely. This provides a baseline fit (minus twice the log likelihood; −2LnL) against which we tested the fit of the Cholesky decomposition models. The difference in −2LnL between the saturated model and nested models is approximately distributed as χ^2, and a χ^2 difference test can be performed to examine whether the models significantly differ.

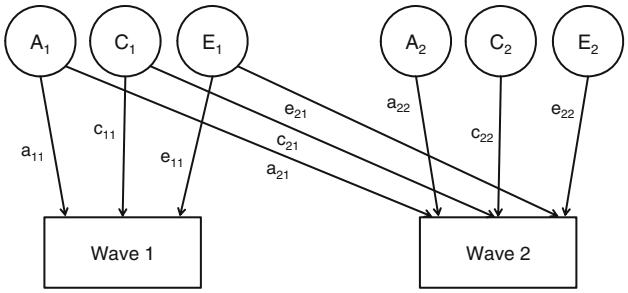

Fig. 1 A bivariate Cholesky decomposition model. A_1: Genetic variance common to wave 1 and wave 2; C_1: Shared environment variance specific to wave 2; E_1: Nonshared environment variance specific to wave 2; A_2: Genetic variance common to wave 1 and wave 2; C_2: Shared environment variance specific to wave 2; E_2: Nonshared environment variance common to wave 1 and wave 2; a_{11}, c_{11}, and e_{11}: the influence of A_1, C_1, and E_1 on wave 1 outcome; a_{21}, c_{21}, and e_{21}: the influence of A_1, C_1, and E_1 on wave 2 outcome; a_{22}, c_{22}, e_{22}: influence of A_2, C_2 and E_2 on wave 2 outcome

We tested whether the magnitude of the variance components differed for males and females by fitting a model in which all the pathways in the Cholesky decomposition model were equated to be the same for males and females, and comparing it with the full Cholesky decomposition model in which parameters were estimated freely across sex. A significant reduction in fit of the equated model compared with the full model indicates that the etiology of the phenotype, as well as the stability and change of that phenotype over time, is significantly different for males and females, whereas a non-significant reduction in fit indicates that religiosity and the development of religiosity over time do not differ across sex.

Results

Means and standard deviations of the raw religious values and religious attendance scores and mean difference data are presented in Table 1. Prior to regression on age, females scored significantly higher than males on religious values at wave 1, religious values at wave 2, and religious attendance at wave 1. There was no significant difference between male and female mean scores for religious attendance at wave 2. Mean scores for both religious values and religious attendance were significantly lower at wave 2 than at wave 1 for males and females. Both phenotypes were moderately correlated across wave; for religious values, the cross-wave correlation was 0.66 for males and 0.67 for females, and for religious attendance, the cross wave correlations were 0.63 for males and 0.59 for females.

Model fitting

The Cholesky decomposition model fit the religious values data well (see Table 2), as indexed by the non-significant reduction in fit of the Cholesky decomposition model compared with the saturated model. This means that the data are explained adequately by the Cholesky decomposition model. Equating the male and female parameter estimates did not result in a significantly worse fitting model when compared with either the saturated model or the full model. Figure 2 shows the model fitting results for religious values with standardized path coefficients and confidence intervals; paths with statistically significant parameters are shown in boldface. Similarly, the Cholesky decomposition model fit religious attendance data well, and no significant difference in variance components across sex was detected. Figure 3 shows the model fitting results for religious attendance with standardized path coefficients and confidence intervals; again, paths with statistically significant parameters are shown in boldface.

Table 2 Model fit statistics for the Cholesky decomposition models for religious values and religious attendance

Model	Fit statistics	Comparative fit	
	$-2\text{LnL}_{(\text{d.f.})}$	$\chi^2_{(\Delta\text{d.f.})}$	p
Religious values			
Saturated	$11090.07_{(4785)}$		
Full	$11122.87_{(4833)}$	$32.80_{(48)}$ [a]	0.95
Equate sex	$11133.10_{(4842)}$	$43.03_{(57)}$ [a]	0.92
		$10.23_{(9)}$ [b]	0.33
Religious attendance			
Saturated	$18255.62_{(4766)}$		
Full	$18291.65_{(4814)}$	$36.04_{(48)}$ [a]	0.90
Equate sex	$18302.63_{(4823)}$	$47.02_{(57)}$ [a]	0.83
		$10.98_{(9)}$ [b]	0.28

−2LnL minus twice the log likelihood fit statistic, *d.f.* degrees of freedom, χ^2 chi-square difference between models, *p* probability. *Full* model in which all parameters are estimated separately across sex. *Equate sex* model in which parameters are constrained to be the same across sex

[a] Comparison with the saturated model

[b] Comparison with the full model

From the path coefficient estimates shown in Figs. 2 and 3, we estimated the heritability, as well as the contribution of shared and nonshared environmental influences to the phenotypic variance of religious values and religious attendance at waves 1 and 2. It is also possible to derive the genetic, shared environmental, and nonshared environmental correlations between the two waves, the contribution of A, C, and E common to both waves to the phenotypic variance of wave 2 scores, and the contribution of A, C, and E specific to the phenotypic variance of wave 2 scores. These data are presented in Tables 3 and 4.

The absolute genetic variance of religious values increased from 0.24 to 0.45. Moreover, the heritability of religious values also increased slightly (although not significantly according to the overlapping confidence

Table 1 Descriptive data for religious values and religious attendance

Phenotype	Male			Female			Cross sex *t*-test**
	n	*M* (SD)	Cross wave *t*-test*	*n*	*M* (SD)	Cross wave *t*-test*	
Religious values							
Age 12–18	1301	2.56 (0.94)	$t = 6.92_{(2275)}$, $p < 0.001$	1434	2.71 (0.94)	$t = 5.96_{(2578)}$, $p < 0.001$	$t = 4.31_{(2733)}$, $p < 0.001$
Age 17–29	976	2.28 (1.04)		1146	2.48 (1.05)		$t = 4.41_{(2120)}$, $p < 0.001$
Religious attendance							
Age 12–18	1294	2.94 (2.16)	$t = 6.92_{(2267)}$, $p < 0.001$	1428	3.11 (2.17)	$t = 8.24_{(2567)}$, $p < 0.001$	$t = 2.02_{(2720)}$, $p = 0.04$
Age 17–29	975	2.31 (2.11)		1141	2.40 (2.13)		$t = 0.987_{(2114)}$, $p = 0.324$

M mean, *SD* standard deviation

* Comparison of mean religious values and religious attendance scores across waves, within sex; ** comparison of mean religious values and religious attendance scores across sex, within waves

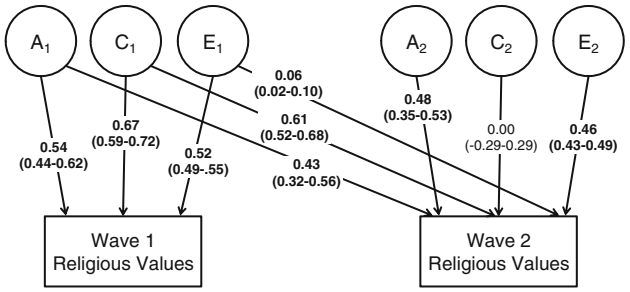

Fig. 2 Standardized path coefficients for the bivariate Cholesky decomposition model for religious values. Estimates in boldface indicate a significant pathway

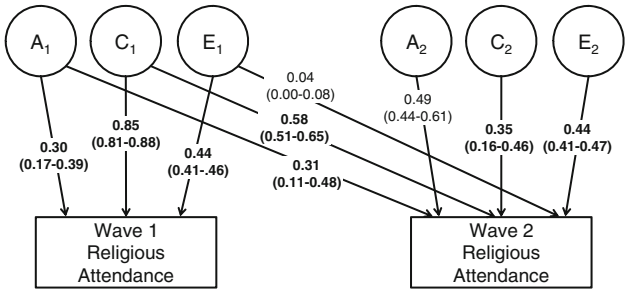

Fig. 3 Standardized path coefficients for the bivariate Cholesky decomposition model for religious attendance. Estimates in boldface indicate a significant pathway

intervals) from 29% at wave 1 to 41% at wave 2, and the magnitude of shared and nonshared environmental influences decreased slightly. Furthermore, the absolute shared environmental variance also decreased, while the absolute non-shared environmental variance remained constant. Approximately 55% of the variance of wave 2 religious values was due to factors in common with wave 1 (i.e. due to persistence or stability of pre-existing factors). Of this 55%, 67% was due to shared environmental effects, with the remainder due to persistent genetic effects.

The change in genetic variance and heritability of religious attendance was more dramatic and statistically significant (according to non-overlapping confidence intervals), with a genetic variance estimates of 0.40 at wave 1 and 1.51 at wave 2, and heritability estimate of 9% at wave 1 and 34% at wave 2. There was a corresponding decrease in the magnitude of shared environmental influences from 72% at wave 1 to 46% at wave 2, and the magnitude of nonshared environmental influences remained the same across waves. The change in religious values over time (45% of the variance at wave 2) was due almost equally to genetic and nonshared environmental effects. The etiology of religious attendance appeared to change slightly more than that for religious values. The proportion of variance of religious attendance at wave 2 attributable to pre-existing factors was largely due to persistent shared environmental effects (77%), and the rest was due to persistent genetic effects. The remaining 56% of the variance of wave 2 religious attendance was due to time-specific variance, which was due to a combination of genetic (43%), shared environmental (22%), and nonshared environmental (34%) influences.

We also conducted ad hoc exploratory analyses on the current data, splitting the data by age at the initial wave, to determine whether cohort differences would exist in those who were most likely to still live at home, compared with

Table 3 Twin-pair correlations, and the contribution of genetic, shared environmental, and non-shared environmental variance components to the variance within waves, the correlation across waves, the stability of religious values, and the change in religious values from wave 1 to wave 2

Outcome	Correlations		Parameter estimates		
	MZ	DZ	A (95% CI)	C (95% CI)	E (95% CI)
Absolute variance at age 12–18			0.24 (0.16–0.33)	0.38 (0.29–0.46)	0.23 (0.21–0.26)
Standardized variance at age 12–18	0.72	0.59	0.29 (0.19–0.39)	0.44 (0.35–0.53)	0.27 (0.24–0.30)
Absolute variance at age 17–29			0.45 (0.34–0.57)	0.40 (0.29–0.52)	0.24 (0.21–0.27)
Standardized variance at age 17–29	0.78	0.57	0.41 (0.31–0.52)	0.37 (0.27–0.46)	0.22 (0.19–0.25)
Cross-wave correlation	0.63	0.53	0.66 (0.55–0.83)	1.00 (0.89–1.00)	0.13 (0.05–0.21)
Stability[a]			0.18 (0.10–0.31)	0.37 (0.26–0.46)	0.004 (0.001–0.01)
			33%	**67%**	**0%**
Change[b]			0.23 (0.12–0.28)	0.00 (0.00–0.08)	0.21 (0.19–0.24)
			52%	**0%**	**48%**

CI confidence intervals, *A* genetic variance, *C* shared environmental variance, *E* non-shared environmental variance

[a] Variance that persists from wave 1 (age 12–18) to wave 2 (age 17–29). The number in boldface is the percentage contribution of A, C, and E to the persistent variance (e.g. the contribution of genetic variance to the total stable variance is due to $0.18/(0.18 + 0.37 + 0.004) = 33\%$

[b] Variance that is specific to wave 2 (age 17–29). The number in boldface is the percentage contribution of A, C, and E to the variance that is specific to wave 2 (age 12–29)

Table 4 Twin-pair correlations, and the contribution of genetic, shared environmental, and non-shared environmental variance components to the variance within waves, the correlation across waves, the stability of religious attendance, and the change in religious attendance from wave 1 to wave 2

Outcome	Correlations		Parameter estimates		
	MZ	DZ	A (95% CI)	C (95% CI)	E (95% CI)
Absolute variance at age 12–18			0.40 (0.11–0.70)	3.33 (2.95–3.72)	0.88 (0.79–0.98)
Standardized variance at age 12–18	0.81	0.76	0.09 (0.02–0.15)	0.72 (0.66–0.77)	0.19 (0.17–0.22)
Absolute variance at age 17–29			1.52 (1.09–1.98)	2.06 (1.58–2.53)	0.86 (0.77–0.98)
Standardized variance at age 17–29	0.80	0.64	0.34 (0.25–0.45)	0.46 (0.36–0.55)	0.19 (0.17–0.22)
Cross-wave correlation	0.60	0.49	0.54 (0.19–1.00)	0.86 (0.76–0.97)	0.09 (0.00–0.18)
Stability[a]			0.10 (0.01–0.37)	0.34 (0.26–0.42)	0.00 (0.00–0.01)
			22%	**77%**	**0%**
Change[b]			0.24 (0.00–0.37)	0.12 (0.02–0.22)	0.19 (0.17–0.22)
			43%	**22%**	**34%**

CI confidence intervals, *A* genetic variance, *C* shared environmental variance, *E* non-shared environmental variance

[a] Variance that persists from wave 1 (age 12–18) to wave 2 (age 17–29). The number in boldface is the percentage contribution of A, C, and E to the persistent variance (e.g. the contribution of genetic variance to the total stable variance is due to $0.10/(0.10 + 0.34 + 0.00) = 22\%$

[b] Variance that is specific to wave 2 (age 17–29). The number in boldface is the percentage contribution of A, C, and E to the variance that is specific to wave 2 (age 12–29)

those who are more likely to have moved out of the family home by wave 2. The 2 groups were age 12–14 years, and 15–18 years, at the initial wave of data collection (corresponding to approximately 17–19 and 20–23 at wave 2), and the etiology of stability and change was assessed separately for each age cohort. The percentage contribution of A, C and E to stability and to change are presented in Table 5. Shared environment contributed the major portion of the variance of stability in the youngest cohort for both religious values and religious attendance, while genetic effects were more salient contributors to stability in the older cohorts, with shared environment still contributing but to a lesser extent. The results of these analyses also showed that change in the younger adolescent group was due to genetic, shared environment and nonshared environmental factors, whereas in the older cohorts, genetic and nonshared environment influences accounted for change.

Table 5 Contributions of genetic (A), shared environmental (C), and nonshared environmental (E) influences to stability and change in different cohorts

Outcome	Cohort age at wave 1	Parameter estimates		
		A (%)	C (%)	E (%)
Religious values				
Stability	Age 12–14	27	73	0
	Age 15–18	58	41	1
Change	Age 12–14	26	25	49
	Age 15–18	46	0	54
Religious attendance				
Stability	Age 12–14	6	94	0
	Age 15–18	62	37	1
Change	Age 12–14	37	35	28
	Age 15–18	29	1	70

Discussion

The aim of the current study was to determine the extent to which religious values and behavior were stable across time, and examine the etiology of the stability and change. Our results showed that religious values and behavior were partially stable over time, and that stability was largely due to persistent shared-environment and to a lesser extent to persistent genetic influences. Change in religious values was due to both genetic and nonshared environmental influences specific to early adulthood, whereas change in religious attendance was due to a similar magnitude of genetic, shared environment, and nonshared environmental influences.

Did levels of religious values and religious attendance change as a function of age?

As shown in previous studies, comparison of the difference between overall mean at wave 1 and wave 2 demonstrated a significant decrease in mean levels for both religious values and church attendance. These results suggest that aspects of people's religiosity declines from adolescence to early adulthood.

Did heritability of religious values and religious attendance change from adolescence to adulthood?

Consistent with previous studies, the heritability for both religious values and religious attendance was lower in

adolescence and higher in adulthood. This was also observed in the absolute genetic variance scores, which also increased across waves for both phenotypes. The heritability of religious values did increase, as expected; however, the increase was small (an increase in heritability of 0.12), indicating that the etiology of religious values did not change much in our sample over the period assessed. In contrast, the heritability of religious attendance, a measure of religious behavior, increased substantially over time (an absolute increase of 0.25). This might be, in part, due to the fact that religious attendance is largely influenced by parental control during adolescence (that is, parents encourage their adolescents to attend the same religious services that they themselves attend). Consequently, MZ and DZ twins would both attend at the same rate as their co-twin, regardless of genetic predisposition, giving rise to the low heritability and higher shared environmental variance seen here. This is consistent with the results reported in a study by Boomsma et al. (1999), in which adolescent MZ and DZ twin correlations were similar to ours for church attendance. As the adolescents age into early adulthood, however, religious attendance would be less likely influenced by parental control, and more a result of their own choices. Consequently, a person who is genetically predisposed to religious values might also be more likely to opt to go to religious services more often, regardless of their parents' attitudes and attendance.

If the hypothesis that a person who is genetically predisposed to religious values might also be more likely to opt to go to religious services more often is true, there should be a greater genetic contribution to the correlation between religious values and religious attendance in adulthood than in adolescence. In order to test this hypothesis, we conducted two further Cholesky decomposition analyses examining the etiology of the covariance between religious values and religious attendance separately at wave 1 and 2. The two phenotypes were correlated 0.64 at wave 1 and 0.76 at wave 2. The proportion of the correlation between religious values and religious attendance due to genes increased from 22% at wave 1 to 40% at wave 2. Conversely, the contribution of the shared environment to the phenotypic correlation decreased from 73% at wave 1 to 48% at wave 2. Finally, the nonshared environment contributed 5 and 11% to the phenotypic correlation between religious values and religious attendance at waves 1 and 2, respectively.

The increase in heritability for both religious values and religious attendance with age is consistent with a large literature describing the same trend for a number of personality, behavioral, and psychiatric phenotypes (Bergen et al. 2007). Increasing heritability with age is thought to be the result of active gene–environment correlation, such that, as people get older and gain independence from the constraints placed upon them as children and adolescents, they select and modify environments that reinforce their genetic predispositions, consequently increasing the heritability of that phenotype.

What factors were responsible for the stability of religious values and religious attendance?

As predicted, the stability of religious values over time is due to genetic and shared environmental influences. Although genes do contribute to the stability, they contribute to a lesser extent than the shared environment. The same pattern of results was found for religious attendance. Our exploratory analyses, where analyses were conducted separately for two age cohorts (age 12–14 and 15–18 at wave 1), showed that shared environment was the largest contributor to stability in both religious values and religious attendance in the youngest cohort, whereas genetic influences also contributed to the stability in the older cohort. The contribution of a shared environmental influence on stability in younger adolescence may arise because they remain in the same environment across waves; i.e., most have not yet left home and are still under the same constraints of the home environment in earlier adolescence. In contrast, the genetic contribution to stability in the older adolescents, who are more likely to have left home and started lives away from their families between waves 1 and 2, may be indicative of the adolescents' increased ability to select their own environment based on their underlying genetic predispositions. This is further support for active gene–environment correlation. Overall, these results are largely consistent with those reported by Koenig et al. (2008), who found stability to be influenced by shared environmental influences from age 14 to 18, but by genetic and shared environmental influences from age 20 to 25.

What factors were responsible for the change in religious values and religious attendance?

Change in religious values was due to genetic and nonshared environmental influences, and change in religious attendance was due to genetic, shared and nonshared environmental influences. These influences may reflect a number of previously identified factors associated with change, such as college attendance. It has been demonstrated that divorce and college attendance are associated with a decline in religiosity (Stolzenberg et al. 1995; Ueker et al. 2007; Sherkat 1998), and both divorce and college attendance might be the result of genetic, shared environmental and nonshared environmental influences. Another explanation might be that different genes that influence religious phenotypes become expressed as the adolescent matures. Furthermore, the development of new friendship

groups outside of the confines of the school and family environment may also account for some of the change identified in the current study. Our findings for an influence of genetic and nonshared environment effects on the change in religious values are consistent with those described for adolescents (14–18), but not adults (20–25), in Koenig et al. (2008).

Our analyses of cohort difference showed that shared environmental influences were responsible for the change in religious values and religious attendance only in the youngest cohort (i.e., those who are more likely to still be living with their families at wave 2). Therefore, it is possible that there were changes occurring at the family level that influenced some of the change in religious values and religious attendance in the youngest cohort. However, for the older cohort (who are more likely have moved out of their childhood homes and started independent lives between the two waves), only genetic and nonshared environmental influences were responsible for change in religious values and religious attendance.

Are there sex difference in the etiology of religious phenotypes, or in the etiology of stability and change?

On average, females reported higher levels of religious values and attended religious services more frequently than males; findings that are consistent with those of previous studies (e.g. Stolzenberg et al. 1995). However, we found no significant sex differences in the etiology, stability, or change in either religious values or religious attendance.

Limitations of the study

One limitation of this study was the lack of information regarding religious affiliation. However, studies do not typically find a genetic influence on religious affiliation. Our measures (with the possible exception of one question referring to God), do not openly specify a religion, and can be applied to many religious groups. The second limitation of the current study is that of attrition. Of the 2754 individuals enrolled at wave 1, 733 did not participate at wave 2. It has been suggested that people who are more religious are more likely to continue participation in longitudinal studies (Koenig et al. 2008), and this was true in the present study as well. For the religious values score, absentees at wave 2 had a mean of 2.56 (SD = 0.96) at time 1, whereas those who participated at wave 2 have a mean of 2.67 (SD = 0.93) at time 1. This difference is small but significant ($t_{(2733)} = 2.417$, $p = 0.01$). For religious attendance, the wave 1 mean for absentees at wave 2 was 2.68 (SD = 2.15), whereas the mean for those who continued to participate was 3.16 (SD = 2.16). Again, this was significant ($t = 5.112_{(2720)}$, $p < 0.001$). Since subjects remaining in the study scored higher than those who no longer remained in the study, it is expected that the wave 2 means were possibly a little higher than would be expected if there were no attrition. However, we still observed the expected reduction in overall mean from waves 1 to 2. Finally, because the models were fit to raw data, including twin pairs with incomplete data, effects of attrition would be less than for analyses that required complete data.

Conclusions

The current study was the first to examine the etiology of the development of religiosity from adolescence to early adulthood in males and females, and the results build upon a similar study observing persistence and change across adolescence and again across young adulthood. Furthermore, the results of the current analyses support previous findings that religious values and religious attendance are heritable, but appear to become more heritable in young adults than in adolescents. The primary source of persistence of religious values and religious attendance is the shared environment, although genetic effects do also appear to be important. The source of change was almost equally attributable to genetic and nonshared environmental variance for religious values, and to a similar magnitude of genetic, shared environmental, and nonshared environmental variance for religious attendance. Finally, we were unable to identify any sex differences in the magnitude of genetic, shared environmental, and nonshared environmental influences on religious values and religious attendance, or in the stability and change of these two phenotypes.

The findings of the current study also have implications for future research. It would be of interest to try to understand the nature of the genetic influence responsible for the persistence of religious values and service attendance. Evidence points towards personality (Emmons et al. 2008; Wink et al. 2007); however, it would be interesting to determine which components of personality are involved, and to what extent they can explain the persistence of religiosity across adolescence and early adulthood. Furthermore, it would also be of interest to find the factors accountable for time specific influences on religious phenotypes in emerging adulthood. As suggested in the introduction, environmental factors, such as college attendance and personal relationships might account for some of this variance, but our results also suggest time specific genetic effects.

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