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Rearing Environmental Influences on Religiousness: An Investigation of Adolescent Adoptees

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Abstract

Religiousness is widely considered to be a culturally transmitted trait. However, twin studies suggest that religiousness is genetically influenced in adulthood, although largely environmentally influenced in childhood/adolescence. We examined genetic and environmental influences on a self-report measure of religiousness in a sample consisting of 284 adoptive families (two adopted adolescent siblings and their rearing parents); 208 biological families (two full biological adolescent siblings and their parents); and 124 mixed families (one adopted and one biological adolescent sibling and their parents). A sibling-family model was fit to the data to estimate genetic, shared environmental, and nonshared environmental effects on religiousness, as well as cultural transmission and assortative mating effects. Religiousness showed little evidence of heritability and large environmental effects, which did not vary significantly by gender. This finding is consistent with the results of twin studies of religiousness in adolescent and preadolescent samples.

Keywords

religiousness; behavior genetics; adoption; heritability

Behavior genetic research on religiousness has supported the conclusion that while there is moderate heritability of religiousness in adulthood (around .40; D'Onofrio, Eaves, Murrelle, Maes, & Spilka, 1999; Koenig, McGue, Krueger, & Bouchard, 2005; Waller, Kojetin, Bouchard, Lykken, & Tellegen, 1990), the heritability of religiousness is much lower in childhood (less than .10; Abrahamson, Baker, & Caspi, 2002; Koenig, McGue, & Iacono, 2008) and increases with age (Koenig et al., 2005, 2008). In childhood/adolescence, similarity among family members is due primarily to shared environmental factors, while in adulthood there are strong genetic but weak shared environmental influences. For example, Koenig et al. (2005) reported heritability of .12 for religiousness using retrospective childhood reports but . 44 for current adulthood reports in a sample of male twins. Since the same pattern of increasing heritability holds for other psychological traits (e.g., intelligence, social attitudes, and

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externalizing behaviors; see Bergen, Gardner, & Kendler, 2007), it is likely that the development of the heritability of religiousness parallels that observed for other traits

As noted above, many twin studies of religiousness have been completed, but few studies have examined the heritability of religiousness with an adoption design. Abrahamson et al. (2002) examined religiousness in a sample of adopted and non-adopted siblings ages 12-15, and found small heritability but large shared environmental influences. In an adoption study with individuals who averaged 18 years old, Beer, Arnold, & Loehlin (1998) found a heritability of .28 for religious orthodoxy. While twin studies have power to detect genetic effects, adoption designs provide a direct estimate of shared environmental influences. Because both types of studies have different strengths and weaknesses, it is important to use adoption designs to corroborate findings from twin research, especially as they relate to environmental influences.

Using a sample of adopted and biological relatives, we sought to replicate and extend previous behavior genetic work on religiousness. The current study's sibling-family design, like Abrahamson et al.'s (2002), allows for an estimate of genetic and environmental effects of religiousness, as well as estimates of assortative mating, genetic-environmental covariance, and the proportion of shared environmental influence that is due to cultural transmission via parents. This expanded model accounts for effects that twin models cannot. Given the adolescent age (mean 14.9 years) of the sample used in the current study, it was hypothesized that genetic influences on religiousness would be small while environmental influences would be substantial. This study also examined sex differences in the heritability of religiousness, which have received little attention via biometric modeling in younger samples. Because of inconsistent findings for gender differences in heritability in adulthood (e.g., D'Onofrio, et al., 1999; Kirk, et al., 1999; Truett, Eaves, Meyer, Heath, & Martin, 1992) and the lack of any theory-driven reasons for differential heritability, we hypothesized there would be no significant differences in heritability by gender.

Method

Participants

The sample consisted of families participating in the Sibling Interaction and Behavior Study (SIBS; McGue, et al., 2007), a longitudinal investigation of adoptive and non-adoptive families with two adolescent siblings and their rearing parents. Recruitment procedures for the SIBS study, as well as evidence for minimal sampling bias can be found in McGue et al. (2007). The total sample here includes 1234 siblings, 549 fathers, and 613 mothers in three types of families: biological, adoptive, and mixed. Biological families (N = 208; 62 male-male, 68 femalefemale, 78 mixed gender pairs) included two biological children and their biological, rearing parents. Adoptive families (N = 284; 53 male-male, 95 female-female, 136 mixed gender pairs) included two adopted children and their adoptive, rearing parents. Mixed families (N = 124; 43 male-male, 54 female-female, 27 mixed gender pairs) included one biological and one adopted child of the rearing parents. Data for some individuals was not used, for several reasons: 1) 45 siblings, 19 fathers, and 5 mothers did not complete the religiousness assessment, 2) some individuals (see below) were missing items on the religiousness assessment, and 3) there were 8 step-fathers and 2 step-mothers in biological families. An additional two adopted siblings were dropped from the sample, one who was biologically related to her sibling and one who had an IQ below 70, suggesting difficulties in interpreting self-report forms. The first of these families was included for non-family analyses, while the second was included for all analyses though the religiousness score for the child with low IQ was not used.

All participants were given a religiousness measure at their intake visit. The average age of the children at this visit was 14.9 (SD = 1.9, N = 1187), with the elder sibling at 16.1 years (SD = 1.5, N = 586, range 12.9-21.0), and the younger sibling at 13.8 years of age (SD = 1.6, N =

601, range 10.7-19.4). The average ages of the fathers and mothers were 48.3 years (SD = 4.3, range = 35.3-64.3) and 46.6 years (SD = 4.2, range = 35.4-60.2), respectively.

The sample of adopted children was ethnically diverse; approximately 67% were of Asian descent and 12% were of another non-Caucasian descent. This was not true of the biological siblings, approximately 96% of whom were Caucasian, or their parents, over 97% of whom were Caucasian.

Measures

A ten-item measure was used to assess religiousness. One item asked religious affiliation, and was not included in these analyses. The other nine items asked about various religious activities and participation, including questions about frequency of attending religious services, seeking guidance through prayer, reading scripture or religious material, and observing religious holidays, as well as the general importance of religion in daily life. For more details about the measure the reader is referred to Koenig et al. (2005). The nine items were summed to create a continuous essentially normally distributed religiousness score (alpha = .85, N = 974 for the siblings; .90, N = 515 for fathers; and .89, N = 599 for mothers). In a principal axes factor analysis, the nine items all loaded highly on one factor that accounted for over 40% of the variance in the sibling sample and over 50% of the variance with the parents. If a participant was missing one of the nine items, the mean for that item was substituted before adding the items to form the scale score. The items most often missing were "how often do you decide moral 'dos' and 'don'ts' in religious terms or for religious reasons?" (19.5% of siblings were missing this item), and "how often do you seek guidance, help, or forgiveness through prayer?" (10.6% missing) and "how often do you review or discuss religious teachings with your family?" (10.9% missing). Any participant missing two or more items was not included in the analyses (17.9% (213) of the siblings, 1.6% (9) of the mothers, and 2.4% (12) of the fathers). Missing two or more items in the offspring was not related to gender, ethnicity, or offspring adopted/biological status. If a parent was missing two or more items from the selfreport measure of religiousness or did not complete the self report, but the spouse had the requisite eight items referring to the partner we used the "other" parent's assessment, thus gaining 24 more father scores and 1 more mother score. A previous study found high self-other correlations using the same measure of religiousness, validating our use of spousal report (Koenig et al., 2005).

Procedure

Mean differences between siblings by sex, age, gender and ethnicity were analyzed using SAS PROC Mixed (Littell, Milliken, Stroup, & Wolfinger, 1996) to account for the correlated family data. The same procedure was also used to assess mean differences between parents. Because of the differences in religiousness scores by sibling gender and the slight differences by age, scores were age- and sex-corrected before computing sibling correlations and fitting the sibling-family model. This was done by grouping the siblings into eight categories by age and sex (four different age groups for each gender) and subtracting the mean for the group from the religiousness scores for the siblings in that group. This transformation changed the mean religiousness score for siblings to zero, but did not affect the variance of the distribution. As parents were already gender segregated, their scores were not adjusted. When gender and age effects were being tested, original non-corrected religiousness scores were used.

Sibling and parent-child correlations were estimated by maximum likelihood using Mx (Neale, Boker, Xie, & Maes, 1999). If biologically related individuals are only slightly more similar phenotypically than non-biologically related individuals, this would suggest weak genetic effects but strong environmental effects. Sibling correlations by family type were computed by estimating common (among sibships) and unique (within sibships) components of variance

and taking the ratio to produce the correlation. Gender differences in the sibling correlations were analyzed by equating correlations across gender and assessing model fit. Age moderation was then incorporated by estimating adopted and biological sibling correlations while allowing each sibling's age to moderate the common and unique components of variance. Test for age moderation was consequently a four degree of freedom likelihood ratio test, where fit of the model in which the moderated portions of both variance components were constrained to zero for both adopted and biological sibling groups was compared to the unconstrained model. Finally, sibling and parent-child correlations were estimated for all three types of families using the age- and sex-corrected raw data.

Genetic and environmental contributions to individual differences in religiousness were estimated by fitting a sibling-family model (see Abrahamson, et al., 2002; Neale, Walters, Eaves, Maes, & Kendler, 1994) to the age- and sex-corrected raw data using Mx (Neale, et al., 1999). This model, depicted in Figure 1, takes advantage of the fact that familial resemblance for religiousness will be a function of both genetic and shared environmental factors among biological relatives but only shared environmental factors among adopted relatives. The path diagram is shown for a mixed family (one biological and one adopted sibling), and looks slightly different for the adopted and biological families. The model is parameterized in terms of genetic (A), shared environmental (C), and nonshared environmental (E) effects on the phenotype; cultural transmission from mother's (m) and father's (f) phenotypes to offspring's shared environment, a residual sibling-specific shared environmental effect (B) to account for shared environmental effects not attributable to cultural transmission; and assortative mating (D). Marital resemblance is assumed to be based on direct phenotypic homogamy. Cultural transmission and direct phenotypic homogamy induce a (passive) correlation between additive genetic and shared environmental effects, which can be derived in terms of the other parameters in the model. (This differs from twin models, where neither the passive A-C correlation nor the assortative mating effects can be modeled, the first of which could produce an overestimation of genetic effect and the latter of which could produce an overestimation of shared environmental effects.) The adoption model assumes that there are no selective placement effects, and thus no A-C covariance, in adoptive families. This assumption bounds the magnitude of the A-C covariance at twice the difference between the variance of the adopted and non-adopted individuals. Since the shared environment includes the effects of cultural transmission (m and f parameters) and effects not attributable to parental phenotypes (B), we were able to determine that portion of the shared environmental effect due to cultural transmission. Nonshared environment includes effects that make the siblings different from one another, as well as measurement error. The Mx script for this model is available on request.

Multiple models were fit to the data to test the significance of: 1) genetic effects (A and A-C covariance fixed to zero), 2) shared environmental effects (C, f, m, and the A-C covariance fixed to zero), 3) assortative mating (D fixed to zero), and 4) differential parent cultural transmission (f and m equated). Model fit was assessed by the difference in -2 log likelihood (-2LL) between the reduced and full models. A significant difference in -2LL, examined on the chi-square distribution with degrees of freedom equal to the difference in degrees of freedom between the two models, means the reduced model does not fit the data as well as the full model.

Results

Means and Mean Differences

The mean religiousness score for the siblings was 16.6 (SD = 7.3, N = 974) for the raw data. Table 1 shows the mean religiousness scores for siblings and parents by gender and family status. Female siblings had a higher religiousness score than males, t(155) = 2.6, p < .05, and the difference in status tended toward significance with adopted individuals having a lower

mean, t(70) = 2.0, p = .05. The effect of age also tended toward significance, with lower religiousness scores for older siblings (b = -.21, p = .05). Ethnicity (trichotomized as Caucasian, Asian, and other) had no effect on religiousness scores (F(2,69) < 1.0, p > .05), with mean scores of 16.9 (SD = 7.2), 16.2 (SD = 7.6), and 16.3 (SD = 6.7) for Caucasian, Asian, and other ethnicities, respectively. The mean religiousness score was significantly higher for mothers (21.1, SD = 7.4) than fathers (17.8, SD = 8.3; t(502) = 11.6, p < .05).

Correlational Analyses

Sibling correlations are reported in Table 2. There was no consistent significant difference between male and female correlations (a non-significant change in model fit, $\chi^2(6) = 12.0$, p > .05, when correlations were equated across gender for each family type). In general, sibling correlations were somewhat greater for the biological pairs than for either the adoptive or mixed pairs. This pattern was found consistently for males and females separately, except where the small sample sizes created outliers.

In the age moderation model, there was no significant effect of age (change in model fit, $\chi^2(4) = 2.2$, p > .05, when the moderators were taken out the model). Because neither gender (see above) nor age was a significant moderator of the sibling correlations, the data were age- and sex-corrected and parental data were added to the model to create a full sib-family model, estimating genetic and environmental effect across gender and age.

The correlations for the full sibling-family model are reported in Table 3, which differ somewhat from those in Table 2 because the former were adjusted for the effects of age and sex while the latter were not. The sibling and parent-offspring correlations between biologically unrelated individuals were generally around .50 and only slightly lower than the corresponding correlations among biologically related individuals, which were around .60. This pattern is most evident in mixed families, where the mother-offspring and father-offspring correlations were .62 and .59, respectively, in biologically related correlated .51 in mixed families and . 51 in adopted families, while biologically related siblings correlated .61. The correlation between parents was stable across family groups (.64 for adoptive families, .69 for biological and mixed families), and indicated high assortative mating or spousal influence for religiousness.

Model Fitting

The results of the sibling-family model are shown in Table 4. The full model estimates are reported (-2LL(2085) = 13704.4), revealing little heritability (.06, 95% CI = .01-12) and large shared environmental effects (.46, .37-.56). Of the shared environmental estimate, 68% was attributable to parental transmission effects: cultural transmission paths were estimated at .31 (.21-.42) for fathers and .59 (.48-.69) for mothers. Assortative mating was high for religiousness, with a standardized estimate of .65 (.60-.70).

Model fitting analyses showed that the full model best represented the data. Fixing the genetic paths to zero (A and A-C covariance) resulted in a small but significant loss of fit ($\chi^2(2) = 6.1$, p < .05), as did the model with no shared environmental effects (C, f, m, and A-C covariance; $\chi^2(4) = 244.3$, p < .001). The two other models fit to the data also fit worse than the full model: fixing assortative mating to zero, $\chi^2(1) = 312.1$, p < .001, and equating f and m to be equal, $\chi^2(1) = 5.3$, p < .05.

Discussion

In this sample of adopted and non-adopted adolescents and their parents, individual differences in religiousness were due primarily to shared and nonshared environmental influences, with the majority (68%) of the shared environmental influence coming from the cultural transmission paths. Genetic factors were found to contribute only minimally to variation in religiousness, and there was no evidence of a difference in heritability by gender. The correlations between biologically related siblings and biologically related offspring and parents were not much stronger than the corresponding correlations between genetically unrelated individuals. This supports the finding in previous studies, primarily twin studies (e.g., Koenig, et al., 2005), that the heritability of religiousness is small in adolescent or childhood samples. The belief that religiousness is largely an environmentally transmitted trait seems to be true in childhood.

The current results are similar to those of Abrahamson et al. (2002), who reported heritabilities for religiousness between .05 and .00 from ages 12 to 15, matching our estimate of 6%. The sibling correlations reported here are slightly higher than the correlation of .36 for religious orthodoxy reported in a sample of adopted siblings with a mean age of 18 by Beer et al. (1998). The stronger adoptive correlations in the current sample lead to a larger estimate of shared environmental effects. Differences in age or maturity level of the samples, as well as differences in the assessment of religiousness, might account for some of the differences, but more research should investigate other possible moderators of genetic and environmental effects on religiousness.

The large shared environmental effects on religiousness, often thought to include parenting styles and other parenting influences, and the significant cultural transmission paths are in accord with developmental socialization theory espousing the impact of environmental transmission of values (Maccoby & Martin, 1983). Also, in the current study, the cultural transmission path was stronger for mothers than fathers. Given the larger correlations for religiousness found between mothers and their children in this study and some previous work (e.g., Bao, Whitbeck, Hoyt, & Conger, 1999; Dudley & Dudley, 1986), this finding is not surprising. One possible reason for this differential effect was examined by Boyatzis and Janicki (2003), who found that mothers initiated conversations about religion with their children much more frequently than fathers did, providing the possibility for increased socialization. More research is needed, however, to examine whether the ways fathers and mothers pass on their religious values differ.

There were several limitations to this study. First, the sample included only families living in Minnesota. Differences may exist in other populations in the United States or other countries. Spilka, Hood, Hunsberger, and Gorsuch (2003) state that fewer people attend church regularly in countries like Britain, France, Australia, and Canada than in the United States. The sample in the current study did involve adolescents of different ethnicities, but the distribution of background was uneven, with a majority of Caucasian biological offspring but Asian adopted offspring. There were, however, no significant mean differences in religiousness by ethnicity. A study on the genetic and environmental influences on church attendance in US and Australian adults found a smaller shared environmental component in the US compared to Australia (.05 and .15 respectively; Kirk, et al., 1999). Though the current results are in-line with those from Abrahamson et al. (2002), where the sample was from Colorado, more research needs to be completed on cross-cultural samples to determine if there are differences in the genetic and environmental architecture of religiousness in adolescence.

A second concern is that individuals in adoption samples may not be representative of the population at large. The mean religiousness score for the biological siblings was slightly higher

than the adopted siblings, but this difference only tended towards significance. The majority of the adopted siblings were placed into homes via Lutheran and Catholic adoption agencies, and it is possible that the parents using these services are more open to religion. However, the adoptive parents were not more religious than the biological parents in this sample. According to McGue et al. (2007) the major differences between adopted and biological families in this sample are less disinhibitory psychopathology and higher SES for adoptive as compared to non-adoptive parents. This earlier study also found that this restriction in range did not affect the adopted sibling correlation for delinquency, drug use, or intelligence, and therefore did not

the adopted sibling correlation for delinquency, drug use, or intelligence, and therefore did not bias the shared environmental estimates for these phenotypes. Selective placement may also be a concern with an adoptee sample, such that adoptees may be placed in a home similar to their biological home, increasing the similarity between adopted siblings and their adoptive parents. Though this similarity would be due to shared genes, it would contribute to the estimate of shared environmental effects. There are clearly limitations of the adoption design, but the similarity in results between the current study and twin studies supports the findings reported here.

Another limitation of the methodology used is that the model assumes that genetic and environmental influences important in childhood are also important in adulthood. If this is not the case, parent-child correlations, along with genetic and shared environmental effects, are minimized. Biological parent-child correlations would be lower than biological sibling correlations as a result of the lack of genetic isomorphism across ages. If this were the case, the cultural transmission parameters in the model would become negative (Abrahamson, et al., 2002). Neither of these (the negative cultural transmission paths nor the attenuated parent-child correlations) occurred here.

Finally, the measure used in the current study is only one way to assess religiousness. Other aspects of religiousness, like Extrinsic and Intrinsic religious motivation (Allport & Ross, 1967), may be more or less heritable in children. Also, it is unknown to what extent adolescents can accurately report their religiousness separately from any parental religiousness, and this may increase estimates of shared environmental effects. Additionally, almost 18% of the siblings had incompletely filled out the religiousness questionnaire. Though the missingness did not differ across gender, ethnicity, or offspring status, the missing data may have affected the results.

The finding of small genetic and large shared environmental effects on religiousness in adolescence is supported by the current research. Though the genetic effect has been shown to increase with age, it is still important to study childhood environmental effects. The impact of adolescent religiousness may be long lasting, or may become important again when individuals begin their own families in adulthood. Investigation of shared environmental effects on religiousness in childhood would and can help us to better understand religious development and religious identity formation.

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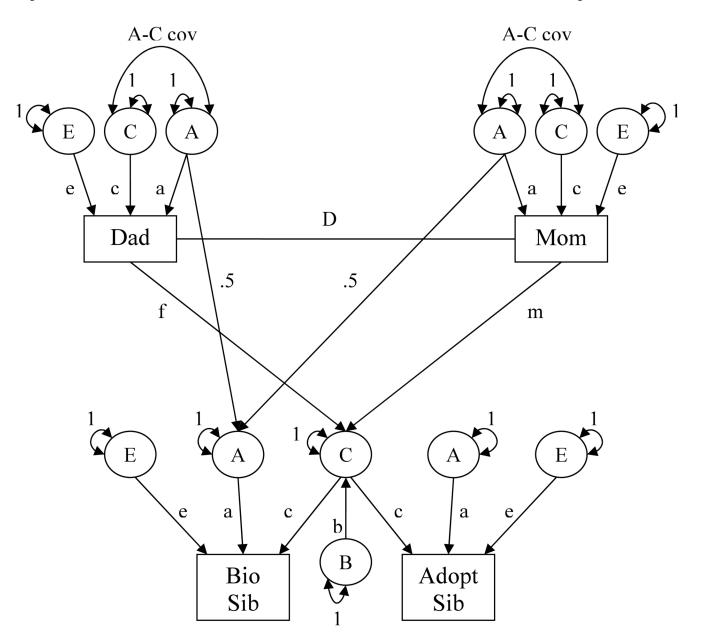


Figure 1.

A sibling-family model showing one biological and one adopted sibling. The model includes genetic (a), shared environmental (c), nonshared environmental (e) effects. Assortative mating was modeled in terms of a co-path (D) between the parents' phenotypes. The sibling shared environment is modeled as an effect of mother's (m) and father's (f) religiousness, as well as a sibling-specific residual effect (B). The existence of the cultural transmission paths (m and f) as well as assortative mating (D) induces a correlation between additive genetic and shared environmental effects (the A-C covariance; the same correlation exists in the offspring generation but is not depicted for convenience).

	Family Status				
-			Mixed		
Family Member	Adoptive	Biological	Adopted	Biological	
Female Offspring	16.4 (7.3)	17.4 (7.1)	17.0 (7.7)	18.2 (7.1)	
	N=256	N=182	N=47	N=27	
Male Offspring	16.4 (7.3)	16.7 (8.0)	14.2 (7.2)	16.1 (6.7)	
	N=186	N=165	N=49	N=46	
Mothers	21.0 (7.2)	21.5 (7.8)	21.0 (6.9)		
	N=278	N=201	N=119		
Fathers	17.5 (8.3)	18.2 (8.1)	18.6 (8.1)		
	N=253	N=157	N=99		

 Table 1

 Mean (and SD) of Religiousness by Gender and Family Status

Table 2Sibling Correlations (with 95% Confidence Intervals) for Adoptive, Biological, and MixedFamilies by Gender

	Family Status			
- Gender of Sibling Pairs	Adoptive	Biological	Mixed	
male-male	.13 (2144)	.71 (.5283)	.51 (.1973)	
	N = 31	N = 42	N = 26	
female-female	.59 (.4072)	.61 (.3975)	.40 (.0764)	
	N = 60	N = 47	N = 29	
mixed gender	.54 (.3767)	.57 (.3872)	.78 (.4691) [†]	
	N = 82	N = 58	N = 14	
pooled across gender	.49 (.3860)	.63 (.5271)	.52 (.3367)	
	N = 173	N = 147	N = 71	

 † Mixed families with mixed gender were divided into two groups, male elder-female younger and female elder-male younger, but the latter had only two pairs and is not shown in the table.

Table 3 Sibling and Parent-Child Correlations for Religiousness by Family Status: Adopted Families, Biological Families, and Mixed Families (with 1 Adopted and 1 Biological Sibling)

	Biological Fam	ilies (N=208)	
	Sib A	Sib B	Mother
Sib B	.61		
Mother	.63	.66	
Father	.52	.64	.69
	Adopted Famil	lies (N=284)	
	Sib A	Sib B	Mother
Sib B	.51		
Mother	.60	.54	
Father	.55	.51	.64
	Mixed Famili	es (N=124)	
	Adopt Sib	Bio Sib	Mother
Bio Sib	.51		
Mother	.45	.62	
Father	.39	.59	.69

Note. Sibling scores have been age- and sex-corrected.

Table 4

Estimates (and 95% Confidence Intervals) of Genetic and Environmental Parameters from the Full Sibling-Family Model in Mx

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Assortative mating (D)		.65	(.6070)		
Cultural transmission		maternal (m)	.59	(.4869)	
		paternal (f)	.31	(.2142)	
	A-C covariance		.12	(.0319)	
	ы		.36	(.2945)	
	C		.46	(.3756)	
	A		.06	(.0112)	
			Tull model	гип пюцеі (.0112) (.3756) (.2945) (.	

Note. Religiousness scores for siblings are age- and sex-corrected. Estimates from raw data without correction differed by a maximum of .02. A, C, and E represent the standardized genetic, shared environmental, and nonshared environmental effects, respectively.