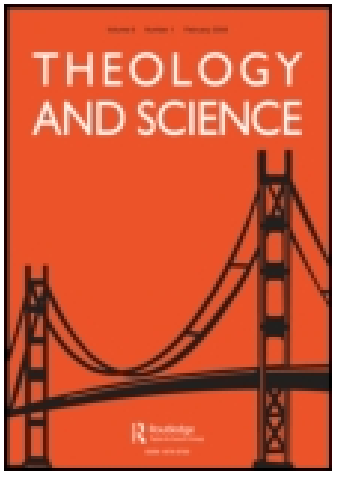

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The Behavioral Genetics of Religiousness

LAURA B. KOENIG and MATT McGUE

Abstract *Behavioral geneticists have sought to characterize the genetic and environmental contributions to individual differences in religiousness. Behavioral genetic methodology is described and twin and adoption studies of religiousness are reviewed. It is concluded that familial resemblance for religiousness is due largely to shared environmental factors in childhood and adolescence, but to genetic factors in adulthood. Additional evidence shows that there is a genetic correlation between religiousness and antisocial and altruistic behavior. Claims for the discovery of a ''God gene'' are premature and unlikely, as any genetic influence is likely to represent the aggregate effect of many genetic factors.*

Key words: Religiousness; twin and adoption studies; heritability; altruism; antisocial behavior

Psychological interest in religion dates from the early twentieth century, when William James[1] wrote his seminal study on the diversity of religious experience. Although part of James' intent was to bring the study of religion into the mainstream of social scientific practice, throughout most of the twentieth century the scientific study of religious behavior and belief developed largely outside the mainstream of the behavioral sciences.[2] The neglect of religion in mainstream social, personality, and clinical psychology is unfortunate and difficult to understand. After all, the majority of individuals in the United States practice religion at some level and state that religion is an important aspect of their lives.[3] Recently, social and behavioral scientists have, however, shown renewed interest in the study of religion, motivated principally by the observation that religiousness is associated with many important life outcomes.[4] Individuals who practice religion regularly have better physical health,[5] live longer,[6] are less likely to have mental health problems,[7] and derive greater satisfaction from life[8] than those who do not practice religion. While the magnitude of most of these associations is modest and the direction of causal influence uncertain, religious behavior is clearly deserving of serious psychological inquiry.[9]

In this article we explore the origins of individual differences in religious beliefs and practices from a behavioral genetic perspective. There are marked individual

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differences in the extent to which people engage in religious behavior and hold religious beliefs, and behavioral geneticists have sought to determine the extent to which genetic and environmental factors are the source of those differences. They have also investigated how religiousness develops over time and changes with age; the basis for the relationships between religiousness and other psychological characteristics; and whether religious behavior can moderate inherited tendencies towards substance abuse and antisocial behavior. While our focus will be primarily on our research program on religiousness, we will also review research investigations from other groups as relevant.

Measurement of religiousness

Religiousness, or the tendency to believe in a higher power and to act in ways consistent with that belief, is clearly a multi-faceted construct that spans attitudes, beliefs, practices, and values. It is consequently not surprising that there is a plethora of ways for measuring religiousness, emphasizing anything from attendance at religious services and knowledge of specific religious dogma all the way through to spirituality and mysticism.[10] Within psychology, much of the approach to the assessment of religiousness derives from the classic work by Allport,[11] who distinguished extrinsic religiousness (i.e. engagement in religious practice in order to satisfy other needs, such as for social contact) from intrinsic religiousness (i.e. religious practice that is motivated out of underlying beliefs and values and is not simply an alternative means to some desired goal). The intrinsic/extrinsic distinction has been a controversial one within the field,[12] which still struggles in achieving a consensus on how best to assess the construct of religiousness.[13]

In our research,[14] we have used a 10-item measure of religiousness that includes one item that asks about religious affiliation (Catholic, Protestant, Muslim, etc.) and nine others that ask about frequency and importance of religious belief and practice. These items are shown in Table 1, along with their response options. Our behavioral genetic analysis has been focused primarily on a total religiousness score, which is created by summing responses to the nine items dealing with religious practice and belief. This score provides a summary of the respondent's religiousness or religiosity, with higher scores indicating that religious beliefs, routines, rituals, and behavior play a central role in the respondent's life. This scale includes some key components of religious behavior while avoiding asking about either the motivations for these behaviors, which are the basis for debate over the intrinsic/extrinsic distinction (e.g. Do you attend church because it's important to you or because it allows you to make friends?) or the specific beliefs the respondent holds (e.g. Do you believe that God created the earth?). Although a nine-item measure cannot possibly capture every important facet of religiousness, the measure does have the virtue of providing an efficient and unitary assessment of the major components of religious practice and belief in a manner typical of self-report measures of trait-like behaviors in psychology. Moreover, our measure has excellent psychometric properties, with estimates of internal consistency reliability of .85 or greater[15] and 4-year stability coefficients ranging from .70 to .81.[16]

Table 1 Items on the Minnesota Religiousness Questionnaire.

Item	*Response options*
How often do you attend religious services?	0 = never 1 = seldom (e.g. on religious holidays)
How often do you seek guidance, help, or forgiveness through prayer?	2 = monthly 3 = weekly
How often do you read scripture or other religious material?	4 = more than once a week
How often do you review or discuss religious teachings with your family?	
How often do you decide moral ''do's'' and ''don'ts'' in religious terms or for religious reasons?	
Do you observe religious holidays and celebrate events like Christmas or Passover in a religious way?	0 = never 1 = sometimes 2 = regularly 3 = always
Do you belong to religious youth or study groups?	0 = no 1 = yes
Do your friends have similar religious beliefs?	0 = no friends have similar religious beliefs 1 = few friends ... 2 = some friends ... 3 = most friends ... 4 = all friends have similar religious beliefs
How important is your religious faith in your daily life?	0 = no importance 1 = some importance 2 = important 3 = very important 4 = extremely important

Behavior genetics methodology

The familial aggregation of religiousness is well established: parents who are religious have children who tend to share their parents' beliefs and practices. In the social sciences, familial resemblance for religiousness has typically been attributed to socialization practices.[17] Myers[18] made the argument most forcibly when he concluded that ''adult *religiosity is determined largely by parental religiosity*'' (p. 864, emphasis in the original). But familial resemblance may owe to common rearing or shared genetic effects, and studies of intact nuclear families cannot help us resolve these possibilities. To characterize the genetic and environmental basis of individual differences, behavioral geneticists have relied primarily on twin studies, adoption studies, and the combination of these, in the study of adopted twins. The rationale for these studies is based on what is called the biometric model.

The biometric modeling approach examines the variance (i.e. variability, or individual differences) in a behavioral trait (otherwise known as a phenotype). The basic assumption of this approach is that the differences between people can

be decomposed (divided) into underlying genetic and environmental effects. The variance for any given phenotype can be explained, in this model, by genetic variance, environmental variance, the covariance between genes and environment, and variance due to gene-environment interaction. Although the investigation of genotype-environment covariance and interaction is an important focus of current behavioral genetic research, both of these effects are usually assumed to be zero in the initial analysis of familial resemblance. (This simplifying assumption is essentially pragmatic, but could potentially bias the resulting biometric parameter estimates discussed below, which consequently should be interpreted as approximations.)[19] Thus, the focus of the current report is the simplified analysis of genetic and environmental influences on variance. Genetic influences can include additive effects, dominant effects, and epistatic effects (the influence of specific combinations of alleles of different genes). The environmental contribution is decomposed into shared (C) and nonshared (E) environmental components. The distinction between shared and nonshared environmental effects represents an important contribution of behavioral genetics to the field and is worth discussing briefly here. The shared-environment component corresponds to the contribution of those environmental factors, such as family socioeconomic status and parental religious practice, that are shared by reared-together relatives, and thus constitute the environmental basis for sibling phenotypic similarity. The nonshared-environment component corresponds to the contribution of those environmental factors, such as peer group, that are not shared by reared-together relatives and thus constitute the environmental basis for their phenotypic differences. The E component also includes measurement error.

Since the variance of a psychological trait is often arbitrary, it is sometimes convenient to set the phenotype variance at 1.0, in which case the biometric decomposition becomes:

$$1.0 = a^2 + c^2 + e^2,$$

where each of the squared lower-case letter terms refers to the proportion of trait variance associated with that specific component. The a^2 here stands for additive genetic influence, often called heritability (the proportion of phenotypic variance due to genetic variance). It is important to remember, however, that, as mentioned above, other sources of genetic effects exist (dominant and epistatic).

The twin study approach

The study of reared-together twins, the classical twin study, is by far the most commonly used research design in human behavioral genetics. Monozygotic (MZ) twins share all of their inherited genetic material, while dizygotic (DZ) twins share on average 50% of their segregating genetic material. If it can be assumed that MZ twins are no more similar on trait-relevant environmental factors than DZ twins (the so-called Equal Environmental Similarity [EES] assumption), then greater MZ than DZ trait similarity implicates the existence of genetic influences. Biometric

models provide a mechanism for formalizing the logic underlying this qualitative comparison. Specifically, if all genetic effects can be assumed to be additive, then the expected MZ and DZ phenotypic correlations are:

$$r_{MZ} = a^2 + c^2$$

$$r_{DZ} = 1/2a^2 + c^2$$

The two equations for expected twin correlations along with the equation for total phenotypic variance can be used to solve for the three unknown variance components to produce the following ACE estimates:

$$a^2 = 2(r_{MZ} - r_{DZ})$$

$$c^2 = 2r_{DZ} - r_{MZ}$$

$$e^2 = 1 - r_{MZ}$$

Alternatively, these estimates can be derived by modeling the observed twin data (including the variances and covariances/correlations) with statistical software like Mx.[20] The models use maximum likelihood procedures to estimate the models' unknown variance components and their associated confidence intervals. These models can also support more complicated data analyses and estimate other paths when appropriate data are included (e.g. they can estimate the assortative mating correlation when spouse or parent-child data are included, and estimate the proportion of a trait correlation that is due to genetic influences when two or more traits are included).

The validity of the classical approach to twin data rests on several key assumptions. Most critical of these is the EES assumption, which has been subject to extensive empirical investigation. In general, there is little evidence to support the proposition that greater environmental similarity is the basis for greater MZ than DZ phenotypic similarity.[21] The generalizability of findings from a twin study also depends on the extent to which twins are representative of the non-twin population. Although twins are more likely than non-twins to experience obstetrical risk, any disadvantages associated with being a twin appear to vanish after the first year of life, and twins appear unremarkable in terms of their cognitive abilities,[22] personalities,[23] and psychopathology risk.[24]

The adoption study approach

The second major research design used by behavioral geneticists is an adoption study. In its idealized form, the logic of an adoption study is relatively straightforward. That is, an adopted individual inherits his or her genes from a set of birth parents and is reared independently by a set of adoptive parents. Consequently, resemblance between the adopted individual and his/her birth

parents should reflect genetic mechanisms, while resemblance between the adopted individual and his/her adoptive parents should reflect (shared) environmental mechanisms. In practice, the evaluation of genetic and environmental effects in an adoption study is not usually so clean. Selective placement could induce a correlation between genetic background and rearing circumstances that would potentially bias both key comparisons in an adoption study. In addition, there is concern that restriction of range in rearing circumstances could downwardly bias estimates of environmental effects in an adoption study.[25] Nonetheless, even though adoptive families do not represent the full range of rearing circumstances, unbiased estimates of environmental effects from adoption studies can be obtained using appropriate statistical corrections.[26]

Perhaps the greatest limitation of adoption research is its feasibility. Unlike records of twin births, which in many countries and US states are a matter of public record, adoption records are private and generally difficult for researchers to access. Consequently, it has been difficult to systematically identify large and representative samples of adopted individuals and their birth and adoptive relatives. Although there are some notable exceptions in the United States,[27] the difficulties in sample ascertainment have resulted in most adoption research being undertaken in Scandinavian countries, where the existence of adoption registers has facilitated systematic sample ascertainment.[28] It is probably for these reasons that there are very few adoption studies of religious behavior.

The study of reared-apart twins

The combination of a twin and an adoption study, the study of reared-apart twins, is in principle the most powerful approach for resolving the separate influences of genetic and environmental factors on individual differences in behavior.[29] In practice, however, reared-apart twins are very rare and difficult to systematically ascertain. As a consequence, there are very few studies of reared-apart twins and to our knowledge only one that has investigated religious behavior.

Twin studies of religiousness

Table 2 provides a summary of major twin studies of various measures of religiousness (including general religiousness [as defined above], church attendance, and religious salience [the importance of religion for the identity of an individual])[30] in terms of estimates of a^2, c^2 and e^2. As can be seen, while most studies report moderate levels of heritability (i.e. between .25 and .50), several studies report more modest estimates of a^2 (i.e. between .00 and .12). Estimates of shared environmental effects, or c^2, show a similar but complementary pattern of heterogeneity, with most estimates in the moderate range (i.e. .30 to .74), but several in the modest range (.02 to .18). One clear contributor to the heterogeneity in the estimates of the biometric components is age of the sample, with estimates

Table 2 Major twin studies of religiousness.

Study	*# pairs*	*Age*	*Measure*	*Biometric components*		
				a^2	c^2	e^2
Kendler et al.[a]	849	30 (Mean)	Personal devotion	.29	.24	.47
	849	30 (Mean)	Personal conservatism	.00	.45	.55
Koenig et al.[b]	273	33 (Mean)	General Religiousness	.44	.18	.38
	273	Childhood (retrospect)	General religiousness	.12	.56	.32
Koenig et al.[c]	231	14 (Mean)	General religiousness	.02	.74	.24
	235	18 (Mean)	General religiousness	.21	.55	.24
	249	20 (Mean)	General Religiousness	.27	.49	.24
	226	25 (Mean)	General religiousness	.46	.30	.24
Bradshaw & Ellison[d]	561	25–74	Religious salience	.27	.33	.40
Kendler & Meyers[e]	1796	8–11 (retrospect)	Church attendance	.24	.50	.26
	1796	12–14 (retrospect)	Church Attendance	.25	.50	.25
	1796	15–17 (retrospect)	Church attendance	.37	.39	.24
	1796	40 (Mean)	Church attendance	.53	.02	.45
Vance et al.[f]	955	43 (Mean)	General religiousness	.56	.09	.35

Notes: a^2 = contribution of additive genetic factors; c^2 = contribution of shared environmental factors; e^2 = contribution of non-shared environmental factors.
a. Kenneth S. Kendler, Charles O. Gardner, and Carol A. Prescott, ''Religion, Psychopathology, and Substance Use and Abuse: A Multimeasure, Genetic-epidemiologic Study,'' *American Journal of Psychiatry* 154:3 (1997): 322–329.
b. Koenig et al., ''Genetic and Environmental Influences,'' 481.
c. Koenig et al., ''Stability and Change,'' 538.
d. Bradshaw and Ellison, ''Do Genetic Factors Influence Religious Life,'' 529–544.
e. Kenneth S. Kendler and John Myers, ''A Developmental Twin Study of Church Attendance and Alcohol and Nicotine Consumption: A Model for Analyzing the Changing Impact of Genes and Environment,'' *American Journal of Psychiatry* 166:10 (2009): 1150–1155.
f. Todd Vance, Hermine H. Maes, and Kenneth S. Kendler, ''Genetic and Environmental Influences on Multiple Dimensions of Religiosity: A Twin Study,'' *Journal of Nervous and Mental Disease* 198:10 (2010): 755–761.

of a^2 being larger in adult than in adolescent samples, while estimates of c^2 tend to be larger in adolescent than in adult samples.

The impact of age on estimates of the biometric parameters has been a particular focus of our research. In Koenig et al.,[31] we asked a sample of 273 adult male twin pairs to report on their religiousness both currently as well as retrospectively when they were children. In adulthood, the estimates were $a^2 = .44$, $c^2 = .18$, and $e^2 = .38$. In childhood, the corresponding estimates were .12, .56, and .32, respectively. This pattern was more evident in the prospective longitudinal twin study by Koenig et al.[32] Figure 1 gives the pattern of estimates of a^2 and c^2 from this study. As can be seen, the estimate of genetic contributions is minimal in early adolescence (.02) but increases monotonically with age until it attains a moderate level of .46 in early adulthood. The estimate of shared environmental contributions shows a converse pattern, being substantial in early adolescence (.74), but declining monotonically with age until it attains a moderate value of .30 at the oldest age.

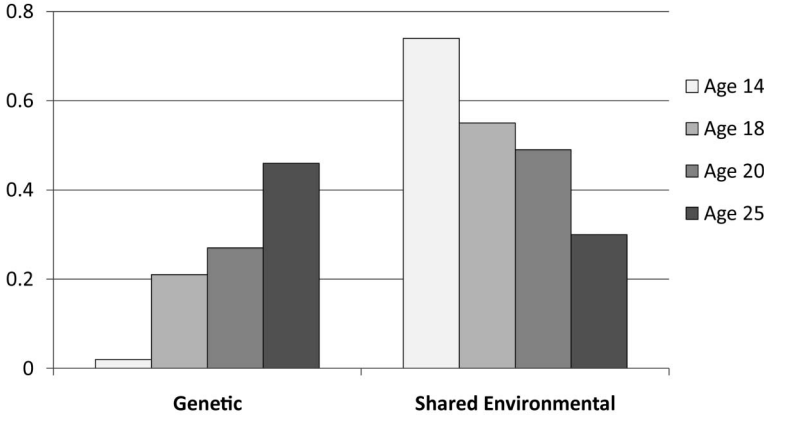

Figure 1 Estimates of genetic (a^2) and shared environmental (c^2) contributions to variance in religiousness as a function of age in the longitudinal twin study of Koenig et al. (Koenig et al., ''Stability and Change,'' 538).

Adoption and reared-apart twin studies of religiousness

Waller et al.[33] is the only published reared-apart twin study of religiousness. The study included 84 pairs of reared-apart twins and also a large sample of 821 pairs of reared-together twins. All reared-apart and reared-together twins were in adulthood and completed multiple measures of religiousness, including the Allport-Vernon-Lindzey Religious Values scale[34] and the Wiggins Religious Fundamentalism scale based on the Minnesota Multiphasic Personality Inventory item pool.[35] Estimates of additive genetic effects (.46 to .52), shared environmental effects (.00 for both scales), and non-shared environmental effects (.48 to .54) were consistent across the two scales and, most importantly, were consistent with studies of reared-together twins in indicating that in adulthood genetic factors are moderate in magnitude while shared environmental factors are modest.

There have been only two published adoption studies of religiousness, both involving adolescent adopted individuals. In a longitudinal study of 654 adolescents whose religious attitudes were assessed annually starting at age 12 and ending at age 15, Abrahamson et al.[36] reported that the estimates of the heritability of a general religiousness scale were non-significant and ranged from .00 to .06, while the contribution of shared environmental factors ranged from a high of .47 at age 12 to a low of .25 at age 15. Similarly, in a sample of 284 adoptive, 208 biological, and 124 mixed adoptive and biological families, with an average child age of 15, Koenig et al.[37] reported that genetic factors accounted for only .06 while shared environmental factors accounted for .46 of the variance in our general religiousness measure. Thus, in demonstrating the predominant contribution of shared environmental contributions to individual differences in adolescent religiousness, the two published adoption studies are consistent with each other and, most importantly, are consistent with studies of adolescent reared-together twins.

Behavior genetic studies of religiousness, antisocial, and altruistic behavior

As discussed above, more complicated biometric models of genetic and environmental influences can be examined, and these models can include more than one phenotype at a time. The goal of these bivariate biometric models is to examine the correlation between traits and to examine the extent to which that correlation is due to shared genetic and/or environmental influences. Instead of examining the difference between the MZ and DZ twin-pair correlations within one trait, these models examine the MZ and DZ difference in cross-twin cross-trait correlations (e.g. MZ Twin 1 trait 1 scores correlated with MZ Twin 2 trait 2 scores). Further research on religiousness has examined the genetic correlation between religiousness and traits like altruism, antisocial behavior, and alcohol use. Results of studies of this nature can, in part, help to explicate how genetic influences may impact differences in religiousness. That is, the genetic effect on religiousness may stem from basic behavioral tendencies that influence other traits as well as religiousness.

Koenig et al.[38] examined 165 MZ and 100 DZ adult male twins' religiousness, antisocial behavior, and altruistic behavior. Antisocial behavior was measured with 27 items on theft, drugs and alcohol, force, and vice. Altruistic behavior was measured with 45 items referencing prosocial behaviors towards friends, acquaintances, strangers, and organizations. Overall, the heritability and shared environmental influences on these three traits provide an interesting comparison. The genetic and environmental influences on adult ratings of these three behaviors were as shown in Table 3. Thus, findings for the heritability of religiousness were similar to those found in other adult samples. The genetic influence on differences in antisocial behavior was also moderate (much like the heritability seen for other aggressive-type problem behaviors), while genetic influence on differences in altruistic behavior was small.

In examining the relationship between religiousness and the other behaviors, the data supported the conclusions that individuals who were more religious were less antisocial and more altruistic. (However, the modest, albeit statistically significant, correlation found between antisocial behavior and religiousness suggests that there are individuals high on both traits, a group that it would be very informative to study.) Antisocial behavior was correlated negatively (−.23) with current religiousness. Altruistic behavior was correlated positively (.24) with

Table 3 Biometric components of adult religiousness, antisocial behavior, and altruism, from Koenig et al.[a]

	a^2	c^2	e^2
Current religiousness	.41	.22	.37
Antisocial behavior	.41	.07	.52
Altruism	.10	.28	.62

Notes: a^2 = contribution of additive genetic factors; c^2 = contribution of shared environmental factors; e^2 = contribution of non-shared environmental factors.
a. Koenig et al., ''Religiousness, Antisocial Behavior, and Altruism,'' 265–290.

current religiousness. Through biometric modeling, the relationships between religiousness and antisocial behavior and altruism were examined at a genetic level. That is, instead of examining the genetic and shared environmental influences on each trait separately (as reported above), the correlation between traits was examined with biometric modeling. It was found that the religiousness-antisocial correlation was due both to genes (A) and to shared environments (C) that influence both behaviors. The same was true for altruism, though altruism also showed genetic and shared environmental effects that were independent of religiousness while the genetic and shared environmental effects on antisocial behavior were entirely shared with religiousness. These types of studies show that understanding genetic effects on differences in religiousness also entails understanding genetic effects on other related traits and behaviors. Religiousness is a complicated trait, and genetic transmission of this trait is not simple.

Discussion and conclusion

Social scientists have typically attributed familial resemblance in religiousness to family socialization practices (i.e. to what behavioral geneticists would call a shared environmental effect). But from a biometric perspective, familial resemblance may owe to the effect of genetic factors (i.e. a^2) or shared environmental factors (i.e. c^2). (Note that non-shared environmental factors, or e^2, are by definition not shared among relatives and so cannot contribute to their behavioral similarity.) Behavioral genetic methodology, specifically in the form of twin and adoption studies, aims to determine the extent to which familial resemblance can be attributed to shared genetic or shared environmental factors.

Our review of existing twin and adoption studies of religiousness reveals a consistent pattern of findings. In childhood and adolescence, familial resemblance for religiousness is due predominantly to shared environmental factors, as socialization researchers predicted. For religious behavior, this is not surprising, as in childhood and adolescence, children in the same family will almost always attend religious services and celebrate religious holidays to the same degree, based on parental/family influences. In adulthood, however, the significance of shared environmental effects on religiousness diminishes as the contribution of genetic factors increases. This pattern of increasing genetic but decreasing shared environmental effects with age is a familiar one in behavioral genetics.[39] For traits as diverse as IQ,[40] social attitudes,[41] parent-child conflict,[42] and anti-social behavior[43] it appears that the impact of the shared family environment is maximal when children are still living with their parents but declines rapidly as the children attain adulthood and establish their independent existence. Conversely, genetic factors may be moderate in childhood and adolescence but grow in importance in age. It is generally recognized that this pattern of findings is due to the increasing role individuals play in shaping their social, physical, and cognitive environments. That is, as we get older our experiences are increasingly a function of the choices we make, and these choices likely reflect our underlying and, in part, genetically influenced dispositions, abilities, and interests. The results

summarized here indicate that religiousness follows this characteristic pattern. The choices we make regarding our religious attitudes, behaviors, and beliefs will not be independent of our dispositions and abilities, and thus will also be genetically influenced.

The existence of significant heritable effects on religiousness, at least in adulthood, implies that differences in the sequence of DNA we inherit have some, admittedly indirect, influence on the extent to which we practice and hold religious belief. In *The God Gene: How Faith is Hardwired into Our Genes*, Dean Hamer[44] makes the bold claim that variation in VMAT2, a gene involved in neurotransmission, influences individual differences in spirituality. Yet everything we have learned from the Human Genome Project makes the likelihood of the existence of a ''God Gene'' essentially nil. Indeed, most attempts to identify the specific genetic variants that underlie heritable effects on behavior have not met the scientific standard of replicability.[45] That certainly is the case for association of VMAT2 with spirituality, as there are no published replications of Hamer's findings. The most likely reason researchers have had difficulty in identifying the specific genetic factors contributing to heritable effects on behavior is not that they do not exist but rather that the relevant effects are very small and thus require very large samples to reliably detect them. For example, a recent study found 180 specific genetic variants affecting individual differences in height,[46] but achieving this finding required a sample of more than 180,000 individuals, and still the vast majority of heritable effects on height went undetected. Similarly, very large samples have been needed to detect specific genetic variants that account for only a minor portion of the heritability of traits as diverse as schizophrenia,[47] diabetes,[48] and plasma lipids.[49] In all likelihood, the specific genetic factors that underlie heritable effects on religiousness will also be hard to detect; they are numerous, small in magnitude, and shared with other aspects of behavior. There is no God gene.

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