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Frequency of church attendance in Australia and the United States: models of family resemblance

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Data on frequency of church attendance have been obtained from separate cohorts of twins and their families from the USA and Australia (29 063 and 20 714 individuals from 5670 and 5615 families, respectively). The United States sample displayed considerably higher frequency of attendance at church services. Sources of family resemblance for this trait also differed between the Australian and US data, but both indicated significant additive genetic and shared environment effects on church attendance, with minor contributions from twin environment, assortative mating and parent–offspring environmental transmission. Principal differences between the populations were in greater maternal environmental effects in the US sample, as opposed to paternal effects in the Australian sample, and smaller shared environment effects observed for both women and men in the US cohort.

Keywords: religion, church attendance, extended kinship model, twins, cultural inheritance, assortative mating, twin environment

Introduction

Frequency of attendance at religious services is an easily determined component of religious behavior, and has been reported to vary as a function of a range of social factors.[1] Numerous studies of religious practice, particularly among the aged, have considered the relationships between church attendance, religiosity and such factors as health, social support and symptoms of depression, with mixed results.[1–6] However, also of interest is church attendance behavior itself, as a characteristic that may or may not be transmitted within families by genetic (eg via personality) or non-genetic (familial/cultural) means, and whose frequency and etiological determinants may differ considerably between cultures.

Religious affiliation has been found in a study of Australian twins and their parents to be transmitted principally by environmental means, with substantial maternal and paternal influences augmented by environmental effects shared by siblings.[7] However, dizygotic (non-identical) twins were found to be more likely to differ in adherence to family traditions after leaving home than monozygotic (identical) twins, indicating the possibility of latent genetic effects influencing adherence to family tradition that first become expressed at this stage. In a study of social attitudes in the same sample, Truett et al[8] obtained estimates of the shared environment contribution to frequency of church attendance to be approximately 46% and 66% in females and males, respectively, with a small but statistically significant contribution from additive genetic effects in females (18%) and no significant genetic effect in males.

A separate study examining family resemblance in a large sample of extended kinships in the USA[9] found genetic contributions to church attendance of 26% and 33% in males and females, respectively, with family environment contributing approximately 21% of variance in both sexes. The more complex modelling technique used in that study incorporated not only the twins and their parents, but also their siblings and children. This allowed closer investigation of a greater diversity of influences on an individual's church attendance behavior, including shared twin and sibling environments, cultural inheritance and phenotypic assortative mating. In this paper we extend Truett et al's investigation[9] by directly comparing Australian and US church attendance data using the correlational methods applied by Truett, as well as maximum likelihood estimation using raw data.

Methods

US cohort

The Virginia 30 000 sample contains data from 14 763 twins, ascertained from two sources.[9] Public

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birth records and other public records in the Commonwealth of Virginia were used to obtain current address information for twins born in Virginia between 1915 and 1971, with questionnaires mailed to twins who had returned at least one questionnaire in previous surveys. A second national group of twins was identified through their response to a letter published in the newsletter of the American Association of Retired Persons (9476 individuals). Twins participating in the study were mailed a 16-page 'Health and Lifestyle' questionnaire, and were asked to supply the names and addresses of their spouses, siblings, parents and children for the follow-up study of relatives of twins. Completed questionnaires were obtained from 69.8% of twins invited to participate in the study, which was carried out between 1986 and 1989.

The original twin questionnaire was modified slightly to provide two additional forms, one appropriate for the parents of twins and another for the spouses, children and siblings of twins. Modifications only affected aspects of the questionnaire related to twinning, in order to obtain self-report data. The response rate from relatives (44.7%) was much lower than that from the twins. Of the complete sample of 29 063 individuals (from 5670 extended kinships) with valid church attendance data, 59.7% were female, with 50% of respondents under 50 years of age.

Australian cohort

Twins for the Australian sample were recruited for two separate 'Health and Lifestyle' studies from the National Health and Medical Research Council Australian Twin Registry (ATR), a volunteer register begun in 1978 which has about 25 000 twin pairs enrolled and in various stages of active contact. The first study, begun in 1988–1990, involved twins registered with the ATR and born prior to 1965 who had previously responded to a mailed questionnaire survey in 1980–1982, whilst the second study was conducted from 1989–1991 and involved ATR-registered twins born between 1964 and 1970. No other major differences existed between the studies, which had an overall twin response rate of 72.3%. As in the US study, modified versions of the original twin questionnaire were sent to parents of twins, and to the twins' spouses, children aged over 18 and siblings aged over 18, with this phase occurring between 1990 and 1992. The response rate from relatives was 58.8%. The complete sample consisted of 20 714 individuals with valid church attendance data, from 5615 families; 12 005 respondents were female (58.0%), and the mean age of the sample was 39.5 (± 15.4) years.

Zygosity determination

In each of the Australian and US samples, zygosity of twins was determined on the basis of responses to standard questions about physical similarity and the degree to which others confused them. This method has been shown to give at least 95% agreement with diagnosis based on extensive blood typing.[10,11] More recently, a sub-sample of 198 same-sex pairs who reported themselves to be MZ twins were typed for 11 independent highly polymorphic markers in the course of an asthma study, with no errors in previous zygosity diagnosis detected.[12]

Measure of church attendance

Self-report data on church attendance were obtained from a single item which asked respondents to indicate the number corresponding to the frequency at which they attend church services. Australian data were scored on a five-point scale: 'rarely', 'once or twice a year', 'every month or so', 'once a week' and 'more than once a week'. The Virginia 30 000 study used a slightly different scale, with six possible response values: 'never', 'rarely', 'a few times a year', 'once or twice a month', 'once a week' and 'more than once a week'.

Statistical methods

The entire data set was corrected for the linear and quadratic effects of age, sex, twin status and interactions between these effects, using SAS 6.11.[13] Data from the US sample was also corrected for source of ascertainment (Virginian birth records vs American Association of Retired Persons). Subsequent analyses are based on the residuals from this regression analysis, which were converted to normal weight scores. Our estimation methods assume multivariate normality and so marginal normality is a necessary, although not sufficient, requirement. Normal weight scores ensure minimal skewness, but for a restricted number of categories (5–6 in our case) will not necessarily ensure minimal kurtosis. Kurtosis in the Australian sample is significant but not large, with values of 0.56 for males, and –0.44 for females, whilst in the US sample kurtosis is not significant (–0.11 for males and < 0.01 for females) (kurtosis defined as $E(x-\mu)^4/\sigma^4 - 3$).

Structural modelling of the data was undertaken using the model described in Truett et al,[9] which assesses the contributions of additive and dominant genetic effects in the presence of effects such as parent-to-offspring environmental transmission ('vertical cultural inheritance'), phenotypic assortative mating, shared twin and sibling environments

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and within-family environment. Phenotypic assortment occurs when like mates with like, with respect to the trait being studied, and is evidenced by a correlation between the observed phenotypes of spouses. Vertical cultural inheritance is the transmission of non-genetic information from parent to child, and refers to the environmental effects of parent on child. Sibling environment effects are those environmental factors shared between all types of offspring reared in the same family. A twin environment is an additional correlation between the environment of twins (in addition to the sibling environment) which makes both MZ and DZ twins more alike than ordinary siblings even in the absence of genetic effects.[14]

Models incorporating these effects were fitted in two ways. First, to the correlations between the regression analysis residuals (as in Truett et al[9]) and second, to the residuals obtained for each individual in a pedigree. In the structural modelling of correlational data, each family member is defined by relationship to the twins within the family, and correlations are calculated for each possible relationship pair. Since some of the expected correlations are identical algebraically under the most simple genetic and cultural inheritance model, correlations can be pooled into groups as defined by the pair's familial relationship. Since pairs of relatives within a family pedigree are not independent from each other, modelling techniques using correlations may result in overestimation of the precision of statistics, although the estimates should be unbiased[15] provided that missing data are missing completely at random.[16] Implementation of structural modelling was via Mx.[17] Due to advances in computational speed and efficiency it is now feasible also to use maximum likelihood methods in modelling genetic and environmental effects in pedigrees of this complexity, allowing us to obtain unbiased estimates and confidence intervals of all parameters. The structural model used in this study is illustrated in Figure 1.

Results

Response frequencies

Response frequencies for the church attendance questionnaire item in the United States and Australian studies are listed in Table 1. Since the age distributions of the two cohorts are quite different, each cohort has been separated into two age groups: those 50 years of age or under (mean- = 33.8 ± 8.0 years and 32.0 ± 8.5 years for the United States and Australia, respectively) and those of over 50 years (65.0 ± 8.6 years; 62.0 ± 8.0 years). In all groups (ie American and Australian men and women) there is a significant increase with age in the proportion of people attending church at least weekly ($P < 0.001$). For each age group, each cohort demonstrates a marked difference between the church attendance behavior of men and women, with greater frequency of church attendance among women in each case ($P < 0.001$).

Direct comparisons of cohort responses for each category are not feasible due to the slightly different structure of the response sets used in each case. However, if the number of response categories is reduced to consider only church attendance once a week or greater vs less often, a direct comparison may be made. A high frequency of church attendance (once a week or more) is substantially more common in the United States cohort than the Australian cohort, for men 50 years or under (29.0% vs 16.1%), men over 50 years (47.8% vs 28.0%), women 50 years or under (36.9% vs 20.3%) and women over 50 years (57.9% vs 38.5%) ($P < 0.001$).

Weighted least squares estimation from correlational data

The correlation values and number of pairs of relatives obtained for each of the 80 possible relationships in each study are shown in Tables 2 and 3 for the Australian and United States data, respectively. Following Truett et al,[9] inspection of these

Table 1 Response frequencies (%) of self-reported church attendance for the United States and Australian cohorts, by age

Cohort	Response frequencies					
United States	Never	Rarely	Few times a year	Once or twice a month	Once a week	More than once a week
Females ≤ 50 (n = 8587)	9.1	20.8	20.6	12.7	24.7	12.2
Females > 50 (n = 8756)	5.2	14.2	13.4	9.3	37.7	20.1
Males ≤ 50 (n = 5904)	11.3	25.8	21.9	12.0	18.3	10.7
Males > 50 (n = 5805)	7.1	20.1	15.0	10.0	32.2	15.6
Australia		Rarely	Once or twice a year	Every month or so	Once a week	More than once a week
Females ≤ 50 (n = 9104)		50.8	19.4	9.5	14.9	5.4
Females > 50 (n = 2901)		38.9	13.5	9.1	26.4	12.1
Males ≤ 50 (n = 6520)		59.3	17.2	7.4	11.7	4.4
Males > 50 (n = 2189)		48.0	14.8	9.2	19.7	8.4

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values is useful as an indicator of the type of model expected to fit the data. For example, the very high spousal correlation (0.69–0.74) observed in both cohorts suggests assortative mating will be required in the model. The substantial sibling (0.28–0.47) and parent–child (0.35–0.49) correlations indicate that

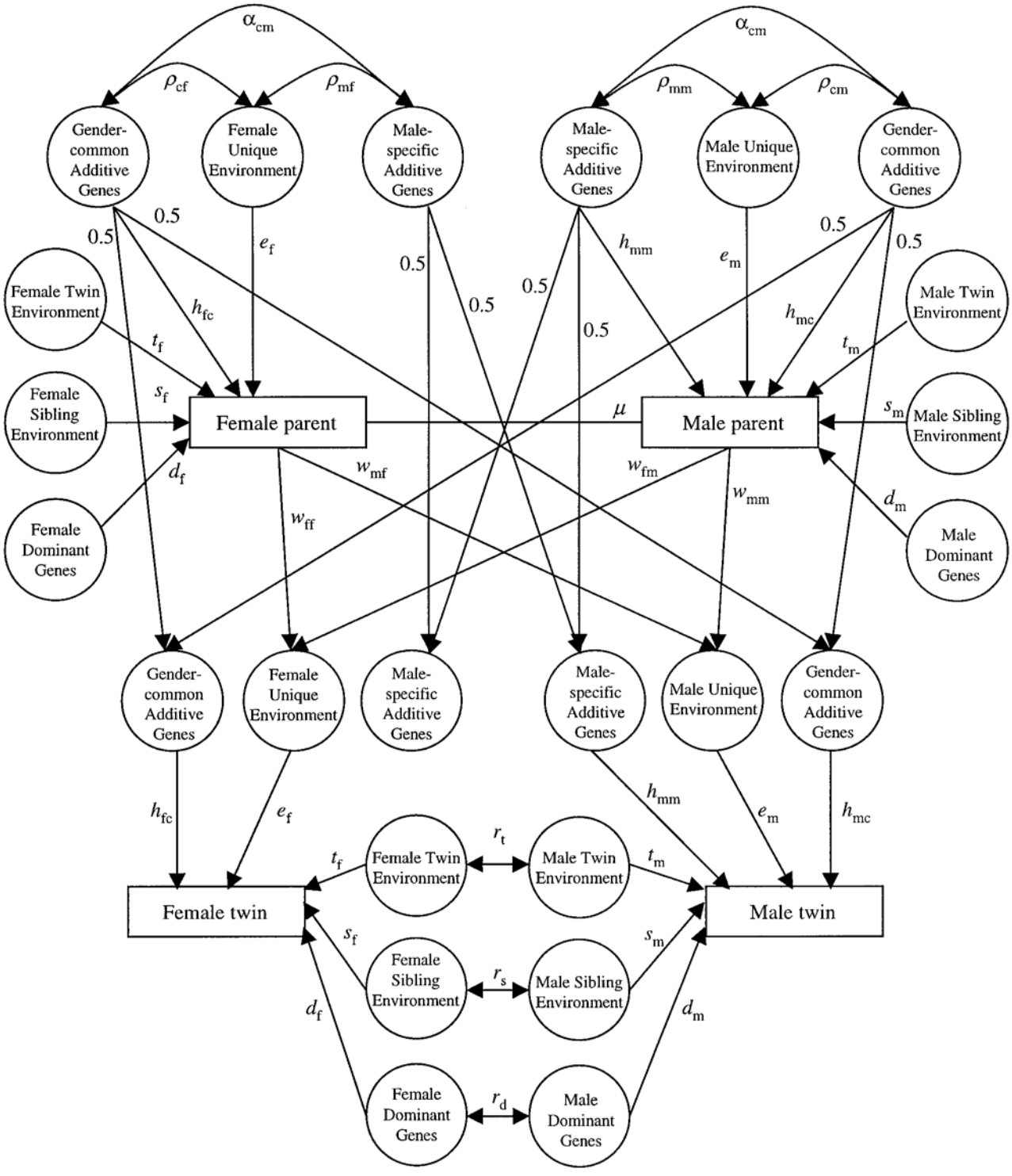

Figure 1 Full extended family resemblance model for opposite-sex DZ twins and their parents. Path coefficients are the same in both generations, and gene–gene and gene–environment correlations occur in both generations

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Table 2 Frequency of church attendance: correlations for relationships in the Australian cohort

	Male–male		Female–female		Male–female		Female–male	
	r	N_{pair}	r	N_{pair}	r	N_{pair}	r	N_{pair}
Nuclear families								
Siblings	0.410	1565	0.467	3344	0.386	4547	–	–
DZ twins	0.487	389	0.531	833	0.410	884	–	–
MZ twins	0.638	630	0.632	1385	–	–	–	–
Parent–child	0.491	2237	0.489	4370	0.488	3064	0.461	2966
Avuncular via[a]								
Father's MZ co-twin	0.361	90	–	–	0.375	111	–	–
Mother's MZ co-twin	–	–	0.403	348	–	–	0.202	291
Father's DZ co-twin	0.354	25	–0.010	51	0.313	42	0.449	32
Mother's DZ co-twin	0.321	46	0.330	194	0.043	90	0.189	107
Father's sibling	0.265	56	0.008	121	0.224	74	0.413	72
Mother's sibling	0.131	197	0.194	292	0.113	239	0.235	226
Cousins via								
Opposite sex DZ twins[b]	0.498	10	–0.119	40	–0.337	20	–0.147	26
MZ father	0.279	78	0.292	94	0.207	172	–	–
MZ mother	0.108	26	0.394	35	0.330	76	–	–
DZ father	–0.263	11	–0.023	36	0.159	49	–	–
DZ mother	0.000	3	0.000	6	0.000	4	–	–
Spouses	–	–	–	–	0.744	3565	–	–
Spouse of twin with[c]								
MZ co-twin	–	–	–	–	0.396	794	0.404	350
DZ co-twin	0.260	208	0.345	196	0.458	406	0.475	201
Sibling of twin	0.333	695	0.266	443	0.391	940	0.378	377
Parent of twin	0.453	543	0.306	402	0.404	751	0.405	315
Spouse of MZ co-twin	0.315	185	0.209	83	–	–	–	–
Spouse of DZ co-twin	0.604	68	0.448	37	0.224	67	–	–
Affine avuncular via[a]								
Father's MZ co-twin	–	–	0.089	60	–	–	0.273	44
Mother's MZ co-twin	0.173	134	–	–	0.282	150	–	–
Father's DZ co-twin	0.284	19	0.968	10	–0.012	35	0.000	5
Mother's DZ co-twin	0.391	31	–0.046	23	0.256	55	0.743	6

[a]Aunt/uncle's sex listed first, niece/nephew's sex listed second; [b]First sex listed is sex of male twin's child; [c]First sex listed is spouse's sex.

there are likely to be genetic or environmental factors influencing family resemblance, whilst differences between correlations for equivalent female and male relationship pairs suggest the possibility that these effects may depend on the sex of the individual. Correlations involving MZ twins are generally, but not consistently, higher than for their DZ same-sex counterparts, suggesting that there may be some genetic influence on family resemblance. Formal testing of the equality of the 80 correlations in the two samples resulted in a highly significant χ^2 ($\Delta\chi^2_{79}$ = 379.5).

Parameter estimates from the structural modelling of correlations are shown in Table4 for both the Australian and United States cohorts. In each case, there is substantial assortative mating (μ), but further comparison of the parameter estimates from the two cohorts reveals substantial differences between the models, which cannot be equated ($\Delta\chi^2_{17}$ = 312.7). In general, genetic effects appear to be greater in the US cohort, whilst twin and shared environmental effects play a greater role in determining the church attendance behavior of Australians. Estimates of vertical cultural transmission effects (w_{ff}, w_{mf}, w_{fm}, w_{mm}) are greater for the Australian cohort than for the US cohort, particularly paternal effects (w_{fm}, w_{mm}).

Parameter estimates may be used to derive proportions of variance attributable to various genetic and environmental effects (Table5). The 95% confidence intervals are obtained from Mx using the method of Neale and Miller.[18] From these results it is evident that major differences do exist between the estimates obtained for the two cohorts. Additive and non-additive genetic effects were not found to be significant for the sample of Australian males and females, but have a statistically significant effect for men and women in the United States sample. Conversely, estimates of the total contribution of shared environment (including twin environment) are higher for Australians than for the United States cohort. In particular, cultural transmission effects are very small for the US cohort, but contribute approximately 18% and 23% of phenotypic variance in Australian females and males, respectively. Genotype-environment covariance accounts for only 6–8% of phenotypic variance in the Virginian cohort and is not statistically significant in the Australian data.

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Table 3 Frequency of church attendance: correlations for relationships in the United States cohort

	Male–male		Female–female		Male–female		Female–male	
	r	N_{pair}	r	N_{pair}	r	N_{pair}	r	N_{pair}
Nuclear families								
Siblings	0.283	1308	0.330	3433	0.331	3145	–	–
DZ twins	0.386	540	0.423	1087	0.302	1226	–	–
MZ twins	0.503	737	0.591	1725	–	–	–	–
Parent–child	0.350	2069	0.398	4280	0.366	2871	0.359	2869
Avuncular via[a]								
Father's MZ co-twin	0.240	211	–	–	0.212	325	–	–
Mother's MZ co-twin	–	–	0.259	1005	–	–	0.226	639
Father's DZ co-twin	–0.147	104	0.226	173	0.268	138	0.070	113
Mother's DZ co-twin	0.051	146	0.234	505	0.212	194	0.191	318
Father's sibling	0.312	50	0.227	190	0.075	134	0.102	77
Mother's sibling	0.270	126	0.110	506	0.183	206	0.158	282
Cousins via								
Opposite sex DZ twins[b]	–0.330	16	–0.031	66	0.008	36	–0.049	47
MZ father	0.217	21	0.150	94	0.259	76	–	–
MZ mother	0.315	81	0.288	335	0.227	309	–	–
DZ father	–0.147	11	–0.189	42	–0.095	33	–	–
DZ mother	0.239	29	0.111	142	0.232	101	–	–
Spouses	–	–	–	–	0.691	4561	–	–
Spouse of twin with[c]								
MZ co-twin	–	–	–	–	0.430	1084	0.377	592
DZ co-twin	0.126	338	0.218	416	0.394	573	0.344	393
Sibling of twin	0.181	421	0.257	444	0.254	699	0.185	352
Parent of twin	0.225	183	0.260	276	0.289	312	0.207	196
Spouse of MZ co-twin	0.404	288	0.263	174	–	–	–	–
Spouse of DZ co-twin	0.414	114	0.435	100	0.154	156	–	–
Affine avuncular via[a]								
Father's MZ co-twin	–	–	0.228	219	–	–	0.220	125
Mother's MZ co-twin	0.243	338	–	–	0.245	478	–	–
Father's DZ co-twin	0.048	68	0.292	82	–0.065	86	0.043	57
Mother's DZ co-twin	0.315	115	–0.210	62	0.313	164	0.072	34

[a]Aunt/uncle's sex listed first, niece/nephew's sex listed second; [b]First sex listed is sex of male twin's child; [c]First sex listed is spouse's sex.

Maximum likelihood estimation from individual observations

Table 6 lists the model parameter estimates obtained using maximum likelihood methods. As we found for the results obtained from correlational data, the models for the United States and Australian cohorts cannot be equated, although the heterogeneity is even more marked ($\Delta\chi^2_{19}$ = 549.5 vs $\Delta\chi^2_{17}$ = 312.7). The principal source of heterogeneity appears to be that maternal cultural transmission is stronger in the US cohort, whilst paternal cultural transmission is stronger in the Australian sample.

Maximum likelihood estimates of the proportions of variance from analysis of individual observations are shown in Table 7. Additive genetic effects from genes expressed in both sexes were found to be significant for males and females in both cohorts. No significant male-specific additive genetic effects were observed in either sample. Small non-additive genetic effects were found for females in both cohorts, but not for males. Despite the high level of assortative mating for church attendance behavior (0.74 and 0.69 in the Australian and United States cohorts, respectively), the amount of phenotypic variance resulting from the consequent increase in additive genetic variance is modest, ranging from 4% to 10%.

Unique environment was estimated by the maximum likelihood method to account for over 36% of all phenotypic variance in all groups. Estimates of common environment effects tend to be greater for males than for females, and greater in the Australian cohort than in the United States cohort. Statistically significant twin environment and cultural transmission effects were observed in both cohorts, although these are quite small in magnitude. Estimates of the genotype–environment covariance range from 3% to 8%.

Discussion

Very similar results were obtained for the United States cohort from correlational data and by using maximum likelihood methods. However, the models obtained via these two methods for the Australian data differed substantially, with smaller cultural

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Table 4 Comparison of model parameter estimates for frequency of church attendance from the Australian and United States cohorts. Estimates obtained from correlations between biological relationships

Genetic parameters			Environmental parameters			Other parameters		
	Australian	US		Australian	US		Australian	US
h_{fc}	0.309	0.525	t_f	0.287	0.350	μ	0.748	0.699
h_{mc}	0.115	0.465	t_m	0.322	0.343	ρ_{cf}	0.191	0.114
h_{mm}	0.000	0.000	r_t	0.171	0.208	ρ_{cm}	0.202	0.103
α_{cm}	0.000	0.000	s_f	0.381	0.247	ρ_{mf}	0.000	0.000
d_f	0.155	0.199	s_m	0.346	0.269	ρ_{mm}	0.000	0.000
d_m	0.354	0.145	r_s	0.802	1.000	rel_f	1.000*	1.000*
r_d	–0.332	1.000	w_{ff}	0.281	0.188	rel_m	1.000*	1.000*
			w_{mf}	0.205	0.139			
			w_{fm}	0.323	0.046			
			w_{mm}	0.444	0.073			
			e_f	0.752	0.651			
			e_m	0.776	0.711			

* Fixed parameter
h_{fc} = gender-common additive genetic path parameter – females
h_{mc} = gender-common additive genetic path parameter – males
h_{mm} = male-specific additive genetic path parameter – males
α_{cm} = induced correlation between gender-common and male-specific additive genetic paths
d_f = non-additive genetic path parameter – females
d_m = non-additive genetic path parameter – males
r_d = correlation between male and female non-additive genetic effects
t_f = special twin environment – females
t_m = special twin environment – males
r_t = correlation between male and female special twin environments
s_f = common environment path parameter – females
s_m = common environment path parameter – males
r_s = correlation between male and female common environment
w_{ff} = maternal cultural transmission – females
w_{mf} = maternal cultural transmission – males
w_{fm} = paternal cultural transmission – females
w_{mm} = paternal cultural transmission – males
e_f = specific environment – females
e_m = specific environment – males
μ = assortative mating parameter
ρ_{cf} = correlation between gender-common additive genetic effects and environment – females
ρ_{cm} = correlation between gender-common additive genetic effects and environment – males
ρ_{mf} = correlation between male-specific additive genetic effects and environment – females
ρ_{mm} = correlation between male-specific additive genetic effects and environment – males
rel_f = reliability of measured phenotype in estimating latent variable – females (fixed to 1)
rel_m = reliability of measured phenotype in estimating latent variable – males (fixed to 1)

Table 5 Comparison of variance components for frequency of church attendance from the Australian and United States cohorts. Variance components and 95% confidence intervals estimated from correlations between biological relationships

Males	Genetic effects					Shared environment effects		
	Gender-common additive genetic (A_m)	Male-specific additive genetic (B_m)	Due to assortative mating	Non-additive genetic effects (D_m)	Unique environment (E_m)	Common environment (C_m)	Twin environment (T_m)	Cultural transmission (C_{mct})
Australia	0.012 (0.000–0.245)	0.000 (0.000–0.000)	0.001 (0.000–0.064)	0.125 (0.000–0.237)	0.377 (0.034–0.422)	0.120 (0.037–0.173)	0.103 (0.026–0.178)	0.226 (0.053–0.288)
USA	0.167 (0.060–0.281)	0.000 (0.000–0.000)	0.049 (0.011–0.106)	0.021 (0.000–0.138)	0.486 (0.443–0.529)	0.072 (0.024–0.130)	0.118 (0.049–0.180)	0.020 (<0.001–0.075)

Females	Genetic effects					Shared environment effects		
	Gender-common additive genetic (A_f)	Male-specific additive genetic	Due to assortative mating	Non-additive genetic effects (D_f)	Unique environment (E_f)	Common environment (C_f)	Twin environment (T_f)	Cultural transmission (C_{fct})
Australia	0.087 (0.000–0.211)	– –	0.009 (0.000–0.053)	0.024 (0.000–0.145)	0.384 (0.355–0.414)	0.145 (0.090–0.201)	0.082 (0.032–0.130)	0.181 (0.076–0.299)
USA	0.213 (0.109–0.318)	– –	0.062 (0.022–0.119)	0.040 (0.000–0.136)	0.404 (0.377–0.431)	0.061 (0.026–0.101)	0.122 (0.072–0.170)	0.021 (0.001–0.070)

transmission and greater additive genetic effect estimates obtained using the maximum likelihood analysis technique. Since the maximum likelihood method uses all available pedigree information and avoids the additional assumptions of the correlational method, it is probable that these estimates provide a more accurate model for family resemblance in church attendance behavior.

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Table 6 Comparison of model parameter estimates for frequency of church attendance from the Australian and United States cohorts. Estimates obtained using maximum likelihood methods

Genetic parameters			Environmental parameters			Other parameters		
	Australian	US		Australian	US		Australian	US
h_{fc}	0.397	0.432	t_f	0.318	0.199	μ	0.736	0.694
h_{mc}	0.422	0.549	t_m	–0.119	–0.248	ρ_{cf}	0.159	0.121
h_{mm}	0.332	0.000	r_t	1.000	1.00	ρ_{cm}	0.027	0.046
α_{cm}	0.108	0.000	s_f	0.357	0.268	ρ_{mf}	–0.073	0.000
d_f	0.240	0.359	s_m	0.340	0.218	ρ_{mm}	–0.017	0.000
d_m	0.000	0.055	r_s	0.753	1.000	rel_f	1.000*	1.000*
r_d	1.000	1.000	w_{ff}	0.060	0.195	rel_m	1.000*	1.000*
			w_{mf}	–0.072	0.129			
			w_{fm}	0.270	0.008			
			w_{mm}	0.138	–0.048			
			e_f	0.674	0.638			
			e_m	0.540	0.725			

*Fixed parameter

h_{fc} = gender-common additive genetic path parameter – females
h_{mc} = gender-common additive genetic path parameter – males
h_{mm} = male-specific additive genetic path parameter – males
α_{cm} = induced correlation between gender-common and male-specific additive genetic paths
d_f = non-additive genetic path parameter – females
d_m = non-additive genetic path parameter – males
r_d = correlation between male and female non-additive genetic effects
t_f = special twin environment – females
t_m = special twin environment – males
r_t = correlation between male and female special twin environments
s_f = common environment path parameter – females
s_m = common environment path parameter – males
r_s = correlation between male and female common environment
w_{ff} = maternal cultural transmission – females
w_{mf} = maternal cultural transmission – males
w_{fm} = paternal cultural transmission – females
w_{mm} = paternal cultural transmission – males
e_f = specific environment – females
e_m = specific environment – males
μ = assortative mating parameter
ρ_{cf} = correlation between gender-common additive genetic effects and environment – females
ρ_{cm} = correlation between gender-common additive genetic effects and environment – males
ρ_{mf} = correlation between male-specific additive genetic effects and environment – females
ρ_{mm} = correlation between male-specific additive genetic effects and environment – males
rel_f = reliability of measured phenotype in estimating latent variable – females (fixed to 1)
rel_m = reliability of measured phenotype in estimating latent variable – males (fixed to 1)

Table 7 Comparison of variance components and 95% confidence intervals for frequency of church attendance from the Australian and United States cohorts. Variance components estimated using maximum likelihood methods

Males	Genetic effects					Shared environment effects		
	Gender-common additive genetic (A_m)	Male-specific additive genetic (B_m)	Due to assortative mating	Non-additive genetic effects (D_m)	Unique environment (E_m)	Common environment (C_m)	Twin environment (T_m)	Cultural transmission (C_{mct})
Australia	0.221 (0.026–0.442)	0.136 (0.000–0.360)	0.058 (0.002–0.171)	0.000 (0.000–0.079)	0.359 (0.322–0.401)	0.143 (0.069–0.218)	0.016 (0.000–0.082)	0.003 (0.000–0.027)
USA	0.278 (0.145–0.375)	0.000 (0.000–0.000)	0.100 (0.039–0.162)	0.003 (0.000–0.119)	0.480 (0.438–0.523)	0.044 (0.004–0.102)	0.057 (0.008–0.112)	0.005 (<0.001–0.033)

Females	Genetic effects					Shared environment effects		
	Gender-common additive genetic (A_f)	Male-specific additive genetic	Due to assortative mating	Non-additive genetic effects (D_f)	Unique environment (E_f)	Common environment (C_f)	Twin environment (T_f)	Cultural transmission (C_{fct})
Australia	0.154 (0.021–0.199)	– –	0.040 (0.003–0.114)	0.056 (0.000–0.178)	0.407 (0.378–0.439)	0.124 (0.067–0.180)	0.099 (0.041–0.155)	0.037 (0.003–0.127)
USA	0.193 (0.098–0.308)	– –	0.069 (0.026–0.136)	0.133 (0.038–0.220)	0.404 (0.379–0.430)	0.074 (0.030–0.119)	0.041 (0.005–0.087)	0.017 (0.001–0.058)

Both genes and environment have been demonstrated to have a significant role in church attendance behavior in both cohorts. Although models fitted to the data from the two cohorts could not be equated, major influences on individual differences in church attendance in both cohorts appear to be additive genetic (15–35%), common environment (7–14%) and unique environment (35–48%) effects, with small contributions from assortative mating (< 10%), twin environment (< 10%) (which could

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also arise from genotype × age interaction), non-additive genetic effects (< 5%) and cultural transmission (< 5%).

Possible explanations of the low contribution of cultural transmission to church attendance behavior have been discussed elsewhere.[9] Differences in the size of genetic effects between the two cohorts may be attributed to different environmental conditions experienced by the two cohorts,[19] whilst the differences observed between the models obtained from weighted least squares estimation from correlational data and maximum likelihood estimation from individual observations indicate that the assumption of data missing at random may not be valid, particularly in the Australian sample.

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