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Religious Affiliation in Twins and Their Parents: Testing a Model of Cultural Inheritance

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The transmission of religious affiliation is analyzed in a sample of 3810 Australian twin pairs and their parents. Twins were classified by sex, zygosity, and whether they were living together or apart. Analysis of twin, spousal, and parent-offspring resemblance shows that several different forms of cultural inheritance operate jointly in the transmission of religious affiliation. Model-fitting methods show that (1) the environmental influence of mothers is significantly greater than fathers; (2) there is a substantial amount of assortative mating for religious affiliation; (3) there is a substantial environmental component shared by twins which does not depend on parental religious affiliation; (4) religious affiliation attributed to parents by their children is biased by the religious affiliation of the children; (5) nongenetic effects on the expression of religious affiliation are much greater in twins living together; and (6) a moderate genetic effect on religious affiliation is expressed in females but only when twins live apart. Implications of the method and findings are discussed for other aspects of family resemblance, including the analysis of social and occupational mobility.

KEY WORDS: twins; religion; cultural inheritance; assortative mating; rater bias.

INTRODUCTION

"So important is the educational system in transmitting familial socio-economic advantages and disadvantages to offspring that some have speculated that the

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achievement of 'equality of opportunity' would require major changes in the family system as we know it" (Alwin and Thornton, 1984). The demonstration that almost any aspect of human variation aggregates in families is one of the most universal and robust findings to emerge from a century of empirical study in the life and social sciences. Beginning with Francis Galton's first attempts to quantify the familial transmission of "genius" (1869), there have been major analyses of familial factors in the maintenance of occupational structure (e.g., Blau and Duncan, 1967; Goldthorpe, 1980; Heath, 1981), inequality of educational attainment (Jencks, 1972; Alwin and Thornton, 1984), income and property ownership (e.g., Behrman, *et al.*, 1977; Henretta, 1984), poverty and deprivation (e.g., Rutter and Madge, 1976), and delinquency (e.g., Rowe and Osgood, 1984). More recent studies have demonstrated that the degree of family resemblance for a given variable is not a universal constant but may change significantly when the social structure is transformed (e.g., Simkus, 1984; Heath *et al.*, 1985a) and alter as a function of age (e.g., Alwin and Thornton, 1984; Eaves *et al.*, 1986).

Such findings in the behavioral and social sciences are matched by a substantial catalog of family resemblance for anthropometric and physiological variables accumulated by researchers in the life and medical sciences (e.g., Pearson and Lee, 1903; Holt, 1968; Sing and Skolnick, 1979). The fact that similar empirical results should appear in two widely different disciplines is less remarkable than the fact that, with notable exceptions, the theories used by biologists and sociologists to interpret such data are almost diametrically opposed. A single set of parent–offspring correlations might be interpreted in purely cultural terms by a sociologist and in genetic terms by a physiologist, yet neither has any reason beyond the mainstream of prejudice in his discipline to prefer either interpretation. In nuclear families parents who transmit their genes to their children may also create major features of their environment. Such "genotype–environment correlation" was dubbed the "double advantage" phenomenon by Jencks *et al.* (1972).

It is tempting to argue that a social interpretation of family resemblance is more likely when the similarity between relatives differs between cultures, is correlated with changes in social structure, changes with age, or alters as a consequence of life events. Such a view is unjustified. Extensive experimental data (e.g., Mather and Jinks, 1982) have shown how response to environmental influences can be partly under genetic control so that the parameters of a purely genetic model are expected to change under different environmental conditions. Heath *et al.* (1985) recently showed that estimates of the genetic contribution to family resemblance for educational attainment increased in Norway after the Second World War following a major increase in access to higher education. Broadhurst and Jinks (1966) showed how the expression of genetic factors in the rat changed significantly with age as a function of changing adaptive re-

quirements during development. Eaves *et al.* (1986) devised a theoretical genetic model for developmental change in human families which gave a significantly better fit to empirical data on cognitive development than genetic models which ignored developmental changes in gene expression. The same empirical issues—secular and developmental change in family resemblance—are addressed by both geneticists and sociologists but with radically different theories.

Any attempt to integrate biological and social models of the intergenerational stability and change, whether or not it deals only with a single culture and age group, requires a strategy which is able to resolve social advantages from biological advantages. The "double advantage" phenomenon ensures that ordinary nuclear family data are no use for this purpose. Other strategies which have been suggested in the past are the study of adoptees with their foster parents and/or their biological parents, the comparison of identical and nonidentical twins, and the study of twins and various constellations of their relatives. Adoption designs have been used mainly for the study of psychiatric disease and psychometric variables (e.g., Horn *et al.*, 1979; Plomin and Defries, 1985) but have not been exploited much in the sociological domain. Twin studies have been conducted for socioeconomic variables (e.g., Behrman *et al.*, 1980) and other socially important traits including delinquency (Rowe and Osgood, 1984). Such studies have suggested that genetic factors do indeed contribute, at least in part, to the correlations between relatives for variables often studied by social scientists.

Twin studies suffer from two main potential weaknesses. The first relates to the basic assumption of the twin method that only genetic effects show a different correlation between monozygotic (MZ) and dizygotic (DZ) twins (e.g., Goldberger, 1976). If MZ twins experience more similar environments than DZ's, then the contribution of genetic effects may be overestimated if these aspects of the environment affect the trait in question. We address this issue again in our Discussion. A detailed evaluation of the major criticisms of the twin method is provided by Kendler (1983), who concluded that the empirical data do not, on the whole, support these criticisms. Although the twin study is able to detect the contribution of genetic effects, and estimate any effects of the shared environment (granted the assumption of equal environments and additive gene action), it is a relatively blunt instrument for the resolution of different environmental sources of family resemblance. Without supplemental data, the classical twin study cannot resolve the genetic consequences of assortative mating from the social effects of cultural inheritance (e.g., Martin *et al.*, 1986) and cannot separate the various sources of nongenetic resemblance which are important for understanding the social impact of parents on their children. If we are prepared to assume that mating is random [which is manifestly not so for socioeconomic variables; see Heath (1981)], then the twin method can yield an overall estimate of the contribution of the family environment to the correlation

between relatives. However, the resolution of the shared environmental component into that contributed by mothers, fathers, peers, and the mutual reinforcement of twins' behavior is impossible with twins alone.

Young *et al.* (1980) proposed an augmented twin design in which parents of twins were studied at the same time as the twins themselves. Such a design provides the information necessary for a first-order resolution of the effects of biological and cultural inheritance and assortative mating, together with the opportunity to discriminate between maternal and paternal sources of nongenetic inheritance. Subsequent theoretical analyses have shown how more subtle effects can be resolved when the twin design is augmented by the offspring of twins (Nance and Corey, 1976), the spouses of twins, and the twins' parents-in-law (Heath and Eaves, 1985). Recent simulation studies by Heath *et al.* (1985b) suggested that the study of twins and their parents was, in many circumstances, the most productive strategy for initial attempts to resolve different aspects of biological and cultural inheritance.

We demonstrate the power and flexibility of the augmented twin design with data on religious affiliation in twins and their parents. Religious affiliation was chosen for study because, unlike many socioeconomic variables, the adoption of different roles after marriage does not preclude its adequate measurement in both sexes and because, of the many sociological and psychometric traits which could be analyzed, religious affiliation is that for which there is the greatest a priori expectation of cultural inheritance. The variable will thus give us the best chance of demonstrating the power of the design to address cultural as well as biological inheritance. In addition, we shall examine the sensitivity of the mechanism of inheritance to changes in the social network surrounding individuals by exploring the differences between parameters derived from kinships of twins living together and apart.

THE SAMPLE

The data were obtained from twins who had joined the Australian Twin Registry (Martin and Jardine, 1986). Between 1980 and 1982 a 12-page questionnaire booklet was mailed to 5967 pairs of volunteer twins enrolled in the registry. Completed questionnaires were obtained from 3810 *pairs* of twins, i.e., 64% of the original sample. The twins ranged in age from 18 to 88 years. Each twin was asked to record his/her own religious affiliation and that of both parents separately in one of eight categories (Table I). For each parent, therefore, we have a separate report of religious affiliation from each twin. In addition, each pair was recorded as living together (T) or living apart (A) at the time of study. Table I summarizes the frequencies of the eight response categories in male and female twins and their parents.

Table I. Raw Frequencies of Religious Affiliation for Individual Twins and Their Parents

		Male Twins						Female Twins					
		Twin		Mother		Father		Twin		Mother		Father	
Code	Affiliation	*N*	%	*N*	%	*N*	%	*N*	%	*N*	%	*N*	%
1	No religion	401	15	99	4	208	8	413	8	135	3	306	6
2	Anglican	846	31	1034	38	984	36	1572	32	1738	36	1687	35
3	Other Protestant	804	29	895	33	857	31	1618	33	1711	35	1681	34
4	Catholic	559	20	601	22	582	21	1072	22	1121	23	1040	21
5	Jewish	25	1	31	1	37	1	40	1	48	1	48	1
6	Greek or Russian Orthodox	20	1	22	1	23	1	25	1	29	1	28	1
7	Other	49	2	42	2	32	1	99	2	72	2	55	1
8	Prefer not to answer	41	1	21	1	22	1	36	1	21	0	30	1
Total		2745		2745		2745		4875		4875		4875	

Indices of Family Resemblance

Students of social mobility have invested considerable effort in devising scales of occupation which reflect the structure and relative "permeability" of social barriers to intergenerational mobility (e.g., Goldthorpe, 1980; Breiger, 1981). Typically, genetic studies have dealt with either continuous variables in which the problems of scaling do not occur or discontinuous variables in which the categories can be represented as discontinuous manifestations of a continuous latent trait. In either case, the starting point for modeling family resemblance has been the correlation coefficient, the covariance matrix between relatives (Young *et al.*, 1980), or estimates of the polychoric correlation coefficient (e.g., Eaves *et al.*, 1978; Kendler *et al.*, 1986). Clearly, religious affiliation cannot be scaled so easily. We have two alternatives: (1) devise a scale which reflects the pattern of intergenerational change in affiliation and then work out how this reflects underlying biological and social differences; or (2) employ a statistic to summarize the similarity between relatives which is agnostic about the issue of scale and try to test alternative hypotheses about the social and biological causes of similarity without reference to specific issues of scaling. The first alternative is, theoretically, the most attractive because ultimately issues of cause and scale are inseparable. However, we adopt the second strategy on the grounds that it is more tractable and focuses on modeling mechanisms of inheritance which transcend the fundamental problems of scale.

The data on religious affiliation are treated as nominal categories and summarized by the symmetric coefficient of uncertainty, $0<U<1$ (Goodman and Kruskal, 1979), reflecting the degree to which the cells of a two-way table can

be predicted from the marginal frequencies. In our case, the rows and columns in a table are formed by the religious affiliations of, for example, mothers and their first twins or first and second twins. A typical cell in a table contains the number of pairs in the sample who show a particular combination of reported affiliations.

Values for U were generated with the FREQ procedure of the Statistical Analysis System (SAS Institute, 1985) using the formula

$$U = 2[H(X) + H(Y) - H(X,Y)]/[H(X) + H(Y)].$$

The "H"'s are negative log-likelihoods, ignoring the constant term. $H(X)$ is the negative log-likelihood of the cell frequencies given the column frequencies; $H(Y)$ is the negative log-likelihood given the row frequencies; and $H(X,Y)$ is the negative log-likelihood when each cell is allowed to take its own frequency. Carey (personal communication) has pointed out that, for the dichotomous case, the value of U is also affected somewhat by the marginal frequencies of the categories. Further study would be needed to determine how far differences of the size reported here would affect the results of our model-fitting analysis.

In practice, the square roots of the U statistics for our data are quite close to the product-moment correlations obtained when the raw category codes are used as ordinal values. Table II gives the U statistics for twin pairs divided into those living together and apart. We omitted responses in the "prefer not to answer" category. The twins living together are consistently more alike than those living apart, suggesting that twins who live together experience more similar environmental effects or that the decision to live apart is partly a function of religious differences. With the possible exception of the female twins living apart, the U's for MZ and DZ twins are very similar, confirming that genetic factors play little or no role in the determination of religious affiliation. In most cases, the similarities in religion are greater for female than male twins.

Table III gives the U's for twins' reports of their parents' religious affiliation

Table II. Symmetric Coefficients of Uncertainty (U) and Sample Sizes (N Pairs) for Religious Affiliation of Family Members

	Twin pairs				
	Living together (T)		Living apart (A)		
Twin type	U	N	U	N	Total
MZ_f	0.802	205	0.554	1008	1213
MZ_m	0.727	124	0.453	431	567
DZ_f	0.813	108	0.426	632	751
DZ_m	0.718	81	0.515	252	352
DZ_{mf}	0.706	187	0.423	702	907

Table III. Interobserver Consistency Based on Twins' Reports of Same Parent

		Reporting on			
		Mother		Father	
Twin type	T/A	*U*	*N*	*U*	*N*
MZ_f	T	0.786	205	0.851	203
MZ_m	T	0.789	127	0.736	128
DZ_f	T	0.868	111	0.814	111
DZ_m	T	0.738	82	0.773	82
DZ_{mf}	T	0.792	190	0.770	189
MZ_f	A	0.756	1015	0.691	1013
MZ_m	A	0.719	436	0.626	435
DZ_f	A	0.733	635	0.683	633
DZ_m	A	0.650	261	0.634	261
DZ_{mf}	A	0.652	705	0.571	705

omitting responses in the "prefer not to answer" category. Similarity between the reports of the same parent by different twins may reflect (a) the extent to which twins' reports are valid reflections of the "true" parental affiliation; and (b) the extent to which the correlation in twins' own religious affiliations introduces a spurious impression of validity because of an inherent attributional bias in reporting which tends to ascribe to others values like those of the reporter. The raw "consistency" statistics are at least as high as the *U*'s for the twins, suggesting that the "validity" might be high, but a cautionary note is sounded by the fact that the similarity is slightly greater for twins living together as might be expected if there were also reporting bias based on the religious affiliations of the twins.

The various estimates of *U* for spouses which may be derived from the data are given in Table IV. These are large, and since they are approximately the squares of corresponding correlations, they point to far greater assortative mating or spousal interaction for religion than for most other variables (e.g., Heath, 1987; Vandenberg, 1972; Eaves *et al.*, 1986). We notice that the estimates are higher for twins living together than for separated twins. This could reflect truly greater similarity between the parents of twins living together or be another manifestation of twins' reporting biases. The latter interpretation derives further support from the fact that the *U* statistics are higher when the same twin reports on both parents, in contrast to the case when each parent's affiliation is recorded by a different twin.

Without recourse to any mathematical model, we see that the data so far support a largely "nongenetic" model for religious affiliation and indicate an important contribution of assortative mating. However, they do not address the important sociological issue of the extent to which the similarity between chil-

Table IV. Spousal Resemblance for Religious Affiliation Based on Twins' Reports of their Parents

		Twins' reports of							
Mother reported by		T1		T2		T1		T2	
Father reported by		T1		T2		T2		T1	
Twin type	T/A	*U*	*N*	*U*	*N*	*U*	*N*	*U*	*N*
MZ_f	T	0.471	204	0.470	205	0.416	204	0.460	204
MZ_m	T	0.550	128	0.546	128	0.500	127	0.489	128
DZ_f	T	0.545	111	0.550	112	0.496	111	0.500	111
DZ_m	T	0.525	83	0.479	83	0.481	82	0.439	82
DZ_{mf}	T	0.536	190	0.535	190	0.499	189	0.457	190
MZ_f	A	0.367	1018	0.382	1014	0.328	1015	0.327	1012
MZ_m	A	0.403	437	0.412	436	0.379	435	0.365	436
DZ_f	A	0.377	636	0.394	633	0.329	633	0.348	635
DZ_m	A	0.463	263	0.550	263	0.405	261	0.422	261
DZ_{mf}	A	0.368	712	0.415	706	0.319	705	0.314	705

dren is a function of parental influences. Table V gives the measures of resemblance for all possible parent–offspring combinations for twins living apart. Comparable statistics for twins living together are given at the bottom in Table V. In computing indices of parent–offspring similarity we can measure the parental phenotype in one of two ways. We can measure the similarity between individuals and their *own* reports of their parents, in which case the parent offspring resemblance is inflated directly by any tendency of individuals to rate their parents like themselves. Alternatively, we can use parental ratings obtained from raters (e.g., cotwins) who do not enter directly into the relationship for which the resemblance is being measured. We present *U* statistics computed for first and second twins of the pairs (T_1 and T_2) related to the reports given by the first and second twin about the religious affiliations of their mothers and fathers. In half the *U*'s, the twin who provides the self-report also describes the parent. In the remainder, twins' self reports are correlated with the reports given by their cotwins about their parents. The data show a striking consistency with those in Tables II–IV because resemblance is greater in the families of twins reared together and the *U* statistics are larger for the resemblance between twins' affiliations and their own reports on their parents. However, the important additional finding is that the resemblance of mothers and children is substantially greater than that between fathers and children for twins living together and apart. There is little evidence that the impact of mothers and fathers is affected by the sex of their offspring.

Table V. Resemblance Between Twins' Religious Affiliation and Their Reports of Religion in Their Parents'

		Twins' reports of							
		Mother				Father			
		Self		Cotwin		Self		Cotwin	
Twin type	Twin	*U*	*N*	*U*	*N*	*U*	*N*	*U*	*N*
		(A) Twins living apart							
MZ_f	T1	0.448	1013	0.450	1007	0.347	1011	0.325	1007
	T2	0.497	1013	0.446	1017	0.382	1013	0.344	1013
MZ_m	T1	0.456	432	0.458	431	0.371	432	0.337	430
	T2	0.461	437	0.429	436	0.367	436	0.335	436
DZ_f	T1	0.466	635	0.423	634	0.361	635	0.319	632
	T2	0.446	632	0.382	633	0.359	630	0.321	633
DZ_m	T1	0.544	258	0.461	256	0.437	258	0.381	256
	T2	0.621	259	0.523	257	0.482	259	0.426	257
DZ_{mf}	f	0.500	709	0.430	701	0.358	708	0.304	701
	m	0.442	703	0.395	705	0.351	703	0.267	705
		(B) Twins living together							
MZ_f	T1	0.610	204	0.629	204	0.533	203	0.515	203
	T2	0.613	206	0.610	205	0.499	205	0.498	204
MZ_m	T1	0.683	126	0.640	126	0.596	127	0.536	126
	T2	0.700	125	0.615	125	0.553	125	0.532	126
DZ_f	T1	0.697	109	0.693	109	0.638	109	0.593	109
	T2	0.726	110	0.673	109	0.616	110	0.579	109
DZ_m	T1	0.700	81	0.546	80	0.537	81	0.499	80
	T2	0.716	82	0.725	82	0.583	82	0.561	82
DZ_{mf}	f	0.667	189	0.616	189	0.532	189	0.535	188
	m	0.652	188	0.609	188	0.557	187	0.483	188

A MODEL FOR FAMILY RESEMBLANCE

Preliminary consideration of the raw statistics suggests that the transmission of religious affiliation is a complex of several different forms of social interaction, including differential maternal and paternal effects, mate selection, and possibly even some genetic effects. Furthermore, some or all of these effects depend on the "network" in which twins are currently living represented by the difference between twins living together and apart. These effects, and others, may be specified in the basic model for nuclear families comprising mother and father, with phenotypes M and F, respectively, and two adult (separated) children of unlike sex. We denote the phenotype of the male children by C_1 and that of the female by C_2. Figure 1 specifies a path model for variation in the phenotypes of the offspring.

The model makes a number of assumptions.

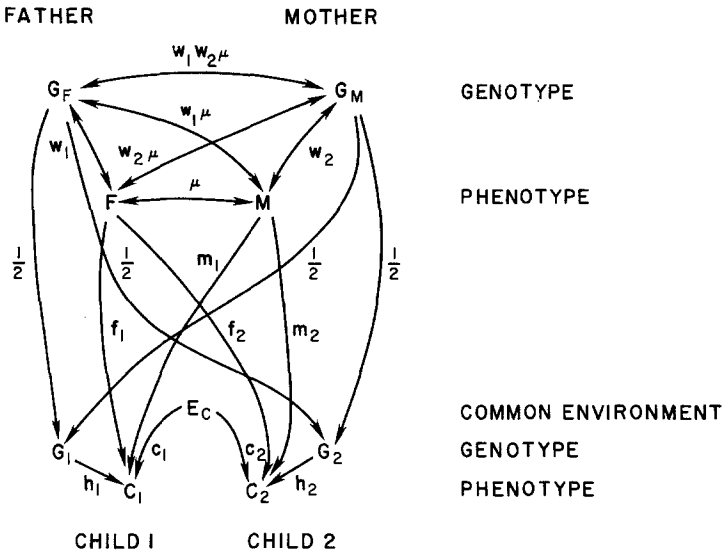

Fig. 1 Path model for family resemblance.

(1) Phenotypic variation in offspring may be due to four main sources.

(a) Genetic effects, represented in the diagram by the genotypes of the offspring G_1 and G_2, with paths h_1 and h_2 to C_1 and C_2, respectively.

(b) The direct environmental impact of the maternal phenotype M, and the paternal phenotype F, on C_1 and C_2, denoted by coefficient m_1, m_2 (maternal) and f_1, f_2 (paternal).

(c) The influence of shared environmental effects E_c, arising from sources independent of parental phenotypes, such as the effects of schooling and peers which affect C_1 and C_2 through the paths c_1 and c_2, respectively.

(d) Residual uncorrelated environmental effects (not shown in the diagram) which contribute a proportion e_1^2 and e_2^2 to the total variation of males (C_1) and females (C_2), respectively.

(2) Genetic effects are assumed (a) to be additive (i.e., there is no dominance between alleles at a locus or epistasis between alleles at different loci) and (b) to be caused by the same genes in both sexes [i.e., h_1 and h_2 may differ but G_1 and G_2 always reflect the same set of loci (cf. Eaves *et al.*, 1978)].

(3) There is no genotype × environment interaction [i.e., genes do not control sensitivity to environmental effects and particular environments do not "switch off" particular sets of genes (cf. Eaves, 1982)].

(4) Assortative mating is based on the phenotypes of parents for the trait. Writing μ for the phenotypic marital correlation and ω_1 for the correlation

between father's genotype (G_F) and F, and ω_2 for the corresponding maternal correlation, it follows from this assumption that the correlation between G_M and G_F is $\omega_1\omega_2\mu$, that between G_F and M is $\omega_1\mu$, and between G_M and F the correlation is $\omega_2\mu$.

(5) Other social interactions between family members, apart from those specified explicitly in the model, have no effect on the trait in question. Such effects include mutual influence of spouses after marriage (see Long *et al.*, 1985; Heath, 1987) and sibling interaction (e.g., Eaves, 1976; Carey, 1986).

(6) "Developmental equilibrium" has been achieved in which genetic and environmental effects have reached final adult values which no longer change with age (see Eaves *et al.*, 1986).

The path coefficients m_1, m_2, f_1, f_2, c_1, c_2, h_1, and h_2 and the correlation μ are all unknown and must be estimated from the data. The residual environmental effects e_1 and e_2 may be obtained by difference (see below). When there is both biological and cultural inheritance, ω_1 and ω_2 change between generations and may approach equilibrium values under certain conditions (see below). The correlations ω_1 and ω_2 can be expressed as implicit functions of other model parameters, if we are prepared to make the additional assumption that the population is at equilibrium (or sufficiently close to it in practice) under the effects of assortative mating and cultural inheritance. The paths from parental genotypes to offspring genotypes are all fixed at 1/2 as predicted by genetic theory under Mendelian autosomal inheritance.

MEASUREMENT MODEL

A unique feature of our study is the fact that parental data were obtained by questioning their children. Such reports are not expected to agree precisely with how parents might describe themselves because of unreliability of reporting and attributional effects of the childrens' own phenotypes. Therefore, we must superimpose on our causal model for family resemblance a measurement model which allows for these effects (cf. Heath *et al.*, 1985a). One model is given in Fig. 2. The "true" phenotypic correlations between family members, ρ, μ, ϕ_1, ϕ_2, ψ_1, ψ_2, may be derived from Fig. 1 (see below). We represent the parental phenotypes as reported by C_1 by R_{F1} and R_{M1}. The corresponding reports by daughters are R_{F2} and R_{M2}. The paths from "true" phenotypes to reported phenotypes are r_1 and r_2 for fathers and mothers, respectively. The attributional biases caused by the children's own phenotypes for the trait are p_{11}, p_{12}, p_{21}, and p_{22}, the first subscript referring to the sex of the child (male = 1) and the second referring to the sex of the parent.

The two figures summarize all that is needed to derive expected correlations for the self-reports of children and the various correlations involving children's reports of their parents.

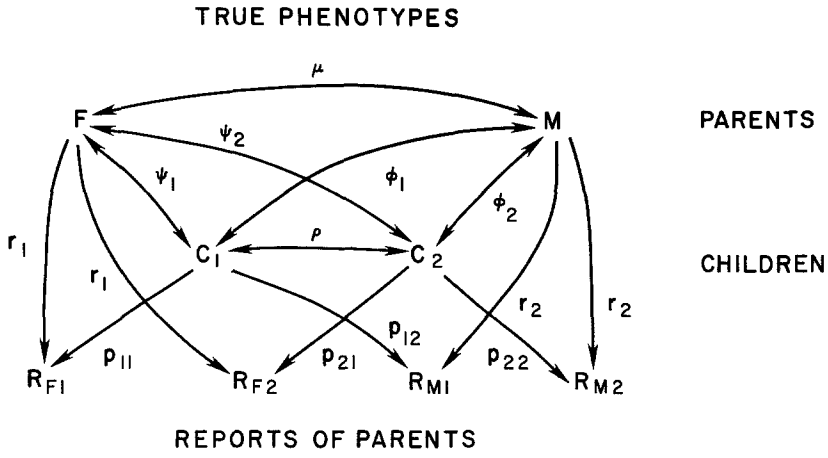

Fig. 2 Measurement model for reports of parental religious affiliation.

If we write

$$\gamma_1 = 1/2\ (\omega_1 + \omega_2\mu)$$
$$\gamma_2 = 1/2\ (\omega_2 + \omega_1\mu)$$

and

$$\alpha = 1/2(1 + \omega_1\omega_2\mu)$$

then the "true" parent offspring correlations are

$\psi_1 = f_1 + h_1\gamma_1 + m_1\mu$ (father–son),
$\psi_2 = f_2 + h_2\gamma_1 + m_2\mu$ (father–daughter),
$\phi_1 = m_1 + h_1\gamma_2 + f_1\mu$ (mother–son),
$\phi_2 = m_2 + h_2\gamma_2 + f_2\mu$ (mother–daughter).

The correlation between unlike-sex siblings (or DZ twins) is expected to be

$$\rho = \alpha h_1h_2 + f_1f_2 + m_1m_2 + c_1c_2 + \mu(f_1m_2 + f_2m_1) + h_1(\gamma_1f_2 + \gamma_2m_2) + h_2(\gamma_1f_1 + \gamma_2m_1).$$

If the total variance is standardized to unity we have the constraints (1)

$$h_1^2 + f_1^2 + m_1^2 + c_1^2 + 2(h_1f_1\gamma_1 + h_1m_1\gamma_2 + m_1f_1\mu) + e_1^2 = 1$$

and

$$h_2^2 + f_2^2 + m_2^2 + c_2^2 + 2(h_2f_2\gamma_1 + h_2m_2\gamma_2 + m_2f_2\mu) + e_2^2 = 1.$$

Furthermore, if the population is at equilibrium under assortative mating and cultural inheritance, we have (2)

$$\omega_1 = h_1 + \gamma_1 f_1 + \gamma_2 m_1$$

and

$$\omega_2 = h_2 + \gamma_1 f_2 + \gamma_2 m_2.$$

All the above apply to the true trait values, derived by self-report. When we impose the measurement model from Fig. 2 we obtain the modified expectations in Table VI. As we expect when the attributional effects (p_{ij}) are zero, the correlations tabulated involve only the true correlations and the reliability coefficients r_1 and r_2. However, when there are attributional effects ($p_{ij} = 0$) complex biases will occur which are a function of the p's and whether the parents are reported by the same child or different children (in the case of the husband–wife correlation) or whether the child entering into the parent–offspring correlation also supplies the report of parental affiliation. It is important to notice that, since the sibling correlation, ρ, enters into many of these correlations, the bias will depend on zygosity in the case of twins if there are genetic effects on the phenotype.

The correlations have been derived for nuclear families with children of unlike sex. The true correlations for male–male sibships (DZ twinships) may be obtained by substituting h_1 for h_2, f_1 for f_2, etc., in the expectation for ρ, and the correlations modified under the measurement model are obtained from those in the table by substituting p_{11} for p_{21}, p_{12} for p_{22}, ϕ_1 for ϕ_2, etc., as needed. The converse substitutions, h_2 for h_1, etc., are made in deriving the expected correlations for female-female pairs.

The correlations for the special, and analytically important, case of MZ twins

Table VI. Expected Correlations Between Relatives When Children Report on Parents[a]

Correlation	Father	Mother	Expectation
	Reported on by		
F–M	C_1	C_1	$p_{11}p_{12}+r_1r_2\mu+p_{11}\phi_1r_2+p_{12}\psi_1r_1$
	C_1	C_2	$p_{11}p_{22}\rho+r_1r_2\mu+p_{11}\phi_1r_2+p_{22}\psi_2r_1$
	C_2	C_1	$p_{12}p_{21}\rho+r_1r_2\mu+p_{21}\phi_2r_2+p_{12}\psi_1r_1$
	C_2	C_2	$p_{21}p_{22}+r_1r_2\mu+p_{21}\phi_2r_2+p_{22}\psi_2r_1$
C_1–F	C_1	—	$p_{11}\rho + \psi_1r_1$
	C_2	—	$p_{21}\rho + \psi_1r_1$
C_2–F	C_1	—	$p_{11}\rho + \psi_2r_1$
	C_2	—	$p_{21} + \psi_2r_1$
C_1–M	—	C_1	$p_{12} + \phi_1r_2$
	—	C_2	$p_{22}\rho + \phi_1r_2$
C_2–M	—	C_1	$p_{12}\rho + \phi_2r_2$
	—	C_2	$p_{22} + \phi_2r_2$

[a] Symbols defined in figures and text.

are identical in form except that the consequences of genetic identity are specified by setting $\alpha = 1$ in the expression for ρ.

The above treatment applies to parents and adult twins who are living apart and who are assumed to have achieved developmental equilibrium. The expectations must be modified for twins who are living together and are still under more direct parental influence.

Such effects may be incorporated into the model by retaining the equilibrium expectations for parents, i.e., keeping μ, ω_1, and ω_2 the same but allowing the effects on offspring to be different. For twins living together, therefore, we specify new paternal and maternal effects, f_1, f_2, m'_1, and m'_2, and new genetic and environmental effects, h'_1, h'_2, c'_1, c'_2, e'_1, and e'_2. In principle, the reliabilities and attributional effects may also differ.

Clearly, the variances of twins living together and apart cannot be expected to be the same if the paths differ. For the twins living together, therefore, we do not constrain the total variance to be unity and allow f_1, etc., to be unstandardized path coefficients. Expected correlations are derived by standardizing the corresponding expected covariances.

The full model, in which e'_1 and e'_2 appear as unconstrained is not identified with the present data so additional constraints are needed. Among many possible choices, it seems reasonable that the total variance due to nonfamilial effects should change least under the two conditions of living together and apart. We thus set (3) $c_1^2 + e_1^2 = c'^2_1 + e'^2_1$ and $c_2^2 + e_2^2 = c'^2_2 + e'^2_2$ but do not require $c_1 = c'_1$ or $c_2 = c'_2$. This amounts to stipulating that the effects of living apart are merely to allow change in the residual correlation of environmental effects which are independent of the parents.

Model-Fitting Method

Although the data exemplify many of the principles specified in the model, the provision of quantitative parameter estimates and statistical tests of hypotheses presents considerable difficulty for a number of reasons.

(1) The measures of family resemblance are not correlations but are, at best, only "correlation like," so they are not distributed as correlation coefficients;
(2) the U statistics are not independent because the same individual enters into many statistics derived from the same set of families; and
(3) there are missing data.

Standard likelihood methods require specification of distributional assumptions, and the analysis of incomplete multivariate data by exact methods is prohibitive in cases such as ours. We are forced to use approximate methods. We first took the square roots of the U values, to convert them into "correlation-

like" statistics, then the statistics were transformed to hyberbolic tangents, i.e., we used $U^* = \tanh^{-1} \sqrt{(U)}$ for model-fitting purposes. Let U_i^* denote the ith transformed uncertainty coefficient based on N_i observations. Let E_i^* denote the transformed expected value of the ith correlation. We then minimized the loss function:

$$s_i^2 = \Sigma N_i \, (U_i^* - E_i^*)^2$$

with respect to variations in the parameters of the model. This approach has also been adopted by Rao *et al.*, (e.g., 1979) in the analysis of conventional correlation coefficients where a distributional model can also be specified in some cases.

The constraints (1), (2), and (3) were implemented by augmenting s^2 by a Langrangian function and minimizing the augmented function with respect to both the parameters and Lagrange multipliers. A convenient algorithm for such constrained problems is provided by the Numerical Algorithms Group (1984) FORTRAN subroutine E04UAF.

The "full" model, M_O, allows all the parameters to take their own values for twins living together and apart, subject only to the equilibrium constraints, the constraint on the total variance of twins living apart, and the fact that parents of both kinds of twin are assumed to be sampled from the same equilibrium population. The full model thus has 29 free parameters (see Table VII). Since

Table VII. Parameter Estimates Under Two Models

Parameter	Full model		Restricted model	
	Apart	Together	Apart	Together
m_1	0.555	1.004	0.527	0.913
m_2	0.382	0.622	0.381	0.564
f_1	0.195	0.317	0.207	0.490
f_2	0.112	0.200	0.109	0.355
μ	0.669[a]	0.669[a]	0.677[a]	0.677[a]
p_{11}	0.161	0.256		
p_{12}	0.121	0.188	0.155	0.179
p_{21}	0.160	0.271		
p_{22}	0.130	0.197		
r_1	0.802	0.758	0.808[a]	0.808[a]
r_2	0.831	0.787		
c_1	0.443	0.497	0.466	0.494
c_2	0.317	0.504	0.331	0.496
h_1	0.018	−0.318	0.022	−0.339
h_2	0.470	0.047	0.468	0.056
s^2	172.46		189.75	
df	121		130	

[a] Constrained to be equal across groups.

there are 150 raw U statistics, the residual s^2 has $d = 121$ df. We have no idea of the sampling variance of the U statistics so we cannot compare s^2 with its theoretical value to provide an overall test of goodness of fit.

However, if we adopt the full model as a baseline we may examine the change in s^2 which results from imposing a number of crucial equality constraints on the parameter values. For example, the increase in s^2 obtained when the h's are not equal to zero provides a test of the importance of genetic factors. If we have s'^2 for the minimum value of the loss function obtained under a reduced model, M_1, involving m constraints on parameters of M_0, then the mean square $= \frac{(s'^2 - s^2)}{m}$ may be divided by the residual mean square (s^2/d) and the resulting $F_{m,d}$, used as a guide to the support for the imposed constraints. F values close to unity imply the data support the particular constraints, while F values significantly larger than unity suggest that the m constraints cannot be justified.

RESULTS

Parameter estimates under the full model are given in Table VII. Table VIII summarizes the results of testing several hypotheses about which effects might be regarded as zero and which effects are homogeneous over network groups or sexes. Model 1 refers to the full model in which the only constraint is the requirement that the correlation between spouses, μ, should be the same in the parents of twins living together and apart. A "—" in the table under a given parameter name indicates that separate parameters were estimated for each group of twins (together and apart) and for each combination of sexes. Zero constraints are indicated by "0" in the table. If parameters were set to be equal between groups but allowed to vary between sexes the table contains "G." An "S" indicates that the parameters were allowed to differ between groups but were forced to be the same between males and females. When a single parameter value was fitted to all sex/group combinations, the table has "GS" under the corresponding parameters. The significance levels are given on the assumption that residuals are normal and uncorrelated so they can be used only as rough guides to statistical significance.

Models 2–8 show the overall contribution of each kind of effect to family resemblance. Clearly, none of them can be ignored. It is comforting that the model fitting shows that the reliabilities (r) are high but that the attributional effects cannot be ignored ($p > 0$). That is, a child's own religion affiliation biases his report of his/her parent's religion.

All sources of nongenetic inheritance make a very marked contribution to the differences in religious affiliation. Maternal and paternal influences are substantial ($m > 0$ and $f > 0$), but the resemblance of offspring is augmented by shared

Table VIII. Goodness-of-Fit Tests and Tests of Significance Under Principal Constraints on Parameter Values

Model	Constraints[a]							s^2	df	σ^2	n	m	$F_{n,m}$[b]
	m	f	μ	p	r	h	c						
1	—	—	G	—	—	—	—	172.46	121	1.43	—	—	—
2	0	—	G	—	—	—	—	242.38	125	—	4	121	12.26***
3	—	0	G	—	—	—	—	202.88	125	—	4	121	5.34**
4	—	—	0	—	—	—	—	873.19	122	—	1	121	491.64***
5	—	—	G	0	—	—	—	228.18	129	—	8	121	4.89**
6	—	—	G	—	0	—	—	4659.67	125	—	4	121	787.07***
7	—	—	G	—	—	0	—	196.73	125	—	4	121	4.24**
8	—	—	G	—	—	—	0	225.25	125	—	4	121	9.23**
9	—	—	G	S	GS	—	—	189.75	130	1.34	9	121	1.34
10	G	—	G	S	GS	—	—	206.95	132	—	2	130	5.89**
11	—	G	G	S	GS	—	—	226.92	132	—	2	130	6.36**
12	—	—	G	S	GS	G	—	195.77	132	—	2	130	2.06
13	—	—	G	S	GS	—	G	205.98	132	—	2	130	5.56**
14	S	—	G	S	GS	—	—	204.39	132	—	2	130	5.04**
15	—	S	G	S	GS	—	—	193.74	132	—	2	130	1.37
16	—	—	G	S	GS	S	—	201.59	132	—	2	130	4.08*
17	—	—	G	S	GS	—	S	196.80	132	—	2	130	2.43
18	GS	—	G	S	GS	—	—	219.16	133	—	3	130	6.72**
19	—	GS	G	S	GS	—	—	227.04	133	—	3	130	8.52**
20	—	—	G	GS	GS	—	—	200.32	131	—	1	130	7.24**
21	—	—	G	S	GS	GS	—	204.28	133	—	3	130	3.32*
22	—	—	G	S	GS	—	GS	470.06	133	—	3	130	64.01***

[a] —, parameters free; G, parameters same in groups, different in sexes; S, parameters same in sexes, different in group; GS, parameters same over sexes and group; 0, parameters zero in all groups/sexes.

[b] Levels of significance of increase in residual sum of squares when constrained model is compared with "full" model: (*) 5%; (**) = 1%; (***) = 0.1%.

environmental effects which do not depend directly on the religious affiliations of parents. The influence of peers, schooling, and even mutual reinforcement of children could explain the substantial residual effect of the shared environment (c).

Model 9 achieves some parsimony by setting the reliabilities, r, constant for the entire data set (GS) and allowing the attributional effects, p, to be constant within a group. Since the removal of these nine constraints has little effect on the residual sum of squares ($F_{9,121} = 1.35$), the reduced model is used as the baseline for subsequent comparisons. The parameter estimates under this model are given in Table VII.

The results for Models 10–13 are crucial for evaluating the effects of development and separation on family resemblance because each of the principal transmission parameters, m, f, h, and c are, in turn, assumed to have the same values in the families of twins living together and apart. The raw data (Tables

II–V) and the parameter estimates in Table VII confirm that much of the environmental impact of parents evaporates when their children leave home. Similarly, the model-fitting results indicate a much larger "peer" effect (c) on twins who are still living together. There is no evidence that the expression of genetic effects differs, on average, between twins living together and apart.

Since we have shown (Model 9) that the attributional effects are uniform *within* groups, the best test of group differences is provided by comparing Model 20 in which the p's are given a constant value for all combinations of sex and shared experience. Clearly, the groups differ in their attributional parameters, with twins living together showing somewhat greater biases than twins living apart.

Models 14–17 test the effects of trying to constrain parameters to be constant between sexes within groups. A large increase in residuals indicates that there are sex differences interacting with group differences. There are obvious sex differences in the average maternal effect, and small effects of sex on the estimate of the genetic parameter, h, which is seen to be due almost entirely to the fact that female DZ twins living apart are less correlated than their MZ counterparts. Further simplification of the model by setting the maternal and paternal effects equal to one another within groups was not justified by the data.

DISCUSSION

Structural models for family resemblance have played a significant part in the recent development of behavioral, social, life, and clinical sciences. Correlations between relatives may reflect quite complex genetic and social effects which need to be resolved in any attempt at causal analysis. Effects may be either genetic or social and still change developmentally or in response to changes in social structure. The addition of twins to the study of ordinary nuclear families provides an important opportunity to resolve issues of biological and cultural inheritance which must otherwise remain a matter of conjecture.

Our analysis of religious affiliation illustrates several important themes. First, it shows that twin methods do not inherently generate "genetic-looking" answers. When we choose a variable for which the a priori likelihood of cultural transmission is great, we do indeed obtain results where the principal features of transmission are environmental.

Within a model for biological and cultural inheritance there may be enormous flexibility in the specification of environmental effects. Our model allows for the social interaction between spouses in the choice of mate, the social interaction involved in the attribution of religious beliefs by one relative to another, the cultural impact of mothers and fathers on their children, and the influence of shared environmental effects which do not depend on parents.

In addition to these effects, we notice that there are important develop-

mental/social effects which depend on whether children are living together or apart. Our data on religious affiliation show that the correlation between the environments of twins is substantially larger for twins living together. This finding applies equally to MZ and DZ twins and is caused in part by the greater apparent impact of parents (both mothers and fathers) on their children but also by other shared environmental effects such as peer effects and even the mutual reinforcement of twins living together. Changing the social network changes the magnitude of the familial effects and also their apparent basis. Removing twins from their parents' influence, as happens when they live apart, and from the day-to-day influence of one another appears to trigger several major changes. First, the environmental impact of parents declines significantly. For "religion" at least, leaving home results in a developmentally significant change of network such that individuals express their own individuality or are exposed to other extrafamilial influences. This is reflected in the sharp decline in all the environmental parameters, *m, f,* and *c*, in twins living apart. The smaller correlation in residual environmental effects in twins living apart suggests that twins no longer share the same network of peers etc. These changes are accompanied by a detectable change in attributional bias since children living apart from their parents are somewhat less likely to allow their own religious affiliation to influence their reports of their parents. The *validity* of the reports, as indexed by *r*, does not alter with a change in network, however.

A final developmental change occurring in twins who live apart is that hitherto latent genetic effects become expressed, at least in the females in our study. Typically, critics of the twin method have ascribed the greater resemblance of MZ twins to greater similarity in the way they are treated by parents and peers, etc. It is difficult to reconcile this view with our finding that the greater DZ differences are confined to twins living apart! It is, however, consistent with the idea that removal of many of the social constraints on behavior permits genetic differences to be expressed more readily. Our result does not mean that there is a "gene" (or genes) for being Catholic, Protestant, or Jewish in Australia so much as that there may be innate tendencies affecting adherence to family traditions which are first expressed when individuals leave home. Genetically different individuals (DZ twins) are more likely to differ in their adherence to family traditions and change affiliation when they leave home. Heath *et al.* (1985a) found a similar decline in cultural impact and a correlated increase in genetic effects on the educational attainments of Norwegian twins born after the Second World War.

Beyond the practical illustration of the feasibility of devising and testing more subtle models for biological and cultural inheritance, such results force us to ask what we understand by "equality of opportunity," what indeed *are* the predicted consequences of social change and what *is* an appropriate model for the interaction between an individual and his environment? A genetic perspective

needs to affirm a number of propositions and to deny a number of others. First, the distinction between biological and social components of intergenerational stability and change need not be left purely to the a priori assumptions of either geneticists or social scientists. Models can be devised and data collected to test many hypotheses which incorporate both kinds of factor simultaneously. Indeed, the same designs which are most informative about biological inheritance happen, as our analysis of religious affiliation shows, to be very informative about the subtleties of nongenetic transfer of information between relatives. Second, developmental and social change do not preclude genetic control. There is no greater reason a priori why the expression of genetic differences should be any more or less constant over age and epoch than the social consequences of parent–offspring interaction. Our analysis of religious affiliation gives some illustration of the possibility that changes in network may be associated with the "switching on" of genetic effects. The analysis of Norwegian educational attainments (Heath *et al.*, 1985a) suggests that major social change in the direction of equal opportunity may actually reduce the effects of cultural inheritance and facilitate the expression of genetic differences. Studies of social attitudes (Eaves and Eysenck, 1974; Eaves *et al.*, 1978; Martin *et al.*, 1986) suggest that, even though attitudes may change rapidly over time, there may still be a genetic component to the particular constellation of attitudes that individuals assimilate from their environment at a given time.

Alwin and Thornton (1984; see above) indicate a possible connection between the familial transmission of socioeconomic and educational variables and the ease with which social changes can be implemented. It is commonly supposed that matters would be worse if there were genetic effects because they are assumed to be inherently less labile. Removing socially transmissible differences *could* leave a hard core of variation which is amenable only to intervention at a molecular level. However, there is as much evidence to suggest that genetic effects could be switched on or off in a way which current theories are inadequate to predict, by changes in social policy.

Contrary to a popular impression, genetic models for individual differences do not necessarily imply pessimism about the consequences of social change. The possibility that much family resemblance for socially important variables has a genetic component, however, may lend support to a "smorgasbord" model of development in which individuals with freer access to a wider range of opportunities gradually accumulate into their phenotypes the consequences of the small but consistent biases which their genotypes impose at each moment in their development [see, e.g., Eaves *et al.*, (1986) for a mathematical treatment of this issue]. These effects could become more marked as purely social barriers to opportunity are removed. A similar model was advanced over a century ago by Galton on the flimsiest of evidence and without any explicit mathematical formulation. Only large samples of genetically informative kinship data, such

as those chosen to illustrate this paper, can resolve biological and cultural inheritance and allow us to explore the interaction between the mechanisms of inheritance and the structural and developmental facets central to current sociological theory.

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