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CHAPTER 11

Biological and Cultural Inheritance of Stature and Attitudes

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Almost a century ago, Karl Pearson (1904) concluded one of the earliest attempts to compare the inheritance of physical and behavioral traits with a generalization that appears, at best, Promethean and, at worst, an example of egregious scientific hubris. He wrote, "the mental characters are not features which differentiate man from the lower types of life. If they are inherited like man's physical characters, if they are inherited even as the protopodite of the water flea, what reason is there for demanding a special evolution for man's mental and moral side?"

Pearson shared with Galton and Darwin the view that the "mental and moral" (Pearson 1904) characters of humans could be detached neither from the evolutionary history of the human species nor from the biological ancestry of the individual. The intervening decades, however, have made it clear that Pearson's claim, even if it should turn out to be true, was premature given the state of knowledge. As Galton

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Note: The authors have worked to ensure that all information in this book concerning drug dosages, schedules, and routes of administration is accurate as of the time of publication and consistent with standards set by the U.S. Food and Drug Administration and the general medical community. As medical research and practice advance, however, therapeutic standards may change. For this reason and because human and mechanical errors sometimes occur, we recommend that readers follow the advice of a physician who is directly involved in their care or the care of a member of their family.

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himself was aware, the early studies of hereditary genius (Galton 1869) may have established a prima facie case for the biological transmission of abilities, but in truth the effects of biological and social factors were confounded. Galton's solution to the problem was to study "the history of twins" (Galton 1883) but in this field his seminal studies were largely anecdotal. Pearson and Lee's (1903) quantitative studies of familial correlations for stature and other physical measures, partly through the genius of Ronald Fisher (1918) have achieved the status of classics of the genetic literature. Even today, their data provide preliminary answers to questions about the role of nonadditive genetic effects and various types of nonrandom mating to quantitative inheritance in man.

Even with respect to human stature, however, Fisher noted that Pearson and Lee's landmark study, based as it was on remarkably large samples, left some questions unanswered. Notably, Fisher pointed out that he was still unsure about the contribution of nonadditive genetic effects to variation in stature and that still larger samples of sibling pairs would be desired. He pointed out that some of the data available (not gathered by Pearson and Lee) for more distant relationships, such as those on first cousins, were inconsistent with the model he had proposed and may point to problems of sampling in more remote relationships. He argued that Pearson and Lee's data were probably more consistent with "genotypic" rather then "somatic" assortative mating (i.e., mate selection was based not on the phenotype for stature per se but on a correlated variable that was a more perfect index of the genotype.

By contrast, Pearson's studies of the "mental and moral" characteristics of humans have been virtually ignored. They are fraught with problems of rater bias because Pearson used ratings of students by their teachers. Furthermore, these studies were confined to a small range of relationships. As a result, resemblance between the findings for physical and behavioral traits could well be coincidental.

Since these early studies, theoretical and empirical developments in social psychology, cultural anthropology, and population genetics have drawn attention to the phenomena of learning, cultural diversity, and nongenetic evolution. Behavioral correlations between nuclear family members, even though they may *look* similar to those for physique cannot now be used by themselves to support genetic theories.

The fact that, with very few exceptions, the linguistic map of human subpopulations can be superimposed on a genetic map (see for example Cavalli-Sforza 1991) confirms that, at the level of the cultural group, the effects of genes and culture are highly correlated. At the level of the human family, Cavalli-Sforza and Feldman (1973, 1981) have developed a variety of nongenetic transmission models in which the mathematical constraints of Mendelian inheritance and evolution no longer apply. These models have been applied to nuclear family resemblance in social attitudes (e.g., Cavalli-Sforza et al. 1982). Recognizing the limitation of the classical genetic theory of family resemblance led, in the 1970s and 1980s, to a flurry of quantitative models for the joint effects of biological and cultural inheritance that differed in their assumptions about the mechanisms of nongenetic inheritance and assortative mating (Cattell 1960, 1963; Cloninger et al. 1979; Eaves 1976; Heath 1983; Heath and Eaves 1985; Morton 1974; Rao et al. 1974, 1977).

Studies of the inheritance of normal behavior have focussed mainly on cognitive and personality variables. Studies of social attitudes have largely been adjuncts to the measurement of personality without any explicit theoretical justification in their own right. However, attitudes may be singled out on a priori grounds as a model system for the study of *non*genetic inheritance in humans (see for example Cavalli-Sforza and Feldman 1981; Cavalli-Sforza et al. 1982). Although no set of "scales" can capture all the nuances of differences within a culture, attitudes could not exist apart from a species that had evolved an extended matrix of social structures and interactions. It thus appears that social attitudes provide one avenue into quintessentially *human* characteristics.

Published studies show substantial parent-offspring resemblance for attitude variables (Feather 1978; Insel 1974) and some evidence of heterogeneity across sexes in parent-offspring correlation. Insel argued that his data supported a significant role of maternal inheritance. A number of large studies of twins reared together (Eaves and Eysenck 1974; Eaves et al. 1989; Loehlin and Nichols 1976; Martin and Jardine 1986; Martin et al. 1986) have generally shown that MZ twins are more similar than DZ twins and have given some support to the view that genetic factors play at least some role in the transmission of social

attitudes. However, these studies have also shown consistently that DZ twin resemblance in attitudes is too high to be explained simply by the usual assumption of additive gene action and random mating. The twin studies have thus established a prima facie case for a significant contribution of the shared family environment, explaining as much as 30% of the total variation in measured attitudes. Many of these authors have noted, however, that the contribution of the family environment is confounded with the genetic consequences of assortative mating in studies of twins reared together. Indeed, Martin et al. (1986) suggested that the spousal correlations for social attitudes were sufficiently high (see for example Eaves et al. 1989; Feather 1978; Insel 1974) to explain virtually *all* of the apparent shared environmental effect in twin studies. The Minnesota studies of separated twins (e.g., Tellegen et al. 1988; Waller et al. 1990) add some weight to this interpretation because they show that the correlations of twins separated at birth do not differ significantly from those of twins reared together. Compelling though such reports are, their relatively small sample sizes inevitably mean, however, that quite large shared environmental effects could still be present yet missed for want of statistical power. Eaves et al. (1978) report correlations in conservatism for a small volunteer sample of adult adoptees and their relatives that are consistent with the overall finding that the shared family environment has little impact. However, the small sample and the possibility of serious volunteer bias do not give us much confidence in these findings. Other adoption studies, notably the Texas Adoption Study (e.g., Loehlin et al. 1985), do not use instruments specifically to assess "social attitudes" in young adults but a wide variety of factors from broadly based personality tests all yield very small correlations for nonbiological relatives. These results are consistent with a very small contribution of the shared family environment. Scarr (1981) arrived at similar conclusions on the basis of a comparison of the correlations for authoritarianism in biological and adoptive families.

Apart from the larger twin studies, few studies have had the power to detect or analyze sex differences in the expression of genetic and environmental effects on social attitudes. This becomes critical for our understanding of the social environment since cultural expectations

may differ for men and women, and mothers may play a different role from fathers in shaping the behavior of their male and female offspring.

At present, a researcher who wishes to gain a picture of the role of biological and cultural inheritance in humans is forced to piece together a picture from a variety of different studies conducted with different restricted designs, in different target populations, and using different instruments. Few studies have the power to address the heterogeneity of biological and social effects over sexes. It is sometimes difficult to decide whether inconsistencies between studies reflect differences between samples, populations, or measurements. Sometimes it is hard to decide, given the small sample sizes available, whether similarities with the findings of smaller studies point to anything more significant than a lack of power.

Design and Rationale of the Virginia 30,000

The Virginia 30,000 study was conceived in the mid-1980s as an attempt to generate a data base sufficiently broad in measurements, rich in relationships, and large in samples, that it might be possible to obtain clearer answers to lingering questions about the role of biological and cultural transmission in human differences. A study was needed that could give estimates of both genetic and nongenetic components of family resemblance without recourse to the dwindling population of adoptees while also allowing for tests of additive and nonadditive genetic effects, sex differences in the expression of genes and environment, resolution of alternative models of assortative mating, and tests of the consistency of any "model" across a broad range of relationships.

Long experience with data analysis and simulation studies (e.g., Eaves 1972; Heath et al. 1985; Martin et al. 1978) had shown that very large samples were likely to be needed to test many hypotheses, especially those about sex differences in genetic and environmental effects. Thus, many of the designs that had obvious intuitive appeal, such as

the various adoption designs and the study of separated twins, required populations that were likely to be too small to yield answers to some of the questions we were asking. Nuclear families and extended pedigrees were easily obtained, but these groups left genetic and social factors as confounded as they were in Galton's original studies of hereditary genius and Pearson's studies of "mental and moral" traits.

Theoretical analyses and simulations in the early 1980s (Eaves 1980; Heath 1983; Heath and Eaves 1985; Heath et al. 1985) suggested that the "extended twin-kinship design" could, with sufficiently large samples, provide adequate tests of many features of the transmission of human differences in a single study.

We illustrate the value and flexibility of the Virginia 30,000 study by outlining some early results that reexamine the early claims of Galton, Pearson, and Fisher about the causes of family resemblance in measures chosen a priori to reflect distinct mechanisms of biological and cultural inheritance in humans. *Stature* is chosen to represent a physical variable, height, that is usually regarded as unquestionably genetic. As a measure most likely to reveal the impact of nongenetic inheritance, we follow the proposal of Cavalli-Sforza and his colleagues (1981, 1982) and study a measure of social attitudes. In this first report, we explore the transmission of individual differences in *conservatism*, a composite measure emerging from the joint analysis of a large number of individual attitudes.

An idealized pedigree for the Virginia 30,000 study is illustrated in Figure 11–1. Each pedigree starts with a pair of adult twins. Twins may be either identical (monozygotic, MZ) or fraternal (dizygotic, DZ). MZ pairs may be male or female. DZ pairs may be male, female, or of opposite sex. There are thus five basic kinds of pedigree depending on the zygosity and sex of the twins. The pedigree is then augmented by tracing outward from the twins to include as many living parents, spouses, siblings, and children of twins as may be ascertained. Typically, we were unable to ascertain many pedigrees in which we could contact all three generations. Most pedigrees comprised either twins with their collateral relatives and parents or twins with their collateral relatives and children.

As Francis Galton himself suggested (1883), twins reared together provide a powerful and easily accessible starting point for the resolution

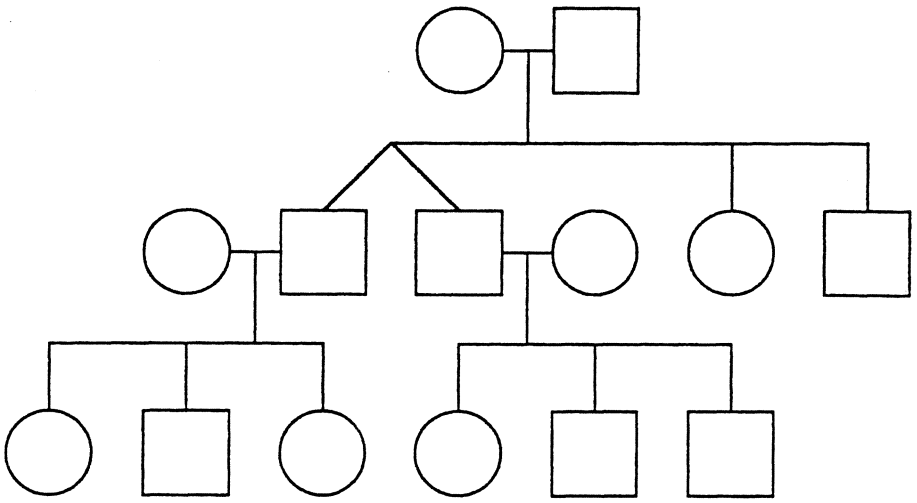

FIGURE 11–1. *Idealized family structure for the Virginia 30,000 study. Standard conventions are used for drawing pedigrees: Circles denote women and squares denote men. Horizontal bars between circles and squares denote spouses. Offspring from a mating are indicated by vertical lines connected by horizontal bars. Twins are connected by diagonal lines.*

of biological and cultural inheritance and thus comprise the core of the present study. However, twins have the weakness that the genetic consequences of assortative mating are confounded with the effects of the shared environment (e.g., Eaves 1970) so we included the parents and spouses of twins to allow us to estimate and analyze the causes and effects of assortative mating (see for example Eaves and Heath 1981; Heath and Eaves 1985) and social interaction between spouses (Heath 1987). Furthermore, since studies of twins alone are always subject to the criticism that twins are not typical of the genetic and environmental effects in the population, we included the siblings of twins in the design to allow for tests of additional environmental similarity in twins. Following Young et al. (1980), we noted that the study of twins and their parents provided a powerful extension of the nuclear family design that allowed some resolution of the additive and non-additive effects of genes from those of the shared family environment and assortative mating contributed by parents. With Nance and Corey (1976; see also Haley et al. 1981) we recognized that the children of MZ twins are socially cousins but genetically half siblings. The MZ

twin of a parent may be called "uncle" or "aunt" but, at least under random mating, shares the same genetic relationship as a biological parent. These novel relationships exploit the genetic similarity of MZ twins to generate a number of additional relationships involving individuals who share comparable biological relationships to those in the nuclear family without entering into the standard social relationships of the nuclear family.

Ascertainment of the Virginia 30,000 was conducted in two stages. Twin pairs were recruited from the Virginia Population-based Twin Registry (Corey et al. 1991) and from the American Association of Retired Persons (AARP) as a result of a letter of invitation published in the AARP newsletter. Twins were mailed the "twin" version of a 16-page "Health and Lifestyle Survey" (HLS). The survey was compiled to assess a number of aspects of physique, personality, lifestyle, environment, life events, and personal history related to health. Respondents were asked to supply names, addresses, and telephone numbers of their living parents, spouses, siblings, and children. The relatives of twins were then contacted to complete their own version of the HLS, which secured identical self-report data for the items analyzed in this report.

The final sample comprised 29,691 individuals. Table 11–1 summarizes the composition of the sample by sex and relationship to the twins. A few other relationships (e.g., half-siblings, adoptees) are also represented in the sample but their numbers are generally too small

TABLE 11–1.

Sample sizes of twins and the principal adult relatives of twins in the Virginia 30,000 study

	Males	Females	Total
Twins	5,325	9,436	14,761
Parents of twins	913	1,447	2,360
Spouses of twins	2,515	1,876	4,391
Children of twins	1,890	2,910	4,800
Siblings of twins	1,260	1,924	3,184
Other	67	128	195
Total	11,970	17,721	29,691

to have a significant impact on the analysis, and these individuals are not included in these numbers.

If, on account of relatively small numbers, we ignore relationships across three generations, but take into account the sex of the relatives and whether avuncular relationships are defined through paternal or maternal siblings, there are 80 unique biological and social relationships in the Virginia 30,000 (see Table 11–3). This contrasts with only 5 relationships in the classical twin study, 8 in the conventional nuclear family study, and 10 in the "twin-parent" design. Thus, the Virginia 30,000 design provides a rich variety of biological and social relationships that may be used to test the generalizability of explanations of family resemblance across a wide range of circumstances.

Measures Chosen for Analysis

Stature was obtained by self-report. Subjects were asked "How tall are you?" and gave their height in feet and inches. A small handful of outliers whose reported heights were less than 4′ or greater than 7′ were excluded from the analysis.

The HLS yields a variety of behavioral measures. This chapter considers a single composite measure of *conservatism* derived from a 28-item social attitudes inventory modeled after the Wilson-Patterson Conservatism Scale (Wilson 1973; Wilson and Patterson 1970) that had been used in several earlier twin studies (see for example Eaves et al. 1989). In these earlier studies, measures of social attitudes were found to be surprisingly reliable and showed patterns of twin and family resemblance that were quite distinct from those widely replicated in studies of the major dimensions of personality (see Eaves et al. 1989 for examples of typical findings for personality measures).

The instrument devised for our study (Table 11–2) comprised selected reliable items from the original inventory supplemented by several experimental items chosen to reflect issues that were controversial in the United States in the late 1980s, when the study was conducted. Items consisted of a single word or phrase (see Table 11–2) to which

TABLE 11–2.

Key to scoring social attitude items for conservatism

Item		Key
(1)	Death penalty	+
(2)	Astrology	−
(3)	X-rated movies	−
(4)	Modern art	−
(5)	Women's liberation	−
(6)	Foreign aid	−
(7)	Federal housing	−
(8)	Democrats	−
(9)	Military drill	+
(10)	The draft	+
(11)	Abortion	−
(12)	Property tax	−
(13)	Gay rights	−
(14)	Liberals	−
(15)	Immigration	−
(16)	Capitalism	+
(17)	Segregation	+
(18)	Moral Majority	+
(19)	Pacificism	−
(20)	Censorship	+
(21)	Nuclear power	+
(22)	Living together	−
(23)	Republicans	+
(24)	Divorce	−
(25)	School prayer	+
(26)	Unions	−
(27)	Socialism	−
(28)	Busing	+

Note. *Subjects are asked to indicate agreement or disagreement by circling "Yes" or "No" as appropriate, or to indicate uncertainty by circling "?." A "+" indicates "Yes" is the keyed direction of response.*

subjects responded in one of three categories ("Yes," "?," "No") to rate the agreement or disagreement with the topic.

Preliminary factor analyses showed that five correlated factors, grouping the items roughly according to major content areas, were probably sufficient to account for the pattern of interitem correlations.

However, the dominant eigenvalue and its associated vector justified a first approximation in terms of a single "radicalism-conservatism" dimension. A scale was constructed (see Table 11–2) that gave equal positive or negative weight to the items based on the sign of salient loadings on the first general factor. Items were scored 0, 1, or 2 giving a possible raw score range of 0 to 56. Scores were imputed for subjects having more than 75% valid responses by taking the average score obtained using the valid responses multiplied by the total number of items in the full scale (28). Subjects with 75% or fewer valid responses were omitted.

Regression analyses were conducted using the SAS regression procedure for both stature and conservatism to examine mean score differences associated with sex, source of data (Virginia Twin Registry vs. AARP), twin vs. nontwin, age and age^2. The corresponding interaction terms were also included in the analysis. The normalized residuals from the full regression model were computed for both stature and conservatism and used to compute product-moment correlations between relatives. Correlations were based on every possible pair belonging to a particular relationship. The same individual may contribute more than once to a given correlation. As a result, the sampling errors of the observed correlations may be larger than expected on the basis of the raw sample sizes. Furthermore, the same individual may contribute to several correlations resulting in correlations among some of the estimated correlation coefficients. Simulation studies have shown (e.g., McGue et al. 1987) that treating such correlations as independent in analyses of family resemblance tends not to bias parameter estimates but may lead to the attribution of too much precision to the analytical results. Current computational methods make a full maximum-likelihood solution impracticable with a data set of this size and number of relationships.

Correlations Between Relatives

Table 11–3 gives the correlations for the residual stature and conservatism scores for all 80 relationships included in this analysis.

TABLE 11–3.

Correlations between relatives for stature and conservatism in the Virginia 30,000

	Stature		Conservatism	
Relationship	N (pairs)	r	N (pairs)	r
Nuclear family				
Spouses	4,751	0.223	4,915	0.619
Male siblings	1,493	0.432	1,551	0.341
Female siblings	3,524	0.429	3,643	0.405
Opposite-sex siblings	4,255	0.411	4,395	0.328
Father-son	2,160	0.439	2,247	0.410
Father-daughter	2,971	0.411	3,095	0.397
Mother-son	3,035	0.446	3,138	0.369
Mother-daughter	4,476	0.430	4,667	0.456
Twins				
Dizygotic male	573	0.483	579	0.379
Dizygotic female	1,164	0.502	1,142	0.432
Opposite-sex dizygotic	1,307	0.432	1,312	0.319
Monozygotic male	775	0.850	790	0.593
Monozygotic female	1,847	0.855	1,839	0.637
Avuncular with sibling of parent				
Paternal uncle-nephew	92	0.427	100	0.334
Paternal uncle-niece	155	0.228	156	0.324
Maternal aunt-nephew	402	0.185	405	0.200
Maternal aunt-niece	536	0.314	547	0.226
Paternal aunt-nephew	131	0.275	133	0.264
Paternal aunt-niece	196	0.231	200	0.112
Maternal uncle-nephew	236	0.253	235	0.175
Maternal uncle-niece	284	0.230	297	0.166
Avuncular with dizygotic twin of parent				
Paternal uncle-nephew	105	0.369	110	0.107
Paternal uncle-niece	137	0.077	144	0.108
Maternal aunt-nephew	345	0.260	332	0.200
Maternal aunt-niece	525	0.239	516	0.250
Paternal aunt-nephew	118	0.242	114	0.282
Paternal aunt-niece	188	0.244	180	0.314
Maternal uncle-nephew	150	0.288	154	0.185
Maternal uncle-niece	202	0.271	206	0.225

(*continued*)

TABLE 11–3.
Continued

Relationship	Stature N (pr)	Stature r	Conservatism N (pr)	Conservatism r
Avuncular with monozygotic twin of parent				
Paternal uncle-nephew	217	0.529	221	0.428
Paternal uncle-niece	337	0.444	341	0.315
Maternal aunt-nephew	673	0.458	661	0.367
Maternal aunt-niece	1,040	0.377	1,035	0.318
Cousins related through monozygotic twin parents				
Male pairs related through MZ male parents	39	0.406	40	0.564
Female pairs related through MZ male parents	92	0.271	95	0.265
Male-female pairs related through MZ male parents	107	0.351	107	0.264
Male pairs related through MZ female parents	153	0.279	158	0.238
Female pairs related through MZ female parents	340	0.215	339	0.287
Male-female pairs related through MZ female parents	449	0.274	459	0.309
Cousins related through dizygotic twin parents				
Male pairs related through DZ male parents	19	0.788	19	0.091
Female pairs related through DZ male parents	41	0.559	42	0.339
Male-female pairs related through DZ male parents	52	0.550	52	0.173
Male pairs related through DZ female parents	52	0.185	53	−0.117
Female pairs related through DZ female parents	138	0.156	141	0.275
Male-female pairs related through DZ female parents	159	0.232	163	0.240
Male pairs with male-female twin parents	38	0.113	39	0.242
Female pairs with male-female twin parents	71	0.343	70	0.227
Male-female pairs with male-female twin parents	51	0.091	52	0.066
Male-female pairs with female-male twin parents	72	0.264	70	0.112
Relationships by marriage				
Siblings-in-law				
Wife of twin with nontwin brother of twin	337	0.092	371	0.386
Husband of twin with nontwin sister of twin	728	0.116	745	0.219
Husband of twin with nontwin brother of twin	422	0.101	443	0.222
Wife of twin with nontwin sister of twin	447	0.069	486	0.175
Wife of twin with husband's DZ male co-twin	387	0.125	417	0.263
Husband of twin with wife's DZ female co-twin	603	0.166	618	0.310
Husband of twin with wife's DZ male co-twin	353	0.155	363	0.202
Wife of twin with husband's DZ female co-twin	458	0.218	455	0.290
Wife of twin with husband's MZ male co-twin	589	0.252	625	0.490
Husband of twin with wife's MZ female co-twin	1,139	0.207	1,153	0.409

(*continued*)

TABLE 11–3.
Continued

Relationship	Stature N (pr)	Stature r	Conservatism N (pr)	Conservatism r
Parents-in-law				
Father-in-law/daughter-in-law	205	0.170	208	0.312
Father-in-law/son-in-law	188	−0.016	211	0.219
Mother-in-law/daughter-in-law	293	−0.013	311	0.250
Mother-in-law/son-in-law	338	0.140	360	0.308
Affine avuncular				
Nephew with wife of father's DZ twin	54	0.483	57	−0.048
Niece with wife of father's DZ twin	80	0.356	82	0.159
Nephew with husband of mother's DZ twin	126	0.196	128	0.158
Niece with husband of mother's DZ twin	169	−0.010	179	0.166
Nephew with husband of father's DZ twin	36	−0.029	38	0.015
Niece with husband of father's DZ twin	68	−0.003	70	0.055
Nephew with wife of mother's DZ twin	64	−0.210	71	0.230
Niece with wife of mother's DZ twin	95	−0.033	99	0.195
Nephew with wife of father's MZ twin	129	0.154	134	0.366
Niece with wife of father's MZ twin	213	0.194	224	0.276
Nephew with husband of mother's MZ twin	342	0.147	353	0.239
Niece with husband of mother's MZ twin	502	0.194	511	0.222
Twin's spouses				
Wives of male DZ twins	100	0.066	104	0.212
Husbands of female DZ twins	120	0.130	129	0.325
Spouses of male-female DZ twins	167	0.008	172	0.185
Wives of male MZ twins	172	0.199	188	0.378
Husbands of female MZ twins	300	0.231	304	0.267

Before embarking on any more rigorous analysis, it is helpful to study some of the correlations between relatives to obtain an overall perspective on family resemblance and its likely causes. Figure 11–2 summarizes the correlations for nuclear family members. We note remarkable uniformity at a little over 0.4 between the various parent-offspring and sibling correlations for stature. The correlations for conservatism also hover around 0.4 but are more heterogeneous. The average parent-offspring correlation for conservatism is a little larger than that for siblings. The correlations for conservatism are more het-

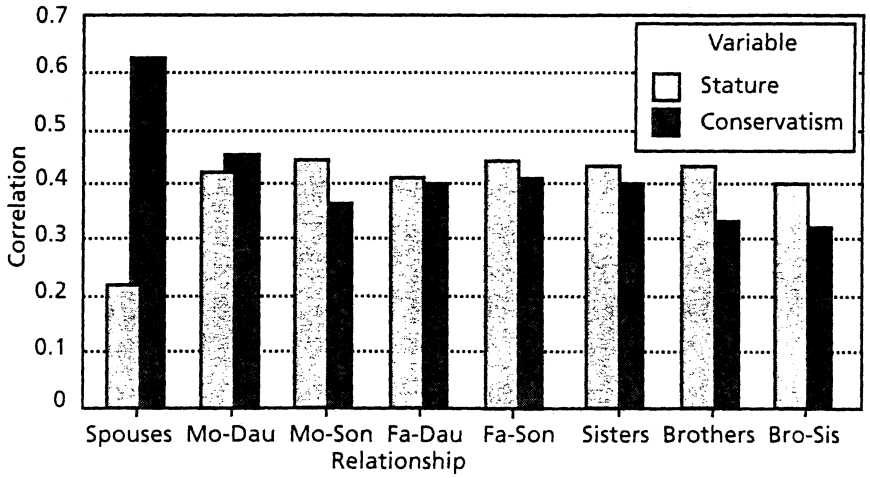

FIGURE 11–2. *Nuclear family correlations for stature and conservatism.*

erogeneous across sexes and are marginally lower for pairs of opposite sex. Thus, our self-report data for conservatism reveal marked family resemblance of the order that Pearson had claimed for his ratings of siblings. The lower correlations for opposite-sex pairs for conservatism, together with the higher correlation for female pairs compared with male pairs, indicates that some modest sex differences may be expected in either the genetic or social transmission of attitudes. However, all the nuclear family correlations are quite large and positive regardless of sex. This finding precludes two common myths about parent-offspring relationships for social variables: that children "react against" their parents (which would produce negative correlations) and that children model on their same-sex parent (because there would be far greater differences between same and opposite-sex parent-offspring correlations). Such effects are not precluded by the data as modest factors in the transmission of attitudes, but clearly the Virginia 30,000 data preclude these effects playing an overwhelming role.

The major contrast between the nuclear family correlations for stature and conservatism is that between spouses. For conservatism, the observed correlation of 0.62 is as great as that for MZ twins and almost three times as large as that for stature. The small spousal correlation for stature may be sufficient to have detectable genetic consequences for such a heritable trait. If the large correlation between mates for

conservatism is due to like marrying like ("assortative mating") rather than spousal convergence due to social interaction, it would have very marked effects on the correlations for other relationships whether these be caused genetically, environmentally, or both. In our data, the Spearman correlation between absolute intrapair differences and duration of marriage for spouse pairs was -0.0298 ($P = .04$, $N = 4{,}884$ pairs). The fact that this correlation is small and barely significant suggests that either convergence has all occurred prior to marriage, or that the large spousal correlation is due to assortative mating. In general (see Heath 1987), spousal interaction will tend to result in disproportionately high correlations between spouses compared with those for other relationships by marriage including the spouses of twins and siblings.

Correlations for nuclear families establish a baseline for the degree of family resemblance but contribute little to the resolution of biological from cultural inheritance. Figure 11–3 summarizes the correlations for MZ and DZ twins. Barring mutations, the genetic effects of MZ twins are perfectly correlated. When mating is random and genetic effects are additive (i.e., there is no dominance or epistasis) the genetic effects of DZ twins (and siblings) are expected to correlate only 0.5. Thus, in the simplest of all worlds in which mating is random, gene effects are additive, and the only environmental effects are uncorrelated between relatives, the correlation for DZ twins is expected, within

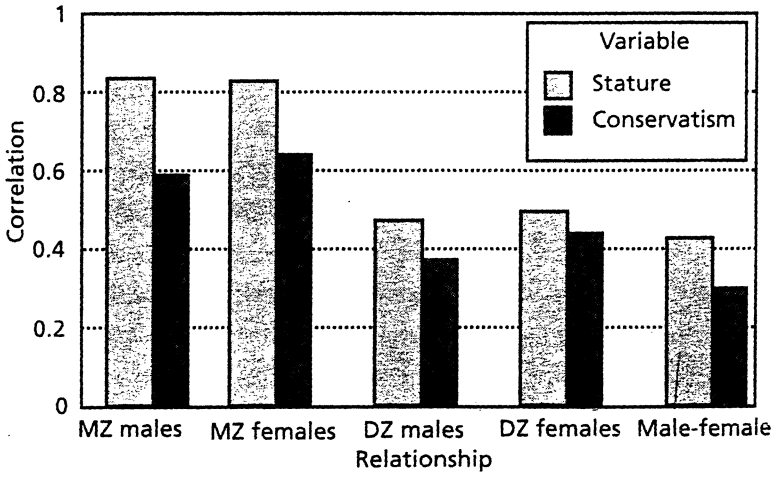

FIGURE 11–3. *Twin correlations for stature and conservatism.*

sampling error, to be one-half of that between MZ twins. DZ correlations in excess of one-half the MZ correlation may point directly to the genetic and environmental consequences of assortative mating, or to effects of the shared family environment. If DZ correlations are less than one-half those for MZ it is possible that the genetic effects are not additive (see for example Jinks and Fulker 1970). If the twin correlations are both significant but equal, there can clearly be no genetic effects but there must be substantial effects of the shared environment. A significant excess of the MZ over the DZ correlations establishes a prima facie case (though not incontrovertible evidence) for the contribution of genetic factors.

For both stature and conservatism, the Virginia 30,000 data confirm the well-documented excess of the MZ correlations over those for DZ. Thus, genetic factors appear to play a role. In the case of stature, it comes as no surprise. For those who are unaware of the twin literature on social attitudes, the findings may be more disconcerting because they appear to challenge commonly held theoretical and philosophical suppositions that humans are largely immune from the effects of their biological inheritance once we transcend the purely physical domain.

The broad trend for DZ correlations is close to that for siblings. The differences across sexes are more marked for conservatism than stature, and the ordering of the three DZ correlations for conservatism mirrors perfectly that for siblings. There is a slight excess of the DZ correlations over those for siblings with respect to stature, but barely so for conservatism.

We note that the correlations for same-sex DZ twins (and siblings) are greater than one-half the corresponding MZ correlations. This excess is present for both stature and conservatism, but more marked for conservatism. This result implies either that the genetic correlation for DZ twins is greater than 0.5 due to assortative mating or population stratification, or that there are significant effects of the shared family environment, or both. It is conceivable that the excess may have different causes in the two variables.

Figure 11–4 presents correlations for 16 of the most salient relationships pooled over sexes. The sample sizes for many of these correlations are extremely large (over 8,000 sibling pairs, and 12,000 parent-offspring pairs) but significant heterogeneity between sexes for

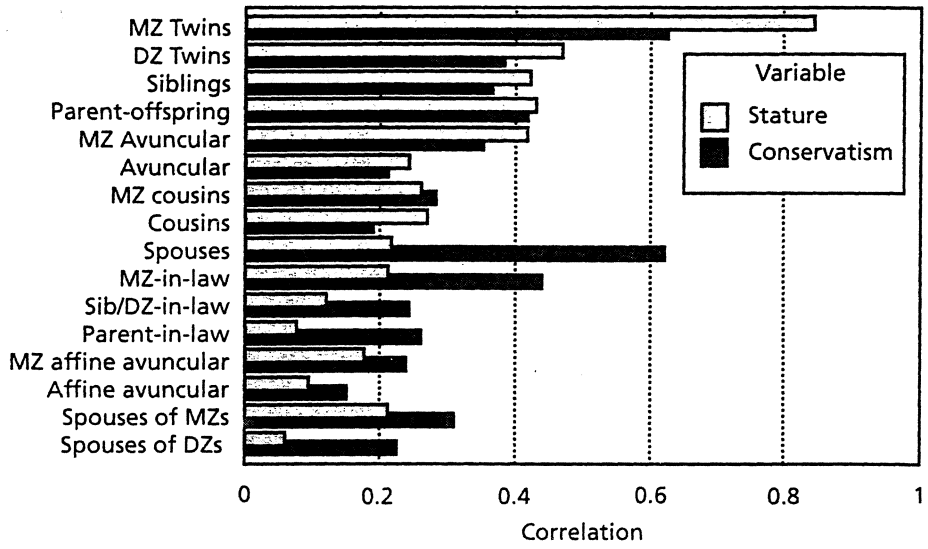

FIGURE 11–4. *Principal correlations between relatives for stature and conservatism pooled over sexes.*

some of the relationships (compare Table 11–4 and Figures 11–2 and 11–3) is ignored so the figure is primarily to assist the visualization of trends.

Considering first the biological relationships, Figure 11–4 reveals the marked similarity in the pattern of correlations for stature and conservatism. There is a striking linear relationship between the observed correlation and the degree of genetic relatedness. The high correlation between cousins related through DZ twins for stature is a marked and inexplicable exception to this pattern. The correlations for stature illustrate as clearly as possible the pattern to be expected for a variable in which there is a large additive genetic component of variance. We note especially that there is no difference between the parent-offspring correlation for stature and the avuncular correlation involving MZ co-twins of parents. Nongenetic explanations of the excess similarity of MZ twins become increasingly strained as we confirm that other biological relationships derived from MZ twins fit closely the predictions of a genetic model when compared with those for other relationships. The correlations for conservatism show a similar pattern, with the lower MZ correlation noted earlier. However, the parent-offspring correlation is slightly higher than the MZ avuncular corre-

lation suggesting that some of the parent-offspring resemblance may have a nongenetic component (e.g., phenotypic assortative mating).

The pooled correlations also demonstrate clearly the impact of assortative mating on the correlations between relatives. Both stature and conservatism show evidence of assortative mating. However, the spousal correlations for conservatism are very much larger than those for stature. The effect of assortment for conservatism pervades all the correlations by marriage for our data. On closer examination, the comparative data for stature and conservatism show some important similarities and differences. For both traits, the correlations by marriage involving DZ twins are lower than those involving MZ twins. This finding does not favor the view advanced by Morton and his colleagues (Morton 1974; Rao et al. 1974) that assortment is based primarily on the *social*, rather then genetic components, of the phenotype. If spouses select one another primarily for aspects of the environment they share, such as the socioeconomic status of their parents, then we would predict no difference between correlations for in-laws of MZ and DZ twins. Our data thus suggest mate selection also involves genetic aspects of the phenotype.

A Model for Family Resemblance in Extended Twin Kinships

The previous discussion provides a preliminary sense of the main features of family resemblance for a physical and behavioral trait. However, it does not provide quantitative estimates of the various social and biological factors contributing to individual differences. Neither does it yield any analysis of how sex affects the transmission and expression of genetic and environmental effects. This goal can only be realized by formulating, fitting, and testing a mathematical model for family resemblance in the 80 correlations. One such model, described in detail by Truett et al. (1994) is presented in Figure 11–5 as a path diagram (see Li 1976, Neale and Cardon 1992; Wright 1921). We stress at the outset that this is not the only model that could be devised, but it represents a good place to start because it allows for additive and

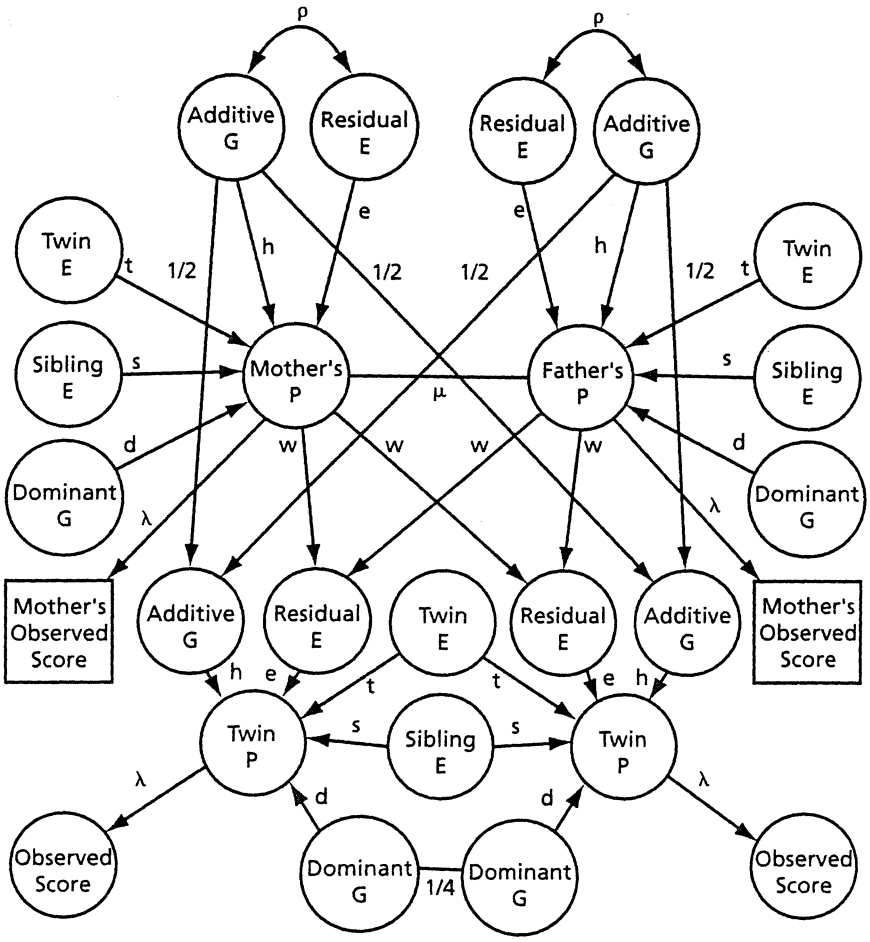

FIGURE 11–5. *Path model for biological and cultural inheritance in the absence of sex-dependent effects.*

The "latent" phenotypes of mothers, fathers, and children on which assortment and cultural inheritance are based are indicated by circles and the observed scores by squares.

Single-headed arrows represent the hypothesized direction of causation. Correlations which have an extraneous cause, such as that between genetic and environmental effects in parents, are denoted by double-headed arrows. A bar with no arrows ("co-path," Cloninger 1980) denotes the correlation between mates whose cause is extraneous to the diagram but which induces correlations between other causal factors in the model (e.g., between the genetic effects of husbands and wives).

Legend: *G, genes; E, environment; P, phenotype; h, additive genetic effects; d, genetic dominance; e, path from environment to phenotype; w, path from parental phenotype to offspring environment; μ, phenotypic correlation between mates; s, path from residual sibling shared environment to phenotype; t, path from additional twin shared environment to phenotype; ρ, correlation between genotype and environment; λ, regression of observed score on the latent phenotype.*

dominant genetic effects, nongenetic inheritance from parents to child, some forms of assortative mating, sex differences in genetic and environmental effects, and some additional nongenetic sources of family resemblance including the shared sibling and twin environments. There is no one "right" way to write the model. Other investigators may choose to devise alternative models or parametrizations.

The model assumes, initially that assortative mating, if present, is based on the measured phenotype, P ("primary phenotypic assortative mating," Heath and Eaves 1985). Similarly, it is assumed that vertical cultural transmission from parent to offspring is based on the measured phenotype of the parents rather than on some latent or correlated variable ("P to E" vertical cultural inheritance). The effects of the sibling and twin shared environments are assumed to contribute to variation among individuals regardless of relationship. However, the sibling environment is perfectly correlated in sibling and twin pairs, and the twin environment is perfectly correlated in twin pairs. Thus, the model assumes that twins and siblings will differ in correlation, but not in variance, as a result of how they are influenced by their shared environment. That is, twins and siblings are assumed to sample the same marginal distribution of environmental effects as other individuals but they differ in the environmental correlation. The genotype-environment correlation, ρ, occurs when the parental phenotype, which contributes to the offspring's environment through parent-offspring transmission, is partly genetic in origin. This results in a correlation between the offspring's environment and genes. The process of transmission and assortment is assumed to be in equilibrium, and thus, ρ is constant between generations. That is, ρ is constrained to be same in parents and offspring. Since models are fitted to correlations, the scale of measurement has unit variance; therefore, we impose the further constraint that the sum of all sources of variation is unity.

As already noted, the measured trait may not correlate perfectly with the trait for which mate selection and cultural transmission are actually occurring. Morton (1974) argued for a model of "social homogamy" in which assortment and cultural transmission are based on a correlated latent variable to which genes make no contribution. Another mechanism of assortment (proposed by Heath and Eaves 1985) presents a model for mixed homogamy in which mate selection on

both the social background of the spouses and the phenotype of the mate. We have used "phenotypic assortment plus error" (Heath 1983) in which the actual measurement is considered a more or less unreliable index of the latent score on which assortment is based. In this model, all expected correlations were multiplied by the square of the path from "true" (or latent) score to "observed" score (the reliability [λ]). When there is significant assortative mating or cultural inheritance, there is sufficient information in the extended twin-kinship design to estimate λ without repated measurements.

Allowing for Sex Differences in Model Parameters

One of the principal advantages of a study involving large samples of relatives is the opportunity to test a variety of models of sex-dependent etiology and transmission. For the simple case of randomly mating populations, a model for sex differences in gene action was specified by Eaves (1977) that allowed for the same genes to have different magnitudes of effect on males and females. This model allows for estimation of separate genetic variances for males and females and a correlation between gene effects in males and females. The genetic correlation between the sexes will be unity if the effects of all autosomal loci on one sex are constant multiples of their effects on the other sex. In this case, we speak of "scalar sex-limitation of the gene effects."

Analogous definitions may be given for the "sex-limited" effects of the shared environment. If the magnitudes of the loci or, by analogy, "environmental effects" on one sex are not constant multiples of their effects on the other sex, then we speak of nonscalar sex limitation of genetic (or environmental) effects. The present model extends the analysis of sex-dependent effects to the more difficult cases of combined assortative mating and cultural inheritance. In the path diagram (Figure 11-6) we employ the following notation for the effects of dominance, sibling enironment, and special twin environment: d_m, s_m, and t_m in males respectively, and d_f, s_f, and t_f for their counterparts in fe-

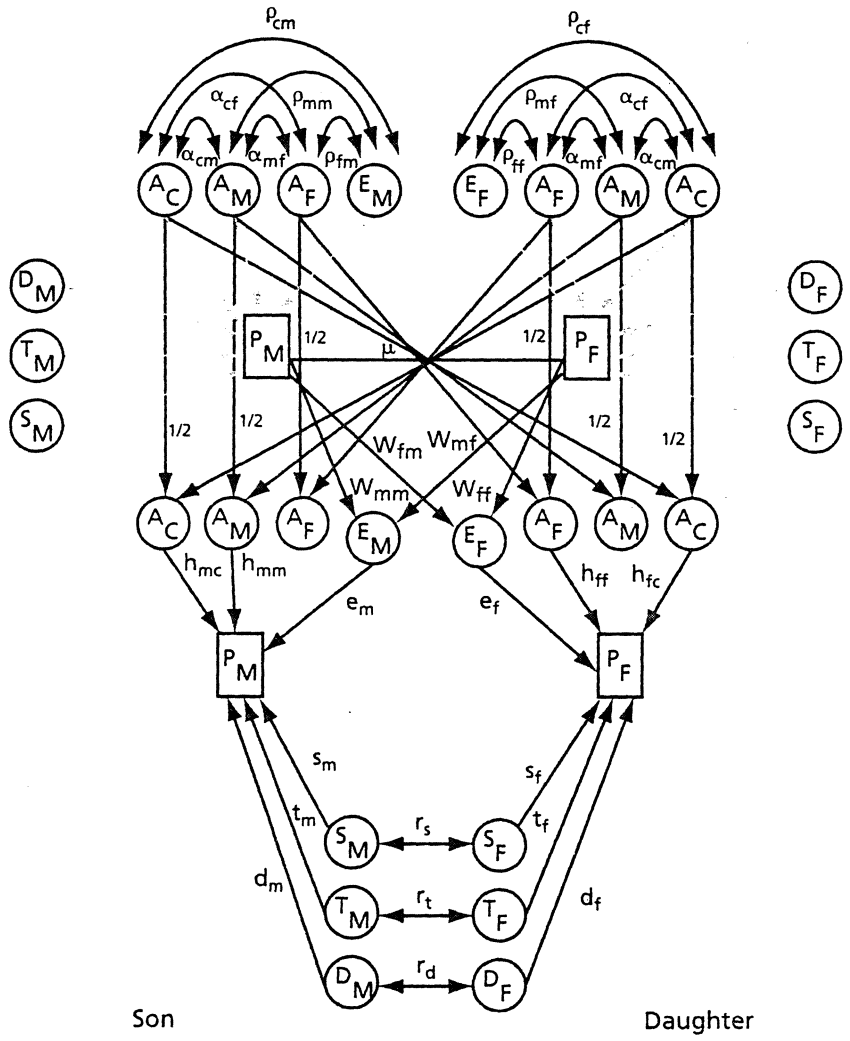

FIGURE 11–6. *Path model for biological and culture inheritance when genetic and environmental effects depend on sex.* Note. *For simplicity, the figure only presents the biological and cultural effects on the* reliable *variance in the phenotype. If assortment and cultural transmission are based on a latent trait then paths from true scores to observed scores may be included for males and females,* λ_m *and* λ_f*, respectively, as in Figure 11–5.*

males; and r_d, r_s, and r_t, for the correlations across sexes of the dominant, sibling environmental, and twin environmental effects.

Since vertical cultural transmission is assumed under this model to be based on the parental phenotype for the trait under investigation,

the question of "nonscalar" vertical cultural transmission does not apply. However, the cultural impact of mothers may differ from that of fathers and may further depend on the sex of the offspring. In the model for sex differences, therefore, we require four cultural parameters: w_{mm}; w_{mf}; w_{fm}; w_{ff}. The first subscript denotes the sex (m = male) of the offspring and the second denotes the sex of the parent.

Specification of sex-limited additive genetic effects is more difficult when there is assortative mating that induces correlations between loci that would otherwise be independent (see for example Fisher 1918). We have adopted one of several formally equivalent ways of parameterizing the additive sex-limited effects. Recognizing that the additive genetic variances in the two sexes and the genetic covariance between them require three free parameters for their complete specification, we assume that one set of genes explains all the genetic variance in females and the genetic covariance between the sexes. The paths from this "common" set of genes to the male and female phenotypes are denoted by h_{mc} and h_{fc}, respectively. A second set of genes has effects specific only to males, and the path from these genes to the male phenotype is specified by h_{mm}. Although the "male-specific" genes are not expressed in females, they are still present in females and correlated, through phenotypic assortment, with the "common genes." We denote the induced correlation between the two sets of additive genetic effects by α_{cm}.

The joint effects of assortment and vertical cultural transmission induce four genotype-environment correlations: two between the "common" additive genetic effects and the environments of males and females, ρ_{cm} and ρ_{cf}, respectively; two between the "male-specific" additive genetic effects and the environments of males and females, ρ_{mm} and ρ_{mf}, respectively. These genotype-environment correlations are estimated as constrained parameters when fitting the model (i.e., they are functions of other parameters). Separate parameters are required to specify the path from male environment to phenotype (e_m) and female environment to phenotype (e_f). Under the simple model for "phenotypic assortment with error," the paths from true score to observed score, λ_m and λ_f, may differ between males and females.

Since the total phenotypic variance is standardized to unity in both

sexes, two further constraints are required to enforce these conditions. Thus, seven constraints are imposed on parameter values under the full model. The full model for sex-limited effects is given for pairs of opposite-sex DZ twins in Figure 11–6.

Fitting the Model

The method of iterative constrained diagonal weighted least squares was used to fit the full (nonlinear) model to the 80 correlations for each personality and social attitude variable in turn. The model is fitted to the z-transforms of the raw correlations to improve the approximation to normality (see Rao et al. 1977). Truett et al. (1996) give further details of the model fitting method. The expected correlations between relatives may be derived algebraically from the path model. These are extremely complex and are not reproduced here. The Numerical Algorithms Group's FORTRAN subroutine E04UEF was used for constrained numerical minimization of the residual sum of squares (Numerical Algorithms Group 1990).

The full model, involving 19 free parameters, was fitted first. The weighted residual sum of squares for $80 - 19 = 61$ df is employed as a guide to the overall goodness-of-fit of the model. The false assumption of independence in the observed correlations is likely to result in our rejecting the model too often if we treat this statistic uncritically as χ^2. However, the implications of this strategy for comparisons of alternative models based on examination of changes in χ^2 associated with reductions in the full model are less predictable (McGue et al. 1987).

In order to test the significance of combinations of effects having particular theoretical importance, a series of reduced models was fitted in every case and the increase in the residual sum of squares noted as a guide to the deterioration in fit associated with deleting specific effects from the model. Reduced models were fitted to test the following specific hypotheses: 1) that all genetic and environmental effects were

homogenous over sexes; 2) that there were no genetic effects ($h = 0$, $d = 0$ in both sexes); 3) that there were no nonadditive (dominant) genetic effects ($d = 0$ in both sexes); 4) that there were no effects of the shared environment of any kind ($s = t = w = 0$ in both sexes); 5) that there was no vertical cultural inheritance ($w = 0$ in both sexes); 6) that assortment and cultural inheritance were based on the measured phenotype rather that a latent "true" phenotype ($\lambda = 1$ in both sexes). These tests do not exhaust all the possibilities. However, in view of the danger of capitalizing on chance with multiple tests it is more appropriate to restrict testing to a few major effects of a priori importance.

Results

Table 11–4 presents the weighted least squares estimates of the parameters of the full model for family resemblance in stature and conservatism. Table 11–5 gives χ^2 statistics testing approximate goodness-of-fit of the model and for testing the major hypotheses enumerated previously. We note that large numerical estimates of the cultural transmission parameters, w, nevertheless may imply a relatively small contribution to the total environmental variance because the path e may itself be small.

Using the parameter values from the full model in Table 11–4, the contributions of the different genetic and environmental sources to variation in the two traits were computed using the formulae given by Truett et al. (1994). The tabulated parameter values, and Truett's formulae, refer only to the *reliable* variance. Hence, for example, the value of 0.012 for the environmental path coefficient for stature in males does not imply that the environment plays no role in creating differences in stature. It does, however, imply that the environment plays little role in creating those differences that contribute to the choice of spouse. Table 11–6 summarizes the estimated contributions of the major sources of variation to differences in stature and conservatism for males and females.

TABLE 11–4.

Results of model-fitting to correlations in stature and conservatism

Parameter	Estimate	
	Stature	Conservatism
Genetic		
h_{fc}	0.940	0.580
h_{mc}	0.921	0.866
h_{mm}	0	0
d_f	0.282	0.360
d_m	0.333	0.295
r_d	−0.063	−0.002
Assortment		
μ	0.275	0.720
μ_{cm}	0	0
Cultural transmission		
w_{ff}	0.280	0.172
w_{mf}	−1.000	−0.368
w_{fm}	0.296	0.026
w_{mm}	−0.135	0.208
Other shared environment		
s_f	0.136	0.233
s_m	0	0
r_s	—	—
t_f	0.295	0.210
t_m	0.235	0.036
r_t	0.505	1
Unique environment		
e_f	−0.145	0.493
e_m	0.012	0.592
Genotype-environment correlations		
ρ_{cf}	0.332	0.124
ρ_{cm}	−0.649	−0.094
ρ_{mf}	0	0
ρ_{mm}	0	0
Latent variable		
λ_f	0.932	0.979
λ_m	0.921	0.878

TABLE 11–5.

Tests of significance and summary statistics from model-fitting analysis of stature and conservatism

Item	χ^2	df	AIC	SS%	χ^2	df	P%
Stature							
Full model	127.02	61	4.90	98.85	—	—	<.01
Sex differences	163.15	73	17.15	98.52	36.13	12	.03
Shared environment	139.21	71	−2.79	98.73	12.19	10	27.25
Vertical cultural inheritance	128.01	65	−1.98	98.84	0.09	4	99.90
Genetic effects	711.66	67	577.66	93.53	524.64	6	<.01
Dominance	140.48	64	12.48	98.72	13.46	3	.37
Assortative mating	600.91	62	476.91	97.53	473.89	1	<.01
Latent assortment	135.93	63	9.93	98.76	8.91	2	1.16
Conservatism							
Full model	65.51	61	−59.49	99.37	—	—	42.23
Sex differences	279.17	73	133.17	97.20	213.66	12	<.01
Shared environment	83.25	71	−58.75	99.17	17.74	10	5.95
Vertical cultural inheritance	79.26	65	−50.74	99.21	9.75	4	4.48
Genetic effects	507.44	67	373.46	94.92	441.93	6	<.01
Dominance	336.28	64	208.28	96.63	270.77	3	<.01
Assortative mating	3,661.04	62	3,537.01	63.33	3,595.53	1	<.01
Latent assortment	88.92	63	−37.08	99.11	23.41	2	<.01

Note. χ^2 = *Chi-square testing goodness-of-fit;* AIC = *Akaike Information Criterion;* SS% = *percentage of sum of weighted squared raw z statistics explained by parameters of model;* χ^2 = *Chi-square testing effects removed from model;* P% = *significance level (%).*

TABLE 11–6.

Estimated contributions of sources of variation to differences in stature and conservatism

	Proportion of total variation (%)			
	Stature		Conservatism	
Source	Males	Females	Males	Females
Genetic				
Additive	55.8	59.6	35.5	19.8
Assortment	16.1	17.2	22.2	12.4
Dominance	9.4	6.9	6.7	12.5
"Total genetic"	83.9	86.7	64.4	44.7
Environment				
Maternal	0.0	0.2	1.5	1.0
Paternal	0.0	0.2	0.0	0.1
Twin	4.7	7.6	0.1	4.2
Sibling	0.0	1.6	0.0	5.2
Within-family	0.0	1.4	17.5	32.4
"Error"	15.3	13.1	22.9	4.1
G-E covariance	−1.2	−7.9	−6.2	8.1

Discussion

The overall fit of the model for conservatism is surprisingly good ($P > .4$). The fit for stature is relatively poor; some of the largest individual contributions to the highly significant weighted residual sum of squares come from male cousins (13.02), opposite-sex cousins (10.48), and female cousins (8.87) (all related through male DZ twins), The fit for 300 other relationships, including those for twins and nuclear families, is quite close given the very large sample sizes. In this respect, our study seems to suffer from similar problems to those reviewed by Fisher (1918) who remarks:

> In general, the hypothesis of cumulative Mendelian factors seems to fit the facts very accurately. The only marked discrepancy from ex-

> isting published work lies in the correlation for first cousins. . . . The values found . . . are certainly extremely high, but until we have a record of complete cousinships without selection, it will not be possible to obtain satisfactory numerical evidence on this question. (p. 433)

Comparison of the results for the two variables reveals similarities and differences in the transmission of stature and attitudes. Both have genetic components. The genetic variation of both stature and conservatism is inflated by assortative mating and nonadditive genetic effects ("dominance" in our model). For stature, Fisher (1918) estimates that about 14% of the total variance is attributable to dominance effects. Our values for both sexes are somewhat lower but significantly greater than zero. The effects of dominance may obscure other nonadditive effects including the interaction of genetic differences with the effects of age and secular trends in the data. The contribution of assortative mating to the genetic variation in stature reflects a relatively low marital correlation operating on a trait that, after correction for errors of measurement, is almost completely heritable. Fisher (1918) observes "some ambiguity still remains as to the causes of marital correlations: our numerical conclusions are considerably affected according as this is assumed to be of purely somatic [phenotypic] or purely genetic origin" (p. 33) but then remarks that his results are in close agreement with partial genetic origin of assortment. Our results for stature agree with this view insofar as our data require us to exclude some of the measured effects on stature from those affecting assortment. In our model, these are referred to as "error." In contrast, the pervasive effects of assortment on family resemblance for conservatism reflect a much higher degree of phenotypic ("somatic") assortment for a trait that is affected significantly less by additive genetic effects. Nongenetic differences within MZ twin pairs are seen to have an impact on the choice of mate for conservatism.

Fisher (1918) notes that most of the evidence for dominance, in Pearson and Lee's data, comes from the sibling correlation. He ignores the possibility that nongenetic effects may also be correlated between relatives. Our design includes MZ twins and their relatives, which

provides additional tests for the effects of nonadditive genetic factors and allows a more subtle analysis of the environment. For stature, Fisher notes (1918, p. 433) that "examination of the best possible available figures for human measurements shows that there is little or no evidence of nongenetic causes." Our data give little reason to disagree with this early finding as far as stature is concerned once we allow for the effects of errors of measurement in our self-reported observations. Our data include MZ twins, which give a more direct measure of nongenetic effects. The results for the Virginia 30,000 also give little reason to infer a marked role for nongenetic inheritance for human stature.

How far is Pearson's attempt to universalize these findings to human behavior justified? By assessing human attitudes we have attempted to operationalize those essentially "human" characteristics that, unlike cognition and personality, defy attempts to develop convincing animal models. Social attitudes cannot exist without culture. Normative attitudes change continually. Yet there are enormous individual differences that we have attempted to quantify in our survey. Pearson's early claim that the inheritance of human behavior owed nothing to any characteristically "human" principle was overstated but not unfounded. It is certainly the case that nuclear family resemblance in conservatism is almost as great as that for stature (see Figure 11–2). However, it is misleading to assert that this result points to an equivalent genetic contribution because the effects of assortative mating on the correlations for conservatism are considerably greater than those for stature. The total environmental variance for conservatism is approximately twice that for stature. Most of the environmental effect, however, is due to differences within families and fluctuations within individuals over time that contribute to greater differences within MZ twin pairs. Although conservatism indeed differs from stature in showing a significant effect of vertical cultural inheritance from parent to child, the contributions of parent-offspring social transmission to adult attitude differences are relatively small compared with other genetic and environmental effects.

There are significant differences between the sexes in the relative contributions of genes and environment. These effects were ignored in the early studies. We believe our study is the first to attempt an empirical resolution of all the different genetic and environmental

sources of sex differences in the expression of genetic and environmental effects. These effects are greater for conservatism than stature and are consistent with women being somewhat more influenced by environmental factors than men. The effects of vertical cultural inheritance, though contributing only a relatively small amount to sibling resemblance, appear to depend slightly more on mothers than fathers.

By focusing on those characteristics that attitudes have in common, as we have done by using a scale score based on a large number of items that are only weakly correlated, it might be argued that we have ignored those idiosyncrasies of style that might be more sensitive to social learning than overall tendencies. Furthermore, the general "conservatism" factor is only a first approximation to a more complex factor structure underlying these items. Any heterogeneity in the causes of variation in among the primary factors will be obscured in this broader analysis.

The similarities in the pattern of family resemblance for stature and attitudes point to important communality between the origins of physique and behavior. They suggest that there is no "sacred territory" in human behavior that can be delineated simply by virtue of its immunity from the effects of genes. The effects of genes reach the highest points of culture. In this respect, Pearson was probably correct in regarding biological inheritance as the theme that unified the "mental and moral" characteristics of humans with "[even] the protopodite of the water flea." This conclusion has important cultural implications for how humans perceive themselves. It is probably for this reason that many critics have resisted it with a zeal that has sometimes precluded creative reflection. Clearly, our findings and others like them have within themselves the seeds of hubris. We may not conclude too hastily that humans are "nothing but" their genes or that because thought and feeling cannot directly alter stature they have no effect on behavior. Behavior-genetic studies like the Virginia 30,000 are still blunt instruments. Within the range of possible pathways from genotype to phenotype there remain a number of radically different alternatives, all of which would lead to patterns of family resemblance suggesting the importance of genetic factors. Cross-sectional studies of family resemblance do not have much power to resolve the ontogenetic role of the

individual as "actor" in the creation of his or her phenotype. The process of human development and adaptation has been committed by evolution to an organism that engages in more or less continual dialogue with the ecosystem. The individual behavioral phenotype at any moment is a selective record of this conversation. If the findings of behavioral genetics are to be believed, the dialogue between the individual and the ecosystem does not begin at birth with a blank tape but with some of the experiments, successes, and failures of previous generations encoded in the genetic material. In humans, these are not primarily the stereotyped patterns of other species but rather clues about salient features of the environment and strategies for dealing with novelty and change. The fact of genetic diversity yields individuals whose conversations with the environment take on different shapes ontogenetically. Different individuals attend somewhat differently to the features of their environment, try different experiments, and respond differently to the answers. The result is not pure "chaos," however, as would be the case if the genotype set only the initial conditions of the ontogenetic dialogue. Rather, the outcome is an ordered set of correlations between relatives, which follow more closely the pattern of biological rather than purely cultural inheritance. Thus, we conclude that the genotype is at the center of human action, even of human creativity, by virtue of setting the basic parameters of the organism's continual adaptation by selecting, assimilating, and transforming information derived from the environment.

The path from gene to behavior is long and complex, involving the nonrandom selection of the environment by individuals of different genetic liability or the nonrandom imposition of environmental treatments on individuals, even within the family, as a consequence of perceived genetic differences. Insofar as inherited differences bias the individual toward different features of the environment, we expect a correlation between the effects of genes and environment on the phenotype ("genotype-environment correlation," Cattell 1960, 1963; Eaves et al. 1977; Jinks and Fulker 1970; Plomin et al. 1977). The type of genotype-environment correlation generated by the individual as "actor" will lead, in cross-sectional studies, to environmental effects that cannot be separated from other genetic effects (Jinks and Fulker 1970). Indeed, in one sense they are "genetic" effects since they arise because

genetic differences between individuals have resulted in the differential selection and elicitation of particular environmental effects. Such genotype-environment correlations constitute one aspect of what Richard Dawkins (1982) has called the "extended phenotype."

Insofar as genetically different individuals respond differently to changes in their environment we expect, in the statistical sense, "genotype × environment interaction." Such interactions were termed "plasticity" in the early work of Cavalli-Sforza and Feldman (1973) on the theme of cultural inheritance. The consequences of the interaction of genotype and environment for family resemblance have been traced theoretically by Jinks and Fulker (1970). Interactions between genetic effects and environmental differences *within* families will be confounded with the effects of the within-family environment in cross-sectional studies. Interactions between genetic effects and environmental differences *between* families (such as those created by vertical cultural inheritance) will, in our types of families, be divided between genetic effects and those of the family environment. Thus, our cross-sectional study gives little leverage on the precise way in which genes and environment correlate and interact during development.

The fact that our study is cross-sectional and limited to adults implies a relatively simple-minded and "static" conception of the role of the family environment and parental treatment in behavioral development. It has commonly been assumed that the effects of parental treatment are additive to those of the genes and persistent throughout life. The primary adaptive role of parents may, however, be to support the organism during development so that it survives long enough with the cognitive and social skills necessary to adapt freely to the niches available to it as an adult. Long-term reliance on parents as sources of specific values in a rapidly changing environment may be maladaptive. Thus, it may be naive to expect a large persistent direct nongenetic effect of parents on the adult attitudes of their children. The study of attitude development in younger people, especially among adolescent twins and adoptees, may tell a different story and reveal a large but transient effect of the shared environment on the attitudes of younger people. On the other hand, if the large spousal correlations for attitudes are any criterion, spouses invest a lot of their resources in choosing a partner for life and reproduction of very similar attitudes. Explanations

of the kind "people who have similar attitudes tend to get on better" beg the more fundamental question of why they should care so much about attitudes and invest so little in other aspects of personality such as extraversion and neuroticism (see for example Eaves et al. 1989) for which spousal correlations are consistently zero.

We caution that the results for social attitudes cannot be generalized to the point where it is assumed that *all* differences in behavior have a genetic component. An analysis of religious affiliation in twins and their parents, for example (Eaves et al. 1990), shows that genes play little or no role in determining whether or not children follow in the religious traditions of their parents. Such counterexamples lead us to give even more credence to our current findings because we are compelled to conclude that the finding that nongenetic effects play such a small part in the transmission of social attitudes cannot simply be explained away by a philosophical prejudice of the researchers or by the inherent bias of the research design.

We have stressed that our use of a single general conservatism factor is an oversimplification of the structure of social attitudes. Truett et al. (1992) show that there are several correlated primary factors underlying the general conservatism factor that show differential patterns of association with indices of religiosity and education. Further analysis of the Virginia 30,000 will clarify these complex relationships.

No human science, genetic or social, is wholly free from the problems inherent in the epistemic distance demanded by scientific "objectivity." Paul Ricouer has observed (1967) that there is no platform from which all human diversity can be viewed with absolute detachment, "the objectivity of science, without a point of view and without a situation, does not equalize cultures except by neutralizing their value; it cannot think the positive reasons for their equal value." As culture tries to deal with the new insights of human genetics, particularly behavioral genetics, humans need to be clear about what is offensive to their humanity. The offense lies not in whether human behavior is conceived in "genetic" or "environmental" terms, or some combination of both. The scientific "facts" do not disengage humans from the moral imperatives that constitute the wellsprings of social action and even of science itself. Genetic *and* environmental explanations both become offensive at the point where they attribute more

or less worth to individuals by the circumstances and gifts of their birth. Critics of too close an alliance between "science" and "state" can point to the historical abuses of the scientific cores of both Marxist and Darwinian theories (see for example Popper 1960).

The impetus to genetic research and technology provided by the human genome initiative has resulted (e.g., Duster 1990; Kevles and Hood 1992) in the resurrection of the specter of "genetic determinism" and the non sequitur that behavioral genetics is inherently a reactionary science that threatens the freedom and dignity of humans. Such views are superficial, scientifically and philosophically. Genetic models are no more nor less "deterministic" than environmental models. Neither are they any more or less "materialistic." They are no more nor less a "threat to humanity" than any other branch of the human sciences. Both seek to analyze the complexity of human behavior in terms of more abstract principles that are conceived as "causes." Whether these causes are genetic or environmental makes little logical or practical difference. We note that one of the most rigorous proponents of "determinism" was a psychologist who set little store by the role of genes in behavior (e.g., Skinner 1971). What genetic studies may have done is to temporarily "disenfranchise" a series of vaguer social paradigms for the understanding of behavior. Among "deterministic" models, genetic models have become serious contenders for the elucidation of "cause." Among "materialistic" models, genetic models have offered a prime candidate for the material ground of behavior in the shape of the double helix.

The human sciences have to be judged by how well they deal with the "*humanum.*" Scientifically speaking, this success does not depend, initially, on whether the underlying principles of explanation are genetic or environmental but on how far such principles render intelligible the most characteristic qualities of the organism. Thus, a model that takes into account the role of genes in the ontogeny and identity of humans who show initiative, creativity, and even "freedom" is, at one level, paying greater tribute to humanity than an "environmental" one that denies the individual any active role in shaping his or her destiny. Our data on social attitudes do not preclude nongenetic effects but they do make it increasingly difficult to discount the role of genes in shaping the developmental trajectory of humans at the level of traits,

such as social attitudes, that have little meaning outside the human domain.

The broader cultural and philosophical implications of genetic studies of human behavior are far from clear. Studies such as the Virginia 30,000 strongly suggest that human's understanding of their humanity will require that they deal at every level with the relationship between the human genome and the highest faculties of affect and cognition with which they have been gifted by natural selection. Legitimate scientific criticism of careless research will continue to play an important part in this process. It is no less urgent that philosophers, historians, sociologists, and other commentators start to engage the findings of behavioral genetics positively in ways that owe more to ingenuity and less to panic.

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