Genetics of Sexual and Social Attitudes in Twins

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While most social scientists have adjusted to the idea that variation in cognitive abilities has a genetic component, many would regard it as improbable that such allegedly malleable traits as attitudes to social or sexual questions could be influenced by genetic differences between individuals. Most would guess that social forces and cultural inheritance were more potent molders of individual differences in political opinions. A simple screening test for the relative importance of these sources of variation is provided by the classical study of monozygotic (MZ) and dizygotic (DZ) twins reared together.

THE DATA

The results from three different twin studies are summarized in this paper. The three twin samples were obtained at intervals of two to three years by postal questionnaire from the Maudsley Twin Register maintained at the Institute of Psychiatry, London, by Professor HJ Eysenck and Mrs J Kasriel. The register consists of volunteer twins, 18—56 years old at the time of sampling, obtained through appeals in the media, and makes no claim to either randomness or representativeness. Nevertheless most pertinent sample means and variances do not differ markedly from those found in carefully randomized samples. Zygosity was ascertained largely by postal questionnaire, the reliability of which was checked by blood grouping a subsample of the twins. The question of zygosity diagnosis in the Maudsley Twin Register is discussed fully in Kasriel and Eaves [1976]. The breakdown of the three samples by zygosity and sex can be seen in Table III.

Study I obtained responses of 823 twin pairs to an early 68-item version of the Eysenck Public Opinion Inventory. This was scored for the two principal factors extracted — a Radical vs Conservative dimension and a Tough- vs

Tenderminded dimension. Preliminary results of genetical analysis of this survey have been reported by Eaves and Eysenck [1974] and more extensively by Hewitt [1974].

Study II was an anonymous survey whose principal aim was to obtain responses to Eysenck's Sexual Attitudes Questionnaire [Eysenck, 1976]. Because of the nature of the survey, only 246 pairs responded. Two main attitudes factors were extracted and labeled "Sexual Satisfaction" and "Libido." In addition, responses to a more recent 88-item version of the Public Opinion Inventory were obtained and scored for factors Radicalism and Toughmindedness. Results of this study have been discussed fully in Martin [1977] and published in Martin and Eysenck [1976] and Martin et al [1977].

Study III used a quite different questionnaire, the Wilson-Patterson Conservatism Scale [Wilson, 1973], and obtained factor scores on the Radicalism dimension for 587 twin pairs. No factor equivalent to Eysenck's "Toughmindedness" could be extracted. The results of this study are discussed fully in Last [in preparation] and the joint results and conclusions of all three studies will be published by Eaves et al [in preparation].

THE ANALYSIS AND ITS POWER

The biometrical genetic approach to the analysis of twin data has now been discussed extensively in the literature [eg, Eaves and Eysenck, 1975; Martin, 1975]. Briefly, after the data have been rescaled to remove/scale-dependent genotype-environment interaction (detected by the regression of MZ pair variances on pair means), between- and within-pairs mean squares for each twin group are obtained by analysis of variance. Variance due to age-dependent regression is removed from the between-pairs mean squares and variance due to a mean difference between males and females removed from the opposite-sex within-pair mean square. Models may now be fitted to the corrected observed mean squares by the method of weighted least squares which produces approximately maximum likelihood parameter estimates and allows a χ^2 test of goodness-of-fit of the model.

Given the practical impossibility of detecting dominance in twin studies of most behavioral characters [Eaves, 1972; Martin et al, in press], the most usual models to be fitted are subsets of that shown in Table I. Here E_1 is the within-families environmental variation which includes environmental experiences specific to the individual and errors of measurement, and D_R is the additive genetic variance component defined by Mather and Jinks [1971]. The third parameter B is a between-families component of variance in which between-families environmental variation E_2 (cultural and family treatment effects) is completely confounded with the extra additive genetic variation which accrues between families as a result of assortative mating. This confounding can be seen in the expression:

$$B = E_2 + \frac{1}{2} D_R \left[\frac{A}{1-A} \right],$$

TABLE I. Basic Model for Mean Squares of a
Classical Twin Study

	E_1	B	D_R
MZF[a]			
b	1	2	1
w	1	0	0
MZM			
b	1	2	1
w	1	0	0
DZF			
b	1	2	¾
w	1	0	¼
DZM			
b	1	2	¾
w	1	0	¼
DZO			
b	1	2	¾
w	1	0	¼

[a]Abbreviations: MZF, monozygous female; MZM,
monozygous male; DZF, dizygous female, DZM,
dizygous male; DZO, dizygous opposite sexes;
b, between; w, within. Other abbreviations defined
in text.

where A is the correlation between the additive deviations of spouses and is related
to the phenotypic marital correlation μ by $A = h^2\mu$, where h^2 is the narrow
heritability.

When significant estimates of both D_R and B are obtained, we cannot even
guess at the relative contributions of E_2 and assortative mating to $\hat{B}$ without some
independent evidence about the size of marital correlation.

A more fundamental statistical question arises when we consider the power of
our experiment to reject inappropriate models of variation. There is no general
solution to this problem, so we must calculate the probability of rejecting inap-
propriate models of variation with samples of given size taken from imaginary
populations whose true components of variation are known. These calculations
are based upon the noncentral χ^2 distribution and are explained in detail in
Martin et al [in press]. We shall consider only a few of their results, shown here
in Table II. This table shows, for example, that in twin samples consisting of 50%
MZ and 50% DZ pairs drawn from a population in which the "true" components
of variation were one-half E_1 and one-half E_2, 430 pairs (215 MZ and 215 DZ)
would be required to reject an inappropriate $E_1 V_A$ ($V_A = 1/2D_R$) model at the
5% level of significance in 95% of such studies, while in the converse case, 640

TABLE II. Total Number of Pairs Required for 95% Rejection of False Hypotheses at 5% Level

True model			False model P (MZ)				
			0.1	0.3	0.5	0.7	0.9
$0.5E_1$	$0.5E_2$	E_1V_A	298	324	430	696	2,055
$0.5E_1$	$0.5V_A$	E_1E_2	2,181	852	640	670	1,344
		$\left\{ E_1E_2 \right.$	1,798	660	466	455	848
$0.4V_A$	$0.3E_1$	$0.3E_2$					
		$\left. E_1V_A \right.$	645	718	966	1,583	4,715

pairs would be required to reject an inappropriate E_1E_2 model when the population variance is one-half E_1 and one-half additive genetic. In general it is easier to reject an inappropriate simple genetic (E_1V_A) model than an inappropriate simple environmental (E_1E_2) model in equivalent cases.

In the lower part of Table II we consider a more complex case in which the true population variance is attributable roughly one-third each to additive genetic, E_1, and between family (E_2 or assortative mating) sources. In this case we want to know the sample sizes required to reject (in 95% of studies at the 5% level) both the inappropriate two-parameter models. For all reasonable sample compositions it is easier to reject the E_1E_2 model than the E_1V_A model, although, with roughly one-third MZ pairs, 95% power of rejection of both models can be achieved with around 700 pairs. For 50% MZ twins, however, roughly twice as many twins are required to achieve 95% power of rejection of the E_1V_A model (966 pairs) as are needed to reject the E_1E_2 model (466 pairs). This is the approximate composition of the three twin samples we shall be considering. In this connection it is worth noting that, for these experiments, roughly 80% power can be achieved with samples around two-thirds the size of these and roughly 50% power with samples around one-third these sample sizes.

RESULTS AND DISCUSSION

With these power considerations in mind, we may now consider the results of model fitting to the data. The mean squares for Radicalism from the three twin studies (measured on three different scales) are shown in Table III, and the goodness-of-fit of various models fitted to these data is shown in Table IV.

It can be seen that the E_1E_2 model is decisively rejected in studies I and III and gives a very poor account of the data in study II (where the sample size is smallest). The simple genetic (E_1D_R) model is rejected in study I, fails marginally in

TABLE III. Observed Mean Squares From Three Twin Studies
of Radicalism

	Study I (823 pairs)		Study II (246 pairs)		Study III (587 pairs)	
	DF	MS	DF	MS	DF	MS
MZF_b	323	8.36	93	337.97	231	112.92
w	324	1.90	95	61.50	233	24.95
MZM_b	141	10.31	37	356.52	81	113.77
w	142	1.78	39	49.20	83	30.02
DZF_b	193	9.51	52	365.26	145	121.84
w	194	2.89	54	100.83	147	39.17
DZM_b	36	8.85	15	272.48	50	124.96
w	37	3.21	17	82.38	52	44.89
DZO_b	126	10.11	39	350.61	70	128.16
w	126	3.29	41	129.35	72	49.11

TABLE IV. Results of Model Fitting for Radicalism

	Study I	Study II	Study III
E_1E_2 model χ_8^2 =	24.74**	15.17*	21.03**
E_1D_R model χ_8^2 =	20.61**	7.70	14.28*
E_1D_RB model χ_7^2 =	7.51	3.26	6.20

*0.05 < P < 0.10.
**0.001 < P < 0.01.

study III, but gives an adequate account in study II. Clearly studies I and III
indicate the need for all three parameters in an adequate model, and this gives an
excellent fit to the data in the two largest studies. Although the third parameter
is not strictly needed in study II, it does cause a significant reduction in the
residual chi-square (χ_1^2 = 4.4), justifying its inclusion in the model.

The congruence of these three studies is even more marked when we examine
the breakdown of the total variation shown in Table V.

In each study, roughly one-third of the variation is attributable to within-family
environmental variation (E_1), one-third to additive genetic (V_A), and one-third
to a between-family component (B) which may be E_2 or additional genetic varia-
tion due to assortative mating, or both.

The only leverage we can get on this question is various estimates of the pheno-
typic marital correlation (μ) for the Radical-Conservative dimension obtained for

TABLE V. Sources of Variance for Radicalism (%)

	Study I	Study II	Study III
E_1	33.3	27.3	35.1
V_A	35.4	44.3	37.6
B ⟨E_2 / A.M.⟩	31.3 ⟨15.7 / 15.6⟩	28.4 ⟨0.0 / 28.4⟩	27.3 ⟨18.6 / 8.7⟩
	($\mu = 0.60$)	($\mu = 0.67$)	($\mu = 0.40$)

the three scales from independently collected husband-wife samples. These were $\hat{\mu} = 0.60$ for the scale used in study I, $\hat{\mu} = 0.67$ for the scale used in study II, and $\hat{\mu} = 0.40$ for the scale used in study III. It might be argued that these remarkably high correlations arise from a convergence of opinions over the years of marriage. We had no direct test of this but were able to regress absolute husband-wife differences on mean pair age (presumably a reasonable index of length of marriage) and, over quite a wide age range, found no significant regression. It appears, then, that the high marital correlation is a good reflection of the degree of assortative mating for this trait rather than of convergence of attitudes. If we substitute these values of $\hat{\mu}$ and the estimates of D_R into

$$A = h^2\mu$$

$$= \frac{\mu([1/2]D_R [1 + A/(1 - A)])}{\hat{V}_T} \quad ,$$

where $\hat{V}_T = \hat{E}_1 + \hat{B} + (1/2) \hat{D}_R$, we can obtain estimates of A and hence $(1/2)D_R A/(1 - A)$ and (by subtraction from B) E_2. This breakdown has been done for each of three studies and is shown at the bottom of Table V. The figures show that all of B could be accounted for by assortative mating in study II (the least reliable), one-quarter in study III, and about one-half in study I. These figures would raise the heritability to around 50%, while "true" E_2 due to cultural influences and parental transmission would account for perhaps less than one-fifth of the total variation. However, to obtain stronger evidence on these points, data on the parents of twins and on adopted families will be needed. These data are currently being collected and analyzed in our laboratory.

For Radicalism, there is no evidence that genetic and environmental components of variation are not the same in males and females. When we inspect the mean squares for Toughmindedness shown in Table VI, however, it appears that,

TABLE VI. Sex Differences in Genetic Architecture

| | Toughmindedness | | | | Libido | |
| | Study I (823 pairs) | | Study II (246 pairs) | | | |
	DF	MS	DF	MS	DF	MS
MZF$_b$	323	10.37	93	383.2	93	312.9
w	324	1.86	95	79.5	93	125.2
MZM$_b$	141	7.82	37	391.0	37	376.0
w	142	3.20	39	93.4	39	93.0
DZF$_b$	193	7.52	52	248.9	52	272.4
w	194	3.00	54	125.1	54	99.8
DZM$_b$	36	6.57	15	548.8	15	312.4
w	37	3.37	17	62.9	17	155.4
DZO$_b$	126	8.13	39	299.1	39	287.3
w	126	4.32	41	148.6	41	180.8

while genetic components of variation are important in females, there is no such evidence in males. Inspection of the mean squares for the attitude trait Libido suggests exactly the reverse, with a strong cultural effect acting in females but a genetic component important in males. These traits both suggest some sort of sex limitation of cultural and genetic effects.

Eaves [1977] has provided a model for such sex-limited effects which is shown in complete form in Table VII. This model is not of full rank, and in any case, a parsimonious description would demand fewer parameters. There are many possible sensible subsets of these parameters, and there is a real danger of fitting them all and picking the one that fits best. Let us, however, formalize our observation that there appears to be no genetic variation for Toughmindedness in males, nor for Libido in females, but allow E_2 variation in both sexes. The results of fitting these models are shown in Table VIII.

The models give an excellent fit to the data in all three cases. For both studies of Toughmindedness there is a large and significant additive genetic component for females with a correspondingly small E_2 component, while for males the E_2 component is large. For both Toughmindedness and Libido in study II the total variances for males and females have been deliberately equalized. This has not been done in study I which explains why different E_1 components are needed for males and females.

The component $\hat{E}_{2MF}$ is an estimate of the covariation between E_2 effects acting in males and E_2 effects acting in females. Consequently $r_{MF} = E_{2MF}/(E_{2M}, E_{2F})^{\frac{1}{2}}$ is a measure of the correlation between E_2 effects acting in males and females. It can be seen from the model that this information comes from the opposite-sex pairs and emphasizes the importance of including these

TABLE VII. Model for the Covariation of Genetic and Environmental Effects in Mean Squares of DZ Opposite-Sex Twin Pairs

	E_{1_m}[a]	E_{1_f}	E_{2_m}	E_{2_f}	$E_{2_{mf}}$	D_{R_m}[a]	D_{R_F}	$D_{R_{mf}}$
MZB_{males}	1	–	2	–	–	1	–	–
MZW_{males}	1	–	–	–	–	–	–	–
$MZB_{females}$	–	1	–	2	–	–	1	–
$MZW_{females}$	–	1	–	–	–	–	–	–
DZB_{males}	1	–	2	–	–	¾	–	–
DZW_{males}	1	–	–	–	–	¼	–	–
$DZB_{females}$	–	1	–	2	–	–	¾	–
$DZW_{females}$	–	1	–	–	–	–	¼	–
DZB_{m-f}	½	½	½	½	1	¼	¼	¼
DZW_{m-f}	½	½	½	½	–1	¼	¼	–¼

[a] D_{R_m}, D_R effect for males; D_{R_f}, D_R effect for females; $D_{R_{mf}}$, covariance of D_{R_m} and D_{R_f}; similarly for E_2 and E_1.

TABLE VIII. Examples of Sex Limitation?

	Toughmindedness		Libido	
	Study I	Study II	Libido	
$\hat{E}_{1F}$	= 1.8***	$\hat{E}_1$ = 80***	$\hat{E}_1$	= 111***
$\hat{E}_{1M}$	= 3.3***			
$\hat{E}_{2F}$	= 1.4**	$\hat{E}_{2F}$ = 25	$\hat{E}_{2F}$	= 94***
$\hat{E}_{2M}$	= 2.3***	$\hat{E}_{2M}$ = 173***	$\hat{E}_{2M}$	= 38
$\hat{E}_{2MF}$	= 1.6***	$\hat{E}_{2MF}$ = 81***	$\hat{E}_{2MF}$	= 49
$\hat{V}_{AF}$	= 2.6**	$\hat{V}_{AF}$ = 108*	$\hat{V}_{AM}$	= 95
χ^2_4	= 3.56	χ^2_5 = 2.96	χ^2_5	= 1.94

*0.01 < P < 0.05.
**0.001 < P < 0.01.
***P < 0.001.

pairs in any twin study. If this correlation is zero, it can be seen that the opposite-sex between mean square will equal the within mean square so that the intraclass correlation is zero. For the two studies of Toughmindedness, r_{MF} = 0.89 and 1.23, respectively, neither being significantly different from 1, indicating

TABLE IX. A Competition Model Fitted to Data for Sexual Satisfaction in Females

		E_1	D_R	D'_R	df	Observed MS	Expected MS
MZ	b	1	1	2	93	269	270
	w	1	—	—	95	141	141
DZ	b	1	¾	1½	52	241	238
	w	1	¼	—½	54	275	275

$$\hat{E}_1 \quad 141.1 \pm 20.4 \qquad c = 6.92***$$

$$\hat{D}_R \quad 331.8 \pm 118.9 \qquad c = 2.79**$$

$$\hat{D}'_R \quad -101.2 \pm 55.5 \qquad c = 1.82$$

$$\chi^2_1 = 0.005$$

$$\hat{h}^2_{MZ} = [(\tfrac{1}{2})(\hat{D}_R + 2\hat{D}'_R)]/[(\tfrac{1}{2})(\hat{D}_R + 2\hat{D}'_R) + \hat{E}_1] = 0.3145 \pm 0.0882$$

$$\hat{h}^2_{DZ} = [(\tfrac{1}{2})(\hat{D}_R + \hat{D}'_R)]/[(\tfrac{1}{2})(\hat{D}_R + \hat{D}'_R) + \hat{E}_1] = 0.4497 \pm 0.0983$$

**0.001 < P < 0.01.
***P < 0.001.

that the same E_2 effects which act in males also act in females but have much less effect.

In Libido, we see exactly the reverse pattern, with the males exhibiting genetic variation but all the female variance adequately explained by E_1 and E_2 effects, although only two of the parameters here reach significance.

Although these results certainly demand further replication, I would like tentatively to suggest that, while genetic variation acts for Toughmindedness in females and for Libido in males, it is suppressed by cultural pressures to conform to family standards in the other sex. A woman's liberationist might interpret this as evidence of conditioning to sexual norms, but it may be commented that there appears to be a great deal of variation in these norms between families.

Finally I wish to discuss the case of variation in the attitudes scale "Sexual Satisfaction." This has a standard pattern of inheritance, with low heritability in males, but in females produces the bizarre pattern of mean squares shown in Table IX.

We note that not only is the total DZ variance much greater than the MZ variance but the DZ within mean square is greater than the between mean square, yielding a negative intraclass correlation. One's first reaction might be to attribute these

results to sampling error, but they happen to be exactly the pattern of mean squares expected to be produced by the genetic effects of competition between siblings. If the extent to which sibs compete for a limited resource is dependent upon their genetic similarity, then it is evident that competition will be much more intense within MZ pairs than within DZ pairs. Consequently, variation between MZ pairs will tend to decrease relative to variation between DZ twins, and to the extent that the competition has a genetic basis, variance within DZ pairs will increase. These considerations are developed and formalized by Eaves [1976], who provides the model shown in Table IX, and fitted to the data for Sexual Satisfaction. The parameter D_R' is almost significantly negative indicating the presence of a large competition effect based upon genetic similarity — a kind of genotype-environment covariation. This case is discussed more fully by Martin and Eysenck [1976] and Eaves [1977], but in verbal terms we may suggest that female twins are competing for male attention and that this competition is more intense among MZ females (where the male has a real dilemma) than among DZ females (where presumably the choice is usually more obvious). Since success in the competition has a genetic component, genetic differences will play a more important part among DZ females, and this explains the higher heritability for DZ twins than for MZ twins shown in Table IX.

The most important point to take from this case, however, is that there can be a rational genetic explanation for unequal MZ and DZ total variances and for negative intraclass correlations.

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