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Sexual Orientation, Sexual Identity, and Sex-Dimorphic Behaviors in Male Twins

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Sexual orientation, sexual identity, and sex-dimorphic behaviors were assessed concurrently and retrospectively, for childhood, in 95 pairs of male monozygotic (MZ) twins and 63 pairs of dizygotic (DZ) twins. There was a significantly higher rate of adult homosexuality among the MZ than among DZ twins. We employed a model-fitting approach using LISREL to test for genetic and environmental influences on variation for each trait singly and on the covariation among all six traits (three for childhood and three for adulthood). Univariate analyses confirmed the presence of familial factors for five of the six variables but were generally unable to distinguish shared environmental from genetic influences. Hierarchial tests of multivariate models supported the existence of an additive genetic factor contributing to the covariance among the variables. More restrictive multivariate models yielded a significant genetic influence on sexual orientation. Because of the different rates of orientation by zygosity and because of the restrictive nature of some of the multivariate models, our results are best considered tentative but do suggest that further biometrically oriented studies of sexual orientation and its correlates would be worthwhile.

KEY WORDS: sexual orientation; twins; heritability.

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INTRODUCTION

Despite increasing interest in biological explanations of sexual orientation (e.g., Ellis and Ames, 1987), there has been little research concerning the relative importance of its environmental and genetic determinants. Considered individually, past genetic studies of sexual orientation tend to be inadequate. Taken together, however, they do suggest that biometrical investigations should be fruitful. The following discussion is restricted to variation in sexual orientation and related traits among human males.

Kallman (1952a, b) reported that 37 of 40 monozygotic (MZ) male twin pairs were concordant for homosexuality, compared to only 4 of 26 dizygotic (DZ) pairs. (In the three MZ pairs not known to be concordant, there was incomplete information on one twin.) Although the study has been severely criticized on methodological grounds (Rosenthal, 1970), the high degree of concordance is difficult to explain without positing substantial genetic influence. Pooling the data from a number of small twin studies, 11 of 18 MZ twin pairs were concordant, compared to 1 of 13 DZ twin pairs (Lange, 1931; Sanders, 1934; Habel, 1950; Heston and Shields, 1968). Although less impressive than Kallman's nearly perfect concordance rate for MZ twins, these data support the likelihood of a strong genetic influence on sexual orientation. However, the fact that 7 of 18 MZ pairs were discordant for homosexuality, in conjunction with several case reports of discordant MZ pairs (Klintworth, 1962; Zuger, 1976; McConaghy and Blaszczynski, 1980), suggests that the etiology of male homosexuality is complex. An explanation involving only a major gene with complete penetrance is untenable from the MZ data alone.

More recently, Eckert *et al.* (1986) reported the sexual orientations of two pairs of male MZ twins who were separated in infancy. One pair, aged 25, was clearly concordant for homosexuality, while the status of the other pair, aged 35, was uncertain. In a family study of sexual orientation, Pillard and Weinrich (1986) found that about 20% of brothers of male homosexuals were homosexual or bisexual, compared to about 4% of brothers of heterosexuals. This study has the strengths of careful methodology and relatively large sample size; however, a family study design is incapable of resolving causality. The aforementioned studies are consistent with the possibility of strong genetic influence on male homosexuality. Their collective deficiencies indicate a need for studies with large, representative samples collected in a systematic fashion.

A further limitation of these studies is their exclusive focus on sexual orientation to the exclusion of important related phenomena, particularly

sex behavior typical of girls (opposite sex-dimorphic behavior) and feminine sexual identity. Maccoby (1987) has emphasized that terms dealing with masculinity, feminity, gender identity, sexual identity, and sex-dimorphic behavior have been defined inconsistently throughout the literature. For the present study, we have defined opposite sex-dimorphic behaviors as those characteristically seen in females and not males, the extreme of which is recognized as "sissy" behavior. Sexual identity is a person's sense of (but not necessarily comfort with) being male or female.

Marked childhood opposite sex-dimorphic behavior in boys is the best predictor of adult homosexuality (Bell *et al.*, 1981; Green, 1987). With few exceptions, previous genetic studies have either preceded or ignored evidence regarding this important development antecedent of a homosexual orientation. The report of a pair of male MZ twins who were discordant for sexual identity and sex-dimorphic behavior at age 8 (Green and Stoller, 1971) but who were concordant for bisexual orientation at age 24 (Green, 1987) suggests that the determinants of childhood sex-dimorphic behavior and sexual orientation may differ. Supporting this view, male-to-female transsexuals, who exhibit the most extreme form of feminine sexual identity and sex-dimorphic behavior, contain a minority who report at least some sexual interest in females (Buhrich and McConaghy, 1977). More recently, Gooren *et al.* (1989) related that one of four MZ pairs and one of four DZ pairs were concordant for male-to-female transsexualism. The authors concluded that genetic factors were unlikely to be important in the etiology of that condition. Given the importance of sex-dimorphic behavior as a predictor of adult male homosexuality, but also given the evidence that sex-dimorphic behavior and sexual orientation do not have identical determinants, multivariate biometrical analyses which explore the causes of the relationship between the two traits would be highly informative. Similarly, although an association between sexual identity and homosexual feelings has been established in adults (McConaghy *et al.*, 1979), no study to date has examined the causes of this association.

A final criticism of previous genetic studies of sexual orientation concerns the data analytic techniques used and subsequent reporting of results. All prior twin studies have merely reported concordance rates and tested for the significance of the difference in rates by zygosity. While this is a valid test for the presence of genetic influence, it is a limited approach. First, it ignores the evidence that sexual feelings (McConaghy *et al.*, 1979) and sexual orientation (Kinsey *et al.*, 1948) have a continuous (if skewed) distribution. Second, if one assumes multifactorial causation, then it is appropriate to employ quantitative genetic

modeling techniques which yield numerical estimates for heritability as well as other important sources of variation (Reich *et al.*, 1975). The plausibility of the multifactorial assumption is discussed below. Finally, multivariate modeling techniques (Heath *et al.*, 1989) can explore the determinants of covariation between related traits, for instance, sexual orientation and sex-dimorphic behavior.

In this study, we explored the determinants of sexual orientation, sex-dimorphic behaviors, and sexual identity using a moderate-sized sample of MZ and DZ twins. Each trait was assessed by self-report both for adulthood and, retrospectively, for childhood. Childhood and adult phenotypes were considered separately because the same trait may have different causes at different ages (Plomin, 1986). We used a model-fitting approach to estimate the relative importance of the additive effects of genes, shared environmental factors, and unique environmental factors. Both univariate and multivariate models were employed to explore variation and covariation among the six measures.

MATERIALS AND METHODS

Subjects

Initially, questionnaires were mailed to 303 pairs of male twins aged 19–40 from the Australian NH & MRC Twin Registry (Martin and Jardine, 1986). Completed questionnaires were returned by 419 individuals (69%), including 162 complete pairs (53% of pairs mailed). Relevant data were unavailable for one pair, thus dropping the final number to 161 pairs. The median age of respondents was 25.

Zygosity was diagnosed via questionnaire items concerning physical similarity and frequency of each cotwin being mistaken for the other. These items have been shown to be over 95% reliable in correctly assigning subjects to the appropriate zygosity (Martin and Martin, 1975).

Measures

The questionnaire completed by twins was developed by McConaghy *et al.* (1979) and contains items relating to subjects' homosexual–heterosexual balance of orientation, sex-dimorphic behaviors, and sexual identity during childhood and adulthood. Items for each of the six relevant domains (homosexual/heterosexual orientation, sex-dimorphic behaviors, and sexual identity for childhood and adulthood) are shown in Table I. The decision regarding which items belong to which domains was made primarily on a priori, rather than on psychometric grounds,

Table I. Items and Scales of Opposite Sex-Dimorphic Behaviors, Sexual Identity, and Sexual Feelings

I. Sex-Dimorphic Behaviors
  Childhood (CB)[a]
    1. Preferred to play with girls (until age 7).
    2. Enjoyed outdoor games (until age 7). (Negative)
    3. Preferred rough to quiet games (until age 7). (Negative)
    4. Enjoyed outdoor sport (8 to 13). (Negative)
    5. Enjoyed playing with dolls (8 to 13).
    6. Peferred contact sports (8 to 13). (Negative)
    7. Avoided physical harm in play (until 10).
    8. Was a loner (until 13).
    9. Was accused of being a sissy (until 13).
  Adult (AB)
    1. Others' perception of subject's gender-atypical behavior.
    2. Subject's perception of subject's gender atypical behavior.
II. Sexual identity
  Childhood (CI)
    1. Ever wished to be member of opposite sex. (6 to 12)
  Adult (AI)
    1. Feel that part of self is female.
    2. Uncertain of gender identity.
    3. Certainty of male gender identity. (Negative)
    4. Feelings of female gender identity.
III. Sexual Feelings
  Childhood/Adolescence (CO)
    1. Crushes on older males. (before 16)
    2. Sex attraction to males. (before 15)
  Adult (AO)
    1. Sex attraction to males now.
    2. Sex fantasies with males now.
    3. Sex contact with males now.

[a] Letters in parentheses are the acronyms by which variables are referred to in some later tables and analyses.

based on their commonly accepted definitions. There is a reasonable consensus among sex researchers on the desirability for distinguishing among sexual orientation, sex-typed behavior, and sexual identity and on the content of each domain (e.g., Money and Ehrhardt, 1972).

After the items were divided into scales, a list of the items was given to two professors of psychology and one advanced graduate student in the field of personality, along with definitions of the three constructs. These individuals were asked to categorize items according to the construct they best represented. On average, only 1.3 items were placed differently from the a priori categorization of Table 1. This strongly suggests that our assignment of items to scales is reasonable, given the definitions of our constructs.

Unfortunately, little is known about the reliability of the scales and the items used in the present study. In one study using these measures, McConaghy *et al.* (1979) found significant associations among homosexual feelings, female sex-dimorphic behavior, and sexual identity. Similar measures also appear to have some validity, as they reliably distinguish among heterosexual, homosexual, and transsexual subjects (e.g., Blanchard *et al.*, 1983). However, the reliability of even well-established scales has not been systematically investigated.

The three Adult Sexual Orientation items were answered with percentages. The items concern same-sex sexual fantasies, attraction, and contact, which are defining features of a homosexual orientation and highly intercorrelated. The overall raw score was obtained by averaging the three percentages. Table II contains the joint frequency distribution (Twin 1 versus Twin 2) of these categories by zygosity. The Adult Sexual Orientation categories were constructed to correspond roughly to the Kinsey scale (Kinsey and Pomeroy, 1948), with the exception that our

Table II. Joint Frequency Distributions for Cotwins' Sexual Orientation[a,b]

	Twin 2					
Twin 1	1	2	3	4	5	6
1	64	9	0	1	0	2
	46	8	0	0	0	0
2	7	2	1	0	0	1
	4	2	0	1	0	0
3	3	0	0	0	0	0
	0	1	0	0	0	0
4	0	0	0	1	0	1
	0	0	0	0	0	0
5	0	0	0	0	0	0
	0	0	0	0	0	0
6	1	0	0	0	1	1
	1	0	0	0	0	0
Total for individuals	151	22	4	4	1	8
	105	18	1	1	0	1

[a] The higher entry in each cell is that for MZ twins; the lower entry is for DZ twins.

[b] Raw Adult Sexual Orientation scores were formed by averaging the three adult sexual orientation items (see Table I). To prepare the data for PRELIS (Jöreskog and Sörbom, 1986), the raw scores were grouped as follows to form six ordinal categories: Category 1, raw = 0; Category 2, $0 < \text{raw} \leq 20$; Category 3; $20 < \text{raw} \leq 40$; Category 4, $40 < \text{raw} \leq 60$; Category 5, $60 < \text{raw} \leq 80$; Category 6, raw > 80. These categories correspond roughly to the Kinsey scale of sexual orientation (Kinsey *et al.*, 1948), with the exceptoin that our scale has one fewer category.

scale has one fewer category (thus, our category "1" corresponds to a Kinsey "0"). There are proportionately more MZ than DZ twins in categories representing a more homosexual orientation. The mean category differs significantly by zygosity ($t = 2.03$, $p < .05$). Similarly, the number of subjects in the highest three categories is significantly greater for MZ twins than for DZ twins [13/190 versus 2/126; $\chi^2(1) = 4.63$; $p < .05$]. Possible causes and implications of this difference are considered below. In none of the other variables (including Childhood Sexual Orientation) did either mean differences or differences in the frequencies in the highest categories, by zygosity, approach significance.

For each of the other three groups of items, all items were first standardized, then summed. Raw total scores were similarly transformed to a smaller number of ordinal categories. For these variables, the principles guiding the transformations were somewhat more arbitrary. The primary concern was that each category contain several cases. Thus, Childhood Sex-Dimorphic Behaviors has nine categories; Adult Sex-Dimorphic Behaviors, four; Childhood Sexual Identity, five; Adult Sexual Identity, five; and Childhood Sexual Orientation, five. Further exploration suggested that magnitudes of the correlations would not change substantially if categories were constructed differently.

The product–moment correlations between age and the scales of interest were low and nonsignificant, with the exception of Childhood Sex-Dimorphic Behaviors, which correlated .16 with age ($p < .05$). Because no plausible explanation exists for such a correlation, and because the correlation is trivial in magnitude, age was not used as a covariate in the analyses reported below. The fact that age did not appear to be an important determinant suggests that cohort effects did not contribute appreciably to intrapair similarity.

Analyses

An assumption fundamental to the analyses reported below is that the causes of the attributes of interest are multifactorial; that is, numerous causes of small effect sum to produce the phenotype. One consequence of the multifactorial model is that the underlying distribution of "scores" on the latent trait is quasi-normal. If so, the distribution of observed scores, which is skewed for all the variables, necessarily results from a nonlinear transformation of "scores" on the latent trait. One solution to the problem of how to analyze scores on the unobservable latent trait is to use polychoric correlations, which assume that the two variables being correlated reflect latent variables (liabilities) which have a bivariate normal distribution (Boomsma *et al.*, 1989). Therefore, the polychoric cor-

relations calculated by the computer program PRELIS, together with their asymptotic covariance matrices, served as the input for the associated structural modeling program LISREL 7.16. Model fitting was by weighted least squares, using the inverses of the asymptotic covariance matrices as weights (Jöreskog and Sörbom, 1988). An extensive discussion of the use of LISREL for analyzing twin data is given in Martin *et al.* (1989).

Univariate analyses were performed for each of the six variables of interest. Each analysis used as input the polychoric correlation between Twin A and Twin B for the respective trait, computed separately for MZ and DZ pairs. The analyses involved a test of four different models employing different combinations of genetic and environmental parameters: the full model (Model 1) contains additive genetic, shared environmental, and unique environmental parameters. Model 2 omits the additive genetic parameter from the full model. Model 3 omits the shared environmental parameter. Model 4 omits both the additive genetic and shared environmental parameters. The unique environmental parameter, which also represents measurement error, cannot be set to zero. All the models assume that for MZ twins, additive genetic effects are correlated 1.0 between pairs, shared environmental effects are correlated 1.0, and unique environmental effects are correlated .0. For DZ twins, the respective correlations are .5, 1.0, and .0.

Additive genetic variance results in the MZ correlation exceeding the DZ correlation; if all covariation between twins is additive genetic in origin, then the MZ correlation will be exactly double the DZ correlation. Shared environmental variance raises both the MZ and the DZ correlations to the same extent; if covariation between twins is due solely to shared environmental influences, both correlations should be equal and greater than zero. If there are both additive genetic and shared environmental influences, then both correlations will be positive and the correlation for DZ twins will exceed half that for MZ twins. Unique environmental variance causes cotwins to be dissimilar to each other for both MZ and DZ pairs; if all variance were due to unique environmental factors, both correlations would be zero.

LISREL yields both estimates for parameters in the model, and a χ^2 test for the goodness of fit of the model. Provided that the MZ correlation does not exceed twice the DZ correlation, the full model will yield a perfect fit, with zero degrees of freedom. Each of the other models can be compared against the full model to see if the omission of the parameter of interest significantly worsens fit. A significant worsening of fit occurs, providing evidence for the importance of that parameter (Neale *et al.,* 1989).

Several multivariate models were also fitted. Such models use as

their input not only the correlations between Twin A and Twin B for the same variable, but also the correlations among variables within each twin, and the correlation between different variables across cotwins (e.g., the correlation of Adult Sexual Orientation of Twin A with Childhood Sexual Orientation of Twin B). Multivariate models are useful for examining the causes of the covariation between traits. The general multivariate model examined here is represented by the path diagram in Fig. 1 and has been called the "independent pathway model" (Heath *et al.*, 1989). The model specifies that there are additive genetic, shared environmental, and unique environmental factors (AG, CG, and EG, respectively) which are common to all six variables. Additionally, it is hypothesized that there may be genetic, shared environmental, and unique environmental determinants which are specific to each variable (A_i, C_i, and E_i; $i = 1$ to 6). The paths representing specific, unique environmental influences also reflect measurement of error. As in the univariate

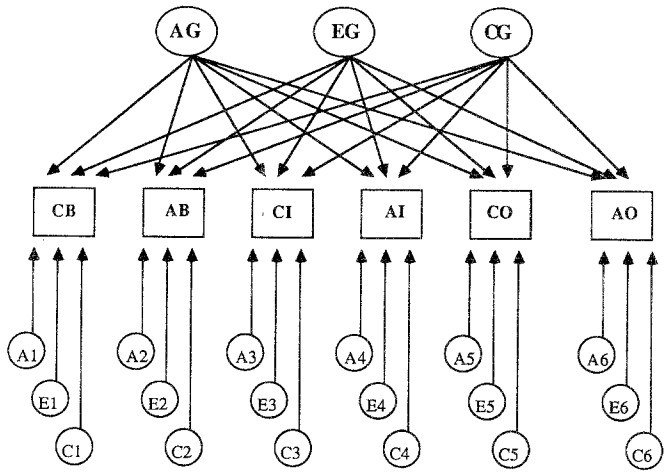

Fig. 1. Full path model for the determinants of Childhood Sex-Dimorphic Behaviors (CB), Adult Sex-Dimorphic Behaviors (AB), Childhood Sexual Identity (CI), Adult Sexual Indentity (AI), Childhood Sexual Orientation (CO), and Adult Sexual Orientation (AO). AG, EG, and CG are the general genetic, unique environmental, and shared environmental factors respectively. A1, E1, and C1 are the specific genetic, unique environmental, and shared environmental influences, respectively, for the first trait (CB), and so on. To simplify, only variables for the first twin from a pair are represented. The following relationships between Twin 1 and Twin 2 are assumed: genetic influences (A's) correlate 1.0 between members of MZ pairs and .5 for DZ pairs. Shared environmental influences (C's) correlate 1.0 between cotwins. Unique environmental influences (E's) are uncorrelated between Twin 1 and Twin 2.

case, the importance of individual parameters (or sets of parameters) can be tested by the decrement in fit caused by their omission.

RESULTS

Univariate Analyses

Table III contains the polychoric correlations and standard errors used in the univariate analyses. The largest differences between the MZ and the DZ correlations favored the MZ correlations (i.e., Childhood Sexual Orientation, Adult Sex-Dimorphic Behaviors); however, for two of the variables (Childhood and Adult Sexual Identity) the DZ correlation exceeded the MZ correlation. There were some differences in the pattern of correlations between adult and childhood versions of the same trait. For instance, the MZ–DZ difference for Childhood Sexual Orientation was .365; for Adult Sexual Orientation it was only .072. The pattern for Adult Orientation suggested that shared environment was most important, while the pattern for Childhood Orientation suggested that genetic variance (perhaps including dominance) was more important. The difference in patterns might reflect either real differences in the determinants of the traits or mere sampling variation in the correlations. Results from multivariate analyses support the latter explanation (see below). However, one must be careful not to overinterpret such differences, since the standard errors of the polychoric correlations are appreciable.

Table IV presents the parameter estimates and significance tests for the four models fitted to each variable. For all scales except for Childhood Sexual Identity (for which both MZ and DZ correlations were quite low), the model in which both additive genetic and shared environmental effects were assumed to be zero could be rejected. At least one of the genetic or shared environmental parameters was necessary to explain the

Table III. Polychoric Correlations (±SE) for MZ and DZ Twins[a]

Trait	N_{MZ}	r_{MZ}	N_{DZ}	r_{DZ}
Childhood Sex-Dimorphic Behaviors	97	.623 ± .059	63	.409 ± .103
Adult Sex-Dimorphic Behaviors	98	.687 ± .113	63	−.049 ± .267
Childhood Sexual Identity	97	−.014 ± .209	63	.117 ± .265
Adult Sexual Identity	96	.230 ± .151	63	.341 ± .176
Childhood Sexual Orientation	96	.492 ± .124	63	.127 ± .192
Adult Sexual Orientation	95	.533 ± .133	63	.461 ± .203

[a] N varies for MZ twins because relevant data are missing for certain twins.

Table IV. Univariate Model Fitting: Parameter Estimates and Statistical Tests

Scale	Model	A	E	C	df	χ^2	*p*
CB	Full	.65	.61	.44	0	.00	1.00
	A=0	.00	.66	.75	1	2.70	.10
	C=0	.80	.60	.00	1	.72	.40
	A, C=0	.00	1.00	.00	2	105.10	<.01
AB	Full	.81	.59	.00[a]	0	2.02	1.00
	A=0	.00	.66	.75	1	6.20	.01
	C=0	.81	.59	.00	1	2.02	.16
	A, C=0	.00	1.00	.00	2	35.21	<.01
CI	Full	.00[a]	.98	.19	0	.15	1.00
	A=0	.00	.98	.19	1	.15	.70
	C=0	.14	.99	.00	1	.19	.67
	A, C=0	.00	1.00	.00	2	.43	.81
AI	Full	.00[a]	.85	.53	0	.22	1.00
	A=0	.00	.85	.53	1	.22	.64
	C=0	.55	.84	.00	1	1.33	.25
	A, C=0	.00	1.00	.00	2	94.82	<.01
CO	Full	.69	.73	.00[a]	0	.34	1.00
	A=0	.00	.79	.62	1	2.46	.12
	C=0	.68	.73	.00	1	.34	.56
	A, C=0	.00	1.00	.00	2	14.93	<.01
AO	Full	.38	.68	.62	0	.00	1.00
	A=0	.00	.70	.72	1	.08	.78
	C=0	.76	.66	.00	1	.77	.38
	A, C=0	.00	1.00	.00	2	19.53	<.01

[a] Parameter on lower bound.

correlations. That is, the causes of five of the six traits appeared to have a familial component.

However, there was, in general, insufficient power to resolve the nature of familiality in favor of genes or shared environment. For only one of the six cases did the omission of a single parameter significantly decrease fit: the additive genetic effect on Adult Sex-Dimorphic Behaviors. Taking the MZ and DZ correlations for Adult Sex-Dimorphic Behaviors at face value, the pattern was inconsistent with an additive genetic hypothesis, since the MZ correlation was quite high, and the DZ correlation essentially zero. However, given the small sample size, particularly for the DZ twins, we do not propose to argue strongly for nonadditive genetic effects such as dominance or epistasis. For all other scales, neither shared environment nor additive genetic factors could be determined separately to be present.

Multivariate Analyses

Table V contains the matrices of polychoric correlations used for the multivariate analyses. One noteworthy aspect of Table V concerns the relatively large correlations between traits in childhood and, respectively, traits in adulthood. The average correlation between the same trait in adulthood and childhood is .605 for DZ twins and .615 for MZ twins.

Table VI presents the results of both overall tests of multivariate models and hierarchical model comparisons. The full model (represented in Fig. 1 above) yielded an acceptable fit to the data. Omitting the general additive genetic factor (Model 2) significantly worsened the fit [$\chi^2(6) = 16.2$, $p < .05$], indicating that there is genetic covariation among the traits. In contrast, omitting the general shared environmental factor (Model 3) did not significantly affect the fit [$\chi^2(6) = 7.5$]. Eliminating the general unique environmental factor (Model 4) caused a large decrement in fit [$\chi^2(6) = 80.2$, $p < .01$].

When the specific genetic and shared environmental variances were eliminated jointly (Model 5), the decrement in fit was not significant [$\chi^2(12) = 17.2$]. That is, the correlation matrices were adequately explained by additive genetic, shared environmental, and unique environmental factors common to all six traits, and unique environmental factors specific to each trait. It should be noted that the test for specific genetic and shared environmental factors is not very powerful, since information

Table V. Within-Individual and Cross-Pair Correlations for Adult and Childhood Sex-Dimorphic Behaviors, Sexual Identity, and Sexual Orientation[a]

	CB_1	AB_1	CI_1	AI_1	CO_1	AO_1	CB_2	AB_2	CI_2	AI_2	CO_2	AO_2
CB_1	1.000	.512	.379	.267	.206	.348	.409	.156	.067	.112	.286	.395
AB_1	.458	1.000	.625	.725	.508	.820	.000	−.049	−.862	.470	.136	.240
CI_1	.397	.236	1.000	.796	.402	.599	.178	.062	.117	.208	.110	.372
AI_1	.324	.583	.546	1.000	.559	.647	.129	−.064	−.068	.341	.222	.521
CO_1	.511	.571	.064	.430	1.000	.651	.045	.332	.348	.326	.125	.343
AO_1	.532	.672	.288	.529	.779	1.000	.125	−.191	−.147	.396	.218	.461
CB_2	.623	.225	.130	.209	.427	.282	1.000	.166	.600	.408	.433	.544
AB_2	.226	.682	−.288	.129	.265	.352	.358	1.000	.650	.461	.014	.208
CI_2	.094	.164	−.024	.118	.106	.097	.355	.550	1.000	.714	.317	.297
AI_2	.224	.333	−.157	.224	.261	.304	.370	.587	.715	1.000	.027	.256
CO_2	.244	.513	.107	.152	.491	.485	.155	.471	.252	.226	1.000	.805
AO_2	.232	.364	−.102	.125	.425	.521	.416	.564	.211	.428	.835	1.000

[a] Correlations for MZ twins are below the diagonal; DZ correlations are above the diagonal. B, I, and O refer to Sex-Dimorphic Behaviors, Sexual Identity, and Sexual Orientation, respectively. C and A refer to childhood and adulthood, respectively. The numerical subscripts, 1 and 2, refer to the scores of the First and Second cotwin, respectively.

Table VI. Overall and Hierarchical Tests of Multivariate Models

Model	Overall test of the model			Hierarchical model tests			
	df	χ^2	p	Benchmark	df	χ^2	p
1. Full model	102	86.2	>.90	—	—	—	—
2. Omitting AG	108	102.4	>.90	Model 1	6	16.2	<.05
3. Omitting CG	108	93.8	>.90	Model 1	6	7.5	>.10
4. Omitting EG	108	178.0	<.01	Model 1	6	80.2	<.01
5. Omitting specific A_i's and C_i's	114	103.4	>.90	Model 1	12	17.2	>.10
6. Common pathway model	123	214.5	<.01	Model 5	9	111.1	<.01
7. Omitting specific A_i's and C_i's, also omitting general A (AG)	120	157.0	<.20	Model 5	6	53.6	<.01
8. Omitting specific A_i's and C_i's, also omitting general C (CG)	120	147.0	<.30	Model 5	6	43.6	<.01
9. Omitting specific A_i's and C_i's, also omitting general E (EG)	120	210.8	<.01	Model 5	6	107.6	<.01

about their importance comes from only two numbers per trait, i.e., the cross twin correlation for the respective trait, for MZ and DZ twins, respectively. Lack of power notwithstanding, this reduced model served as the benchmark against which further models were tested.

Support for theoretical distinctions between the phenotypic variables would exist if it could be shown that their causal architecture differed. In order to see if such differences existed, we tested the "common pathway model" (Heath *et al.,* 1989), which specifies that covariation between traits is due to the influence of one latent phenotype. The general genetic, shared environmental, and unique environmental factors affect the observed variables through the latent phenotype only. In this model, the relative importance of the general factors, AG, AE, and AC, is invariant across measured phenotypes. Accordingly, if the model is rejected, this implies that the relative importance of these factors differs among the variables. One important advantage of this approach is that the common pathway model allows for the fact that measurement error may vary among the scales. When we fitted the common pathway model (Model 6), we found a highly significant decrement in fit compared to Model 5 [$\chi^2(9) = 111.1, p < .01$], providing evidence for the discriminant validity of the traits.

Model 7 was obtained by eliminating the general genetic factor from Model 5. For that model, the fit worsened significantly [$\chi^2(6) = 53.6$, $p < .01$]. Thus, there appear to be additive genetic influences common to the six traits. Similarly, when Model 8 was generated by eliminating the general shared environmental factor from Model 5, there was also a significant decrement in fit [$\chi^2(6) = 43.6$, $p < .01$]. Eliminating the general unique environmental factor from Model 5 (Model 9) caused an even greater worsening in fit [$\chi^2(6) = 107.6$, $p < .01$]. Hence Model 5 could not be plausibly simplified without worsening the fit significantly.

Table 7 contains the path coefficients for Model 5. The pattern of loadings on the three general factors varied markedly across the six variables. For instance, the general additive genetic factor appeared to be much more important than either of the environmental factors for Adult Sexual Orientation. In contrast, for Childhood Sex-Dimorphic Behaviors the general shared environmental factor appeared most important. One must be careful not to overinterpret such differences, given the instability of parameter estimates. However, the difference across traits in the relative importance of AG, EG, and CG was apparently not due merely to sampling error. When loadings on the three general factors were set to be equal within each variable (i.e., so that loadings could vary only across variables), the fit worsened considerably [$\chi^2(12) = 110.0, p <$

Table VII. Path Coefficients for Model 5[a]

Scale	Latent variable			
	AG	EG	CG	E_i
CB	.32	.26*	.68*	.61
AB	.55*	.51*	.04	.65
CI	.01	.98*	.20	.09
AI	.31*	.66*	.12	.67
CO	.74*	.24*	.09	.62
AO	.82*	.38*	.10	.41

[a] Values represent the estimated path coefficients from the latent variables to the phenotypic (scale) scores for Model 5. (See text for description.) AG, EG, and CG represent the common additive genetic, unique environmental, and shared environmental factors, respectively. The E_i are the specific unique environmental factors for each variable (i = 1 to 6).

* $p < .01$. Significance was determined by setting the relevant path equal to zero and testing whether the fit worsened significantly. The specific environmental paths were not tested for significance.

.01]. This finding suggests that one can take seriously at least the largest differences in the patterns of factor loadings. As differences in the importance of the general additive genetic versus the shared environmental factor are of interest, a similar test was performed in which the loadings on these two factors were set equal within each trait. This decrement in fit was also significant [$\chi^2(6) = 45.0$, $p < .01$], indicating that some reliable differences in the relative importance of genes and shared environmental common to all six variables do exist.

There appeared to be some dramatic shifts in the determinants of the same trait from childhood to adulthood. For instance, the general environmental factor was quite important for Childhood Sex-Dimorphic Behaviors, but was unimportant for Adult Sex-Dimorphic Behaviors. When we forced corresponding loadings on the general factors to be equal for childhood and adult phenotypes, there was an appreciable decrease in fit [$\chi^2(9) = 110.0$, $p < .01$]. However, the interpretation of developmental differences is complicated by the fact that measurement error probably differed between variables representing the same trait at different times. Thus, it was not clear whether the decrement in fit reflected, in part, actual differences in the importance of the general factors at different stages of development.

DISCUSSION

Before considering the implications of our study, we discuss its limitations, actual and possible.

One obvious concern is the higher rate of homosexual feelings among MZ than DZ twins. The significance which one attaches to this fact hinges on one's explanation of why it occurred. If some process caused DZ twin pairs concordant for a homosexual orientation differentially to refuse cooperation, this would lead to inflated genetic parameter estimates. The opposite would occur if homosexually oriented DZ twins with heterosexually oriented cotwins were less likely to cooperate. In this case, shared environmental variance would be overestimated. Both on a priori grounds and on the basis of past research, we believe that the latter explanation is more likely. Lykken *et al.* (1987) suggested than an important factor in twin cooperation is intrapair similarity; similar twins cooperate more readily than dissimilar twins. This was proposed to explain why DZ twins tend to be underrepresented in volunteer twin samples, a phenomenon which also occurs in the present study. The inhibiting effect of intrapair dissimilarity may be particularly important for socially salient traits such as sexual orientation. If it has occurred here, then the heritability estimates are probably too low, and the estimates for shared environment too high (but see Martin and Wilson, 1982).

Even if this explanation is correct, the extent to which results are biased remains an open question. There is, in fact, variation in Adult Sexual Orientation among DZ twins. Specifically, 21 of the 126 DZ twins are above the lowest category, a rate which does not differ significantly from that for MZ twins [39 of 190; $\chi^2(1) = .73$]; most (18) of these DZ twins are at the second lowest category. Thus, how seriously one views the MZ–DZ difference in Adult Sexual Orientation depends on whether one accepts the assumption that the lower categories of homosexual feelings are merely quantitatively "milder" than the higher levels.

Another possibility is that MZ twins may, in fact, be more likely than DZ twins to have homosexual feelings. McConaghy and Blaszczynski (1980) noted that MZ twins appear to have a more stressful prenatal environment than DZ twins, as evidenced by their higher incidence of malformation and fetal death and by the fact that up to 30% of monochorionic twins suffer from the twin transfusion syndrome. Stress hormones are known to alter the synthesis and release of androgens, and in some nonhuman animals prenatally stressed males exhibit sexual behavior typical of females (Ward, 1984). One final possibility which might explain a higher rate of homosexual feelings among MZ twins is an imitation (Carey, 1986) or cooperation (Eaves, 1976) effect, in which twins influence each other to become more similar. In this case, MZ twins would show more variance than DZ twins, and hence, there would be more MZ than DZ twins who cross the threshold for homosexuality.

A second possible concern regards the assumption of multifactorial causation which underlies quantitative genetic modeling (Plomin *et al.*, 1989); however, the values of the correlations used by the model (*a fortiori*, the resulting parameter estimates) are not heavily dependent on the accuracy of this assumption, provided that gene action is additive in nature. The multifactorial assumption has been frequently employed in the study of psychopathology, where diagnosis is dichotomous (Reich *et al.*, 1975). Although multifactorial causation is not universal, it is the single most reasonable possibility for complex traits (Plomin *et al.*, 1989). Although a common major gene for homosexuality cannot be ruled out—indeed, such a gene has been hypothesized (Schlegel, 1983)—the search for major genes for complex behavioral traits has not yet challenged the polygenic, multifactorial model.

A third methodological concern regards our decision to analyze scales rather than items. Heath and Martin (1990) have demonstrated that this approach can be misleading, as items comprising one scale can differ radically in their genetic and environmental structures. They recommend that items be analyzed using multivariate genetic techniques before they are summed to form scales. Although there is demonstrated merit to this approach, the required sample size is perhaps an order of magnitude larger than the current sample. Furthermore, the content of the items within each scale used in the present study seems, on the whole, rather homogeneous; this contrasts with the Eysencks' psychoticism scale, on which Heath and Martin performed their analyses. We hope that a future twin sample of sexual orientation will be large enough to allow multivariate genetic analyses of items.

Finally, twin studies are relatively weak designs for detecting nonadditive genetic effects such as dominance (Grayson, 1989). Nonadditive genetic variance is often found for traits with evolutionary significance (Falconer, 1981). One might reasonably hypothesize that sexual orientation is such a trait. Further exploration of this issue must await data from different kinds of relatives.

Assuming that our results are trustworthy, what do they tell us? What one takes from this study depends somewhat on whether one considers the univariate or the multivariate analyses. With one exception, the univariate analyses provided evidence for familiality, but they were generally unable to resolve the causes of the familiality. In contrast, multivariate analyses provided evidence for the importance of both genetic and shared environmental factors.

In contrast to the univariate analyses, which yielded quite different parameter estimates for Adult and Childhood Sexual Orientation, the multivariate analyses gave similar estimates (see Table VII). Both ap-

peared to be highly influenced by the general additive genetic factor, and much less so by the general shared environmental factor. This finding supports the possibility that differences between the univariate parameter estimates for Childhood and Adult Sexual Orientation were due to sampling error in the relevant correlations. Because parameter estimates in multivariate analyses depend, roughly speaking, on averages of correlations, sampling error will tend to be less than in univariate procedures. This difference may help explain why the multivariate analyses yielded evidence for both genetic and shared environmental factors where the univariate analyses had failed to distinguish between the two. We restrict the remainder of our discussion to the results of the multivariate analyses.

Our results suggested that over half the variance in Adult Sexual Orientation ($.82^2$) was attributable to the additive effects of genes. Only about 1% ($.10^2$) of the variance was attributable to the influence of environmental factors shared by siblings. This supports a genetic interpretation of the familiality found by Pillard and Weinrich (1986).

In contrast, almost half the variance in Childhood Sex-Dimorphic Behaviors ($.68^2$) was attributed to the general shared environmental factor, compared to only about 10% ($.32^2$) attributed to the general additive genetic factor. This finding is compatible with the notion that sex roles are products of social expectations which can vary between families. It is less compatible with the evidence that extremely effeminate boys usually develop a homosexual orientation (Green, 1987). Indeed, since many homosexual adult males deny a history of childhood effeminacy, one might hypothesize that childhood effeminacy is a more extreme manifestation of the prehomosexual phenotype. If so, one would expect childhood effeminacy to show a pattern of determinants similar to that for sexual orientation. It is possible that the items comprising our Childhood Sex-Dimorphic Behaviors scale do not adequately measure the "Sissy Boy Syndrome" (Green, 1987). Perhaps, for example, nonathleticism can result both from effeminacy related to a homosexual orientation and from aspects of rearing which are unrelated to sexual orientation. For instance, parents might have considerable influence over whether their children prefer rough-and-tumble to noncontact sports. One would expect that effeminate children would tend to have the same preference, although for reasons quite independent of parental influence. Thus, such a preference would be a poor guide to the effeminacy described by Green (1987) and to subsequent homosexuality. Moreover, it remains controversial whether extreme opposite sex-dimorphic behaviors are on a continuum with or are categorically distinct from the "milder" cross sex-dimorphic behaviors reported by more typical subjects (Constantinople,

1979). Further investigation of this possibility will require more elaborate specification of the core traits of the Sissy Boy Syndrome.

Sexual identity was the trait least influenced by factors operating between families. For Childhood Sexual Identity, the vast majority of the variance ($.98^2$) was accounted for by the general unique environmental factor. Although within subjects Childhood Sexual Identity was highly correlated with other variables, it was essentially uncorrelated across twins of the same pairs. Although the role of genes appeared larger for Adult Sexual Identity, the unique environmental factor was still the most important of the general factors. Since psychodynamic formulations generally stress the importance of events constituting the unique environment, Gender Identity may be the trait most conducive to such explanations.

The only developmental trend common to all three traits was an increase in the importance of genetic factors. Although one must keep in mind the aforementioned interpretive difficulties stemming from differences in measurement error between scales, this difference cannot account for the apparent increase in the importance of genes. For only one trait, sexual orientation, is the specific unique environmental path larger for adulthood than childhood. Fixing the genetic paths to be equal across the two stages of respective traits produced a significant decrease in fit [$\chi^2(3) = 7.9, p < .05$]. However, because this test was both post hoc and barely significant, it should be regarded cautiously. Furthermore, it is important to remember that because childhood measures are retrospective, the possibility of systematic bias is appreciable. This problem is not unique to the present study—retrospective measures are commonly used in the study of precursors of adult homosexuality (e.g., Grellert *et al.,* 1982; Harry, 1983).

Causes of individual differences in sexual orientation are particularly interesting, because of the low a priori likelihood of a significant genetic contribution to a trait with such low relative fitness. Bell *et al.* (1981), for instance, reported that a sample of homosexuals had roughly one-fifth the number of children (via marriage) compared to heterosexuals. Because of the evolutionary difficulties associated with a genetic explanation of individual differences in sexual orientation, Dörner and coworkers' (1983) hypothesis that human male homosexuality may arise from maternal stress during gestation has seemed intriguing. That hypothesis provides an environmental model which might account for the incomplete brain masculinization hypothesized by the neurohormonal theory (Ellis and Ames, 1987). It could also account for high twin correlations, since cotwins share many aspects of the prenatal environment. However,

the maternal stress hypothesis implies that MZ and DZ cotwins should be equally similar for sexual orientation. Indeed, the respective correlations obtained in the present study for adult sexual orientation, .53 and .46, are not inconsistent with this expectation (although the reader is again warned of their large standard errors). However, the multivariate model-fitting provided evidence for substantial genetic influence on sexual orientation. Because of the restrictive assumptions of the relevant multivariate models, we do not claim convincingly to have demonstrated the existence of genetic variance for sexual orientation. For an extended discussion of possible mechanisms for the maintenance of genetic variation in sexual orientation, see Ruse (1988).

In conclusion, our study represents the first attempt to analyze the determinants of sexual orientation and its correlates using biometrical data-analytic techniques and twin data. We believe that the results we obtained in this modest-size study justify investigations using larger samples and a variety of kinships. Given the implications of past investigations and our own findings, such research is long overdue.

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