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A Test of the Maternal Stress Theory of Human Male Homosexuality

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Both the neurohormonal theory of sexual orientation and previous research on humans and animals suggest that male homosexuality may arise from prenatal stress during the brain's sexual differentiation. Stress-proneness and retrospective reports of stress during pregnancy were obtained from mothers of male and female heterosexuals, bisexuals, and homosexuals. Each mother also rated pregnancy stress for a heterosexual sibling of the subject. For males, neither between-family nor within-family analyses revealed a maternal stress effect for either sexual orientation or childhood gender nonconformity. However, mothers of effeminate children reported more stress-proneness than other mothers. Male homosexuality nevertheless was strongly familial, suggesting a reconsideration of genetic and familial environmental mechanisms.

KEY WORDS: maternal stress; sexual orientation; etiology; homosexuality; familiality.

INTRODUCTION

A growing body of evidence implicates a biological, and particularly a neurohormonal, role for the etiology of human male homosexuality (Ellis and Ames, 1987). The hypothesized etiological mechanism is a relative insufficiency of prenatal testosterone during the brains's sexual differentiation in future male homosexuals.

Evidence for a neurohormonal theory includes findings that behavioral antecedents of adult homosexuality often arise early in development

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(Green, 1987); homosexual-like behavior in lower animals can be induced by manipulating androgen production at a critical period (Dörner and Hinz, 1968; Ward and Renz, 1972; Baum, 1976; Phoenix, 1974; Ward, 1972, 1984); endocrine anomalies in humans can affect subsequent psychosexual development or sexual behavior (Money *et al.*, 1984); human male homosexuals may differ physiologically from male heterosexuals in a manner predicted by neurohormonal theory (Gladue *et al.*, 1984; but compare Hendricks *et al.*, 1989, and Gooren, 1986); and finally, psychosocial explanations have not been supported (Bell *et al.*, 1981).

One major weakness of the neurohormonal theory is the lack of evidence concerning mechanisms that might trigger the hypothesized prenatal testosterone insufficiency. The few existing twin studies of male homosexuality are, on balance, consistent with a substantial genetic contribution—monozygotic (MZ) twin pairs tend much more often to be concordant for homosexuality than expected by chance—but the deficiency of dizygotic (DZ) twins in these studies, as well as other methodological defects, makes a genetic interpretation problematic (Rosenthal, 1970). A recent study of two separated monozygotic twin pairs (Eckert *et al.*, 1986) found one pair to be concordant and the other possibly concordant for a homosexual orientation. Pillard and Weinrich (1986) have provided persuasive evidence that male homosexuality is familial, but the family study design does not permit resolution of the effects of genotype and shared environment.

Aside from the weakness of previous research, there are theoretical grounds for doubting high heritability for male homosexuality. Given reduced rates of reproduction of male homosexuals (e.g., Bell *et al.*, 1981), it seems likely that the homosexual phenotype has been strongly selected against. Yet, male homosexuality is appreciably more prevalent than even the highest known mutation rate (Weinrich, 1987). Although there has been speculation that genes for homosexuality might be maintained in the population gene pool via evolutionary processes such as kin selection (e.g., Wilson, 1978), supportive data are lacking.

One animal paradigm has been considered a promising etiological model for human male homosexuality, and its common developmental precursor, childhood effeminacy: maternal exposure to stress during pregnancy (Dörner *et al.*, 1980). Maternal stress in response to environmental provocation has the advantage of circumventing the evolutionary difficulties associated with genetic explanations. Maternal stress demasculinizes and feminizes male sexual behavior in rats (Ward, 1972), apparently by delaying the surge of testosterone needed for sexual differentiation of the brain (Ward and Weisz, 1980).

On the other hand, both inconsistencies in the literature and theoretical concerns necessitate caution in applying the maternal stress model to human male homosexuality. Chapman and Stern (1978) failed to find any effect of prenatal stress on the sexual behavior of male rats. Moore (1989) reported no effects on offspring sexual behavior from crowding stress during pregnancy, which is the most naturalistic stressor investigated so far. Furthermore, the adrenal response to stress in humans is much less dramatic than in animals (Sachar, 1980), suggesting that humans may be less prone to maternal stress effects. Finally, it is not clear that the demasculinized and feminized rats studied by Ward (1972) constitute an appropriate model for human male homosexuality. A more compelling analogy would have found male rats preferring to engage in masculine sexual behavior with other male rats. Indeed, studies of nonhuman animals frequently cited as supporting the neuroendocrine theory of sexual orientation are often of questionable applicability (Adkins-Regan, 1988). Nevertheless, the animal evidence, including the maternal stress studies, serves as a useful heuristic for researchers in human sexuality (Ruse, 1988).

A maternal stress theory would predict relatively high concordance for homosexuality in both MZ and DZ male twins. It is less clear that the maternal stress hypothesis could account for the familiality of male homosexuality in nontwins. One unexamined possibility is that some mothers are particularly stress-prone, and thus, more likely to have homosexual sons.

Dörner *et al.* (1980) surveyed a group of homosexuals born in Germany between 1934 and 1953 and found that a disproportionately high number were born during and just after World War II, presumably a stressful time in Germany. Dörner *et al.* (1983) attempted to assess the incidents of stressful prenatal events in bisexual, homosexual, and heterosexual men. Homosexual and bisexual subjects more frequently reported that their mothers were exposed to stressful situations while pregnant with the subjects. For homosexuals, bisexuals, and heterosexuals, the reported incidences of moderate prenatal stress were 33, 25, and 6%, respectively. The same figures for severe stress were 35, 15, and 0%, respectively. Although this study yielded a very large effect for maternal stress, the probands, rather than their mothers, reported on prenatal stress. Most recently, Ellis *et al.* (1988) contacted mothers of heterosexual, homosexual, and bisexual subjects. Mothers of 39 male homosexuals reported greater stress during the second trimester of pregnancy than mothers of 68 male heterosexuals, a marginally significant ($p < 0.05$, one-tailed test) excess.

Two studies failed to demonstrate a maternal stress effect for human male homosexuality. Schmidt and Clement (1988) found no evidence for

increased homosexual behavior for a West German cohort conceived during the war. Wille *et al.* (1987) failed to find any relationship between psychosocial and physical stress during mothers' pregnancies and the sexual orientation of their sons. However, only 50 sons were interviewed. Given the relatively low base rate of homosexuality, a modest relationship between prenatal stress and sexual orientation might not be detected due to insufficient power.

Although some of the aforementioned work is suggestive of a maternal stress effect for male homosexuality, the evidence is far from conclusive. The possibility that Dörner *et al.*'s (1983) impressive results stem from a response bias renders those findings suspect. Though Ellis *et al.* (1988) avoided this problem, their relatively small sample precluded results that are statistically convincing given the modest effect size obtained. The study reported below attempts to improve methodologically on these prior studies and to extend their scope.

METHOD

Subjects

Subjects were recruited from several sources. Heterosexual, homosexual, and bisexual subjects were enlisted from undergraduate psychology classes. Additionally, homosexual and bisexual subjects were obtained through an advertisement in an alternative news magazine and through several university gay and lesbian organizations. In order to insure that the university and nonuniversity subjects would not differ importantly in terms of age and education, the ad specified that subjects must be under age 30 and have completed at least 1 year of college; however, one or the other requirement was waived several times to increase sample size.

After completing several questionnaires (described below) subjects were shown the questionnaire to be completed by their mothers (see below) and asked for permission to contact their mothers. There was no hint in the questionnaire that the study had to do with sexual orientation. Subjects were encouraged to look at the questionnaire in order to reassure themselves of its innocuous appearance.

Table I contains the frequency distribution of Kinsey Fantasy ratings (Kinsey *et al.*, 1948) for subjects of each sex. Subjects were categorized for sexual orientation on the basis of sexual fantasy alone. Sexual fantasy is more closely linked to the concept of sexual orientation than is sexual behavior (Money, 1987). For instance, two of our homosexual subjects had not had any sexual experience, but had a clear preference for same-sex sex

Table I. Frequency Distribution of Kinsey Fantasy Ratings, by Sex

	All subjects		Mothers responded	
	Males	Female	Males	Female
Kinsey rating	*n*	*n*	*n*	*n*
0	64	49	45	37
1	16	16	11	13
2	4	7	4	3
3	3	6	2	5
4	12	10	9	8
5	70	8	53	5
6	31	1	19	1
Totals	200	97	143	72

partners. For some analyses described below, subjects were divided a priori into four groups: male nonheterosexuals (which includes both homosexuals and bisexuals), male heterosexuals, female nonheterosexuals, and female heterosexuals. The criterion for inclusion among the nonheterosexual groups was a Kinsey fantasy score of 3 or greater, which indicates at least equal sexual interest in the same sex.

The male nonheterosexual subsample consisted of 116 with an average Kinsey Fantasy score of 5.11 (SD = 0.68). Eighty-three of the mothers of members of this group returned their questionnaires, a return rate of 83%. (Of the male nonheterosexual group, 10 refused permission to send questionnaires to their mothers, 3 others were adopted, and 3 mothers were deceased.) The female nonheterosexual subsample contained 25 (mean Kinsey Fantasy rating = 4.16, SD = 0.85). Two female nonheterosexuals were unwilling to give permission for maternal contact. Nineteen mothers responded, a return rate of 83% of mailed questionnaires. The male heterosexual subsample consisted of 84 (mean Kinsey score = 0.29, SD = 0.55); 60 mothers returned questionnaires, a return rate of 71.4%. The female heterosexual subsample contained 72 subjects (mean Kinsey rating = 0.42, SD = 0.67); 53 mothers returned questionnaires for a return rate of 73.6%. The slightly higher return rate by the mothers of nonheterosexual subjects is due to an extra mailing to those mothers only.

Instruments

The following two questionnaires were completed by the subjects and the remaining questionnaires were answered by mothers of subjects.

Behavior Questionnaire

The Behavior Questionnaire assesses whether subjects' childhood behaviors were gender nonconforming. Only childhood gender nonconformity was measured, as male homosexuals' gender nonconformity appears to be greatest in childhood (Harry, 1983). The five items on the questionnaire are adapted from Whitam (1977) and ask whether in childhood the subject (i) was considered a sissy; (ii) was a loner; (iii) wished he had been a girl; (iv) preferred playing with girls; and (v) ever dressed in female clothes. The items were altered appropriately for female subjects. It was decided a priori to construct a scale adding all five items. Responses of "don't know" were treated as intermediate to "yes" and "no." This scale is this study's measure of Self-Rated Childhood Gender Nonconformity (SCGN). The reliability (coefficient alpha) of the scale was 0.63 for males and 0.72 for females.

Relatives Questionnaire

Subjects were asked to give the number of their male and female siblings who were homosexual or bisexual. Pillard and Weinrich (1986) demonstrated that subjects making similar estimations were quite accurate.

Life Events Questionnaire

The Life Events Questionnaire was constructed to measure retrospective reports of stress during mothers' pregnancies. It assesses 28 potentially stressful life events as well as the degree to which each was stressful during the year before the pregnancy and the three trimesters of the pregnancy. The events were chosen from among those listed in the Psychiatric Epidemiological Research Interview (Dohrenwend *et al.*, 1978). Several events were added because of special relevance to pregnant women (e.g., unwanted pregnancy). At the end of the questionnaire, space was provided for mothers to list pertinent events not explicitly listed elsewhere. Mothers were also asked to complete a questionnaire on a sibling of the subject, if available. (Subjects had been asked to give the first name of a heterosexual sibling.)

Each event was to be rated from 0 to 4, for each of four time periods: the year before pregnancy, the first, second, and third trimesters of pregnancy. An additional category was provided for instances in which mothers remembered that a stressful event occurred during the pregnancy, but not the specific trimester in which it occurred. An event was to be rated for

any period in which it caused stress, even if the actual event occurred during an earlier period: 0 indicated no occurrence; 1 indicated occurrence but no stress; 2, 3, or 4 indicated occurrence and mild, moderate, or severe stress, respectively.

For each subject and sibling for whom mothers provided information, 12 stress composites were computed from the three different measures for each of four time periods. The three measures were the number of events rated as severely stressful (4), summed stress, and number of events rated greater than 0. The four periods included the three trimesters of pregnancy and the entire pregnancy. The latter included those events that mothers could not place during a specific trimester.

For the sibling analyses, 12 additional variables were computed, namely, the differences between subjects' and siblings' corresponding stress composites. A positive difference score indicated that a mother rated her pregnancy with the subject as more stressful than that with the sibling. The inclusion of sibling data allowed a more powerful test of the material stress hypothesis because it would control for systematically over- or under-reported stress. This analysis also allowed an additional test of the maternal stress hypothesis; namely, that it should operate within as well as between families.

Personal Views Survey and the EASI-Emotionality Scale

These scales served as measures of maternal stress-proneness. The Personal Views Survey is the short form of the composite measure of Hardiness employed by Kobasa and Puccetti (1983). The personality dimension, Hardiness, has consistently been found to decrease the effect of stressful life events in producing illness symptoms (Kobasa, 1979; Kobasa and Puccetti, 1983; Kobasa *et al.*, 1982). The short Personal Views Survey has a reliability (coefficient alpha) of 0.86, and correlates 0.89 with the longer composite from which it is derived (Kobasa and Maddi, personal communication, 1982).

Because Hardiness may derive its predictive validity from being negatively associated with neuroticism (Funk and Houston, 1987), all subjects were given the Emotionality scale of the Adult EAS Temperament Survey (Buss and Plomin, 1984). This is essentially a neuroticism scale whose three subscales consisting of Distress, Fear, and Anger. Hardiness and emotionality correlated -0.46. High stress-proneness is thus defined as high Emotionality or low Hardiness.

It is important to note that hypotheses regarding maternal stress-proneness concern mothers' stress-proneness during pregnancies; such

pregnancies occurred some 20 years before the personality measures were administered. Although 20-year reliabilities for the Hardiness and EAS Emotionality scales are unavailable, measures of neuroticism typically yield stability coefficients near 0.50 (Conley, 1984).

Mothers Assessment of Subjects' Gender Conformity

Mothers were asked to rate subjects on 11 attributes of childhood personality. Of the 11, only 4 were of interest a priori: masculine, dominant, aggressive, and athletic. These were embedded among the remaining seven traits in order to conceal the nature of the study. Because two additional items (high activity level and extraverted) correlated highly enough with the four primary items to load on the same factor of a principal factor analysis, a 6-item scale was constructed for males. Coefficient alpha was 0.74. For females, no corresponding factor emerged. Therefore, the single maternally rated item "masculine–feminine" was used as a measure of maternally rated gender nonconformity for females. Childhood gender nonconformity was studied along with sexual orientation because neurohormonally induced behavioral effeminacy might occur in the absence of eventual homosexuality.

RESULTS

Relationships Among Orientation and Gender Nonconformity Measures

For males, the correlation between Kinsey Fantasy scores and Self-rated Childhood Gender Nonconformity was 0.53; Kinsey Fantasy correlated 0.39 with Maternally rated Childhood Gender Nonconformity (MCGN). The two measures of Childhood Gender Nonconformity correlated 0.29. For females, these correlations were 0.53, 0.32, and 0.42, respectively. (All correlations were significant at the $p < 0.001$ level.) It should be noted that the overrepresentation of nonheterosexuals in this sample undoubtedly produces larger correlations than would be obtained using a random sample.

Tables for the following analyses contain data for both sexes; however, results are discussed separately for each sex.

Stress and Orientation/Gender Nonconformity

Male

The most commonly endorsed stress item was "moved residence" (endorsed by 49% of mothers), while the least frequently endorsed item was "had a mental illness" (2%). The highest mean stress rating was obtained by the item "death of a friend" (2.83), while the least stressful item was "change in pattern of work or job" (1.90). The relative sizes of these frequencies and mean ratings comport with one's intuition and hence provide some face validity for the stress ratings.

Table II gives the correlations of the 12 stress composites with Kinsey Fantasy, Self-rated Childhood Gender Nonconformity, and Maternally rated Childhood Gender Nonconformity, separately, for male and female. For males, the correlations were uniformly low and nonsignificant. The correlations were at least as likely to be in the negative as in the positive direction. There was no tendency for mothers of males with higher Kinsey Fantasy scores to report greater stress during the relevant pregnancies.

A further analysis tested the hypothesis that the stress pattern across gestation differs between heterosexuals and nonheterosexual males. Three repeated measures ANOVAs were performed, one with each stress composite as the dependent variable, trimesters as Levels, heterosexuals *vs.* nonheterosexual males as Group, and stress during the year before pregnancy as covariate. None yielded a significant Group × Levels interaction.

Within-sibling analyses were uniformly unsupportive of the maternal stress hypothesis. Correlations of Kinsey Fantasy with sibling differences in stress ranged from -0.06 to 0.07, with a median correlation of 0.04. For SCGN, the correlations ranged from -0.05 to 0.01 with a median correlation of -0.03. For MCGN, the correlations ranged from -0.19 ($p < 0.05$; the direction of the correlation is opposite that predicted) to -0.01, with a median of -0.11. Contrary to the maternal stress hypothesis, mothers did not report that homosexually oriented subjects had more stressful gestations than their heterosexual siblings.

Female

In contrast to the results for males, Table II displays a tendency for females with higher Kinsey Fantasy scores to have mothers with higher

Table II. Correlations Between Stress and Orientation/Effeminacy[a]

	Orientation/effeminacy					
	Kinsey fantasy		SCGN[b]		MCGN[c]	
Stress composite	Male	Female	Male	Female	Male	Female
First trimester						
Sum of ratings	–0.06	0.05	–0.04	–0.07	–0.00	0.06
No. events rated > 0	–0.08	0.13	–0.08	–0.01	0.03	0.03
No. events rated 4	0.04	–0.09	0.05	–0.10	–0.00	0.15
Second trimester						
Sum of ratings	–0.02	0.25[d]	–0.02	–0.07	0.03	–0.10
No. events rated > 0	–0.02	0.31[d]	–0.04	–0.03	0.06	–0.05
No. events rated 4	0.11	0.15	0.07	–0.13	–0.01	–0.13
Third trimester						
Sum of ratings	–0.02	0.22	–0.06	–0.06	0.03	–0.08
No. events rated > 0	–0.04	0.31[e]	–0.04	–0.02	0.08	–0.08
No. events rated 4	0.08	–0.01	0.04	–0.12	–0.03	–0.07
Throughout pregnancy						
Sum of ratings	–0.02	0.18	0.01	–0.03	0.04	–0.05
No. events rated > 0	–0.03	0.25[d]	–0.02	–0.00	0.07	–0.04
No. events rated 4	0.09	0.01	0.07	–0.11	0.00	–0.01

[a] All correlations for males are based on a minimum of 141 subjects. For females, the minimum n is 72.
[b] SCGN = self-rated childhood gender nonconformity.
[c] MCGN = maternally rated childhood gender nonconformity.
[d] $p < 0.05$.
[e] $p < 0.01$.

stress ratings. The number of stress events rated above zero was reliably related to Kinsey Fantasy scores for the second and third trimesters, and throughout pregnancy. The sum of stress ratings during the second trimester was also significantly correlated with sexual orientation. The gender nonconformity scales, however, were essentially uncorrelated with mothers' stress ratings. It should be noted that even the significant correlations are quite small.

Repeated measures ANOVAs (with stress during the year before pregnancy as covariate) yielded significant Trimester × Group interactions for both summed stress, $F(1.4, 186) = 3.53$, $p < 0.05$, and the total number of events rated, $F(1.4, 188) = 4.63$, $p < 0.05$. (Degrees of freedom were computed using the Greenhouse-Geisser correction, which controls for the fact that scores from adjacent time intervals are more closely related than scores from more distantly related time intervals. See Winer, 1971). The interactions were due to nonheterosexual females experiencing an increase

Table III. Correlations Between Maternal Stress-Proneness and Orientation/Effeminacy[a]

	Orientation/effeminacy					
	Kinsey fantasy		SCGN[b]		MCGN[c]	
Stress composite	Male	Female	Male	Female	Male	Female
Hardiness[d]	–0.14	0.09	–0.18[e]	–0.00	–0.28[f]	–0.02
EASI-emotionality	0.08	–0.08	0.20[e]	–0.05	0.17[e]	–0.06

[a] All correlations for males are based on a minimum of 139 subjects. For females, the minimum n is 72.
[b] SCGN = self-rated childhood gender nonconformity.
[c] MCGN = maternally rated childhood gender nonconformity.
[d] Hardiness is hypothesized to be maternal stress-resistance, the opposite of stress-proneness.
[e] $p < 0.05$.
[f] $p < 0.01$.

in stress between the first and second trimesters, whereas heterosexual females experienced a decrease.

Within-family analyses yielded weak support for a maternal stress effect for female sexual orientation. Correlations of Kinsey Fantasy with sibling differences in stress ranged from -0.14 to 0.26 ($p < 0.05$), with a median of 0.13. For SCGN, the correlations ranged from -0.14 to 0.11 with a median correlation of 0.04. For MCGN, the correlations ranged from 0.07 to 0.20, with a median of 0.15. The one significant correlation, between Kinsey Fantasy and the number of events rated as extremely stressful during the second trimester, is consistent with a near-significant corresponding rating for the third trimester. Moreover, both between- and within-family analyses are consistent with a small maternal stress effect for nonheterosexual females.

Maternal Stress-Proneness, Orientation, and Gender Nonconformity

Table III contains the univariate correlations between measures of maternal stress-proneness, sexual orientation, and childhood gender nonconformity.

Male

There was a clear preponderance of low, significant correlations in the expected direction for the gender nonconformity measures. Correlations for sexual orientation were also in the expected direction but were

not significant. Tests for interactions between maternal stress-proneness and stress ratings were uniformly nonsignificant.

Female

The correlations were all low and nonsignificant. As with the males, tests for interactions between maternal stress-proneness and stress ratings were uniformly nonsignificant.

Familiality of Nonheterosexuality

Male

Table IV gives the frequencies of known and suspected nonheterosexuality among brothers of subjects. The proportion of brothers known to be nonheterosexual was significantly greater for nonheterosexual males (10%) than for the other groups combined (2%); $\chi^2(1) = 11.3; p < .001$. Including suspected nonheterosexual brothers, the percentages are 21 and 4%, respectively $\chi^2(1) = 21.9$, $p < 0.001$.

Female

Table IV contains the frequencies of known and suspected nonheterosexuality among sisters of subjects. The percentages of known nonheterosexual sisters did not differ between the nonheterosexual females and the other groups combined (4 *vs.* 1.5%, respectively; $\chi^2(1) = 0.88$). However, if suspected nonheterosexual sisters are included the percentages do differ significantly (21 vs. 7%, respectively; $\chi^2(1) = 5.2, p < 0.05$).

DISCUSSION

Contrary to the primary hypothesis of this study, mothers of male nonheterosexuals reported no more stress during their pregnancies than did mothers of heterosexuals. This negative finding contrasts sharply with the highly significant results obtained by Dörner *et al.* (1983). It also contrasts—though not so sharply—with the smaller and marginally significant effect found by Ellis *et al.* (1988).

The present study and the Ellis *et al.* (1988) study were quite similar methodologically, both relying on retrospective maternal reports of prenatal

Table IV. Familiality of Homosexuality and Bisexuality[a]

	NM		HM		NF		HF	
Siblings	*n*	%	*n*	%	*n*	%	*n*	%
Brothers								
Total	143	100	84	100	30	100	60	100
Known gay or bisexual	15	10	0	0	1	3	2	3
Suspected gay or bisexual	15	10	0	0	3	10	1	2
Known or suspected gay or bisexual	30	21	0	0	4	13	3	5
Sisters								
Total	132	100	69	100	24	100	59	100
Known lesbian or bisexual	2	2	0	0	1	4	2	3
Suspected lesbian or bisexual	11	8	0	0	4	17	4	7
Known or suspected lesbian or bisexual	13	10	0	0	5	21	6	10

[a]NM = nonheterosexual males; HM = heterosexual males; NF = nonheterosexual females; HF = heterosexual females.

stress. Even the instruments by which stress was measured were similar. Ellis *et al.* tested several (nonindependent) relevant hypotheses, only one of which was significant, using a one-tailed test. This suggests that the average relevant effect size of that study is somewhat smaller than that yielded by the one significant analysis. Thus, the difference between the results of the two studies is not large.

In contrast, the difference between our results and those of Dörner *et al.* (1983) cannot plausibly be attributed to sampling error. The most important methodological difference between the two studies was the use of maternal instead of proband reports of prenatal stress. Mothers' reports are preferable, as the accuracy of subjects' reports depends not only on mothers' memories but on how much mothers have told the subjects about relevant events. That both studies asking mothers directly yielded much weaker effects than did Dörner *et al.*'s suggests an unspecified reporting bias in Dörner's work. The extent to which Dörner *et al.*'s subjects were familiar with the primary hypothesis of that study or were responding to demand characteristics is not clear.

Our study's inclusion of a within-family component presents both an additional hurdle and an additional opportunity for the maternal stress hypothesis. The fact that this test was also negative lends more confidence in the present conclusions. Results of the present study are inconsistent with a large effect of prenatal maternal stress on subsequent nonheterosexual orientation in males.

Is it possible that the results of all three studies may be accurate? Perhaps severe prenatal stress *can* cause male homosexuality. In times of severe stress—such as World War II in Germany—stress may be a more

common cause. But in contemporary North America, that degree of stress is too rare to be detected in a retrospective study. This explanation could also explain why, despite the negative findings regarding maternal stress reports, mothers of effeminate boys were found to be more stress-prone than mothers of gender-conforming boys. Individual differences in personality appear to be important in determining physiological stress responses in humans (Wolff *et al., 1964; Katz et al.*, 1970). Perhaps when the base rate of events that anyone would find severely stressful is low, personality is the more important determinant of the stress response.

The most obvious problem with this explanation is that stress-prone mothers presumably still require stressful events before secreting stress hormones. Maternal ratings of subjective stress failed to correlate with sexual orientation or childhood gender nonconformity. Furthermore, Ellis (personal communication, 1989) assessed maternal stress susceptibility along with maternal stress, but failed to find any relationship between that measure and offspring's sexual orientation.

Unexpectedly, there were significant relationships for females between maternal stress and sexual orientation. The effect appeared to involve primarily the second and third trimesters of pregnancy, and was found to some extent in within-families analyses as well.

The primary obstacle to taking this finding at face value is the absence of an etiological theory. The maternal stress hypothesis for human male homosexuality seems reasonable because stress hormones impede the production of hormones necessary for male sexual differentiation. No such scenario exists for human female homosexuality. Although some investigators have found that prenatally stressed female rats show abnormalities in reproductive capacity and sexual behavior (Herrenkohl and Politch, 1978; Politch and Herrenkohl, 1984), there have been no reports of increased masculine sexual behavior or preferences for sexual interaction with other females.

In contrast to the generally negative results for prenatal stress as a determinant of sexual orientation in males, the present study did yield evidence of familiality. If one includes suspected nonheterosexuality, male nonheterosexuals reported a 21% rate in their brothers, compared to only 4% in the other groups. These rates are in remarkably good agreement with those reported by Pillard and Weinrich (1986), who found rates of 18 to 22% in brothers of nonheterosexuals (percentages varied depending on the analysis), compared to a 4% rate in brothers of heterosexuals. Furthermore, Pillard and Weinrich actually contacted siblings and found that subjects' estimates of siblings' sexual orientation were generally accurate.

If one assumes that the determinants of sexual orientation are multifactorial (i.e., genetic variance is polygenic, and environmental influences are each of small effect) and hence approximately normally distributed, it is possible to estimate the proportion of variance in sexual orientation accounted for by familial factors (Reich *et al.*, 1975). Assuming a base rate of 4% for nonheterosexuality, a 20% rate in brothers of nonheterosexuals implies the following resolution of familial variance (for details, see Gottesman and Carey, 1983): If the resemblance of brothers in sexual orientation were due solely to shared genes, this correlation would imply a heritability of 0.90, indicating that 90% of the variance in sexual orientation is determined by differences in genotypes. If the resemblance were due solely to brothers' sharing the same relevant environmental factors, the correlation would imply that 45% of the variance could be explained by shared environment. We cannot argue for one or the other interpretation here because the family study design does not allow for separation of genetic and environmental effects, but the magnitude of the effects potentially explainable by familial factors is appreciable. We are suggesting a shift in focus away from idiosyncratic environmental factors (e.g., peculiarities of parent–offspring relationships or prenatal stress), and towards factors shared by siblings (genetic or environmental), as the most productive route to elucidating the etiology of individual differences in male sexual orientation.

Familiality of female nonheterosexuality was murkier. This may have been due in part to the small number of nonheterosexual probands. Because a significant familial effect was found only when including both known and suspected nonheterosexual sisters, the effect is questionable. Indeed, other investigators have been unable clearly to demonstrate familiality of female nonheterosexuality (Weinrich, 1987). A study comparing MZ and DZ twins would be a more powerful method of determining whether female nonheterosexuality is familial.

In conclusion, results of the present study suggest that prenatal stress cannot account for much of the variance in sexual orientation in contemporary America. A more promising route towards elucidating the etiology of male homosexuality is resolving the causes of its familiality.

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