*Archives of Sexual Behavior, Vol. 14, No. 1, 1985*

# Sexual Orientation after Prenatal Exposure to Exogenous Estrogen

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*Thirty women aged 17 to 30 years with documented prenatal exposure to the nonsteroidal synthetic estrogen diethylstilbestrol (DES) were compared to thirty women of similar demographic characteristics from the same medical clinic who had a history of abnormal Pap smear findings. A subsample of the DES women were also compared to their DES-unexposed sisters. Sexual orientation in its multiple components was assessed by systematic semistructured interviews. In comparison to both control groups, the DES women showed increased bisexuality and homosexuality. However, about 75% of the DES women were exclusively or nearly exclusively heterosexual. Nonhormonal and hormonal interpretations of these findings are discussed.*

**KEY WORDS:** homosexuality; diethylstilbestrol; prenatal hormones; sexual orientation.

## INTRODUCTION

Since the majority of heterosexual and homosexual individuals do not show consistent differences in peripheral hormone levels (Meyer-Bahlburg,

This research was supported in part by U.S.P.H.S. research grants MH-34635 (NIMH), Y01-CN-00711 (NCI), and MH-30906 (NIMH).

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0004-0002/85/0200-0057$04.50/0 

1977, 1979, in press), it is unlikely that sexual orientation is related to endocrine differences in adulthood. A potential contribution of prenatal hormones to the development of sexual orientation has not been ruled out and therefore has become a focus of current psychoendocrine research. This approach, which is particularly represented by Dörner (1968, 1976), is derived from research on lower mammals for which, regardless of genetic sex, exposure to sex hormones during certain sensitive periods of early brain development leads to structural changes of the brain and specific shifts in subsequent sex-dimorphic behavior and gonadotropin regulation. Two major pathways seem to be involved (McEwen, 1983): an androgenic one that mainly utilizes testosterone and/or DHT, and an estrogenic one that relies primarily on estradiol derived from testosterone by aromatization at the target organ cellular level. Estradiol is widely available in the fetoplacental unit of the rat; it seems to be rendered relatively inert by binding to $\alpha$-fetoprotein and has, therefore, effects on sex-dimorphic behavior only when given in pharmacological doses (Plapinger and McEwen, 1978). The nonsteroidal synthetic estrogen diethylstilbestrol (DES) is of particular interest since it does not bind to $\alpha$-fetoprotein and is able to reach the brain in a biologically active form; there it exerts organizational (masculinizing/defeminizing) effects similar to those of androgens converted to estrogens (Plapinger and McEwen, 1978). Indeed, recent studies—as yet unreplicated—have shown that pre- or perinatal DES alters features of sex-dimorphic childhood social play in female rats (Hines *et al.*, 1982a), increases masculine mounting behavior and decreases feminine lordosis in adult female guinea pigs (Hines *et al.*, 1982b), and leads to the expected paradoxical effect of decreased mounting and intromission behavior in adult male rats (Monroe and Silva, 1982). It also affects the timing of puberty in female rats (Slaughter *et al.*, 1977). Döhler *et al.* (1982) have shown structural effects of DES in the preoptic area of the female rat brain.

Data on the effects of prenatal or perinatal hormones on subhuman primates are scant. In prenatally androgenized female rhesus monkeys, the expected effects of prenatal androgens on the development of sex-dimorphic social play behaviors in childhood have been found (Goy and Resko, 1972). The same animals showed increased aggression (and, after testosterone priming, increased proximity toward stimulus females) between 5 and 7 years of age, but their mounting rate did not differ from control females, and only one animal out of seven showed the full masculine copulatory sequence (Eaton *et al.*, 1973). Finally, at the more mature age of 15-17 years, these monkeys gave little evidence of masculinization in their sex-behavioral responsiveness to testosterone (Phoenix and Chambers, 1982). Also, early hormonal influences on gonadotropin regulation in adulthood appeared to be much weaker than in lower mammals (Steiner *et*

*al.,* 1976). A recent study of a new sample of prenatally androgenized female rhesus monkeys (Thornton and Goy, 1983) showed some degree of defeminization in the sexual interaction of these monkeys with males. Due to the small number of monkeys with prenatal hormone treatment investigated in these studies, these findings have to be considered preliminary.

Behavioral studies of prenatal or perinatal DES exposure in nonhuman primates are not available. It has recently been demonstrated (Slikker *et al.,* 1982) that in rhesus monkeys substantial amounts of DES reach the fetal compartment unaltered, in contrast to natural estradiol. Of potentially great significance is another recent study (Fuller *et al.,* 1981) that shows lasting effects of prenatal DES exposure on gonadotropin patterns of infant rhesus monkeys; it is the first study suggesting direct organizational effects of prenatal DES on sex-dimorphic areas of the primate brain.

The generalizability of animal behavior to human analogues, in particular in relation to sexual orientation, is very controversial (Beach, 1979). Nevertheless, that humans are not exempt from prenatal hormone effects on sex-dimorphic behavior has been shown in previous studies using two paradigms, spontaneous endocrine abnormalities of the fetus and sex hormone treatment during pregnancy (Ehrhardt and Meyer-Bahlburg, 1981; Money and Ehrhardt, 1972). Prenatal androgenization of genetic females shifts many childhood behaviors toward the masculine pole (e.g., Ehrhardt and Baker, 1974; Reinisch, 1981) and may slow down the attainment of psychosexual milestones (Ehrhardt, 1979; Money and Schwartz, 1977; Schwartz and Money, 1983). Hypoandrogenization of males leads to behavior alterations compatible with the animal studies, but the hormonal effects usually cannot be separated from the social effects, since most of the data are based on severe forms of intersexuality (Meyer-Bahlburg, 1977). Nonandrogenizing progestogens administered either alone or in combination with estrogens support the notion of demasculinization of childhood behavior in general (Ehrhardt *et al.,* 1977, 1984; Meyer-Bahlburg *et al.,* 1977) and specifically of aggression (Meyer-Bahlburg and Ehrhardt, 1982; Yalom *et al.,* 1973).

If prenatal hormones play any part in the development of sexual orientation, evidence needs to be presented that individuals who differ only in levels of prenatal hormones differ in adult sexual orientation. Apart from a number of small-scale uncontrolled studies suggestive of such hormonal effects (reviewed in Meyer-Bahlburg, 1977, 1979, in press), only one recent study in females employed a clinical group design: Schwartz and Money (1983) have presented data showing increased bisexuality and homosexuality in gonadally intact females with congenital adrenal hyperplasia, a prenatally virilizing condition, when compared to women with vaginal atresia or androgen insensitivity. The only available controlled study that evaluated

sexual orientation in males with an unusual prenatal hormone history, namely prenatal DES exposure, did not find a shift in sexual orientation (Kester *et al.,* 1980).

The current study examines the influence of prenatal DES exposure on sexual orientation in females. The working hypothesis to be tested is that prenatal exposure to DES increases the rate of bi- or homosexuality in women as compared to nonexposed controls.

## METHODS

### Sample Selection

This investigation was approved by the relevant institutional review board of the first author's institution; the Code of Ethics of the World Medical Association was adhered to. All participating subjects gave written informed consent.

From the DES-screening clinic of one of the authors (N.P.V.), we selected women (DES subjects) aged 17-30 years whose prenatal DES exposure had been documented by physician, hospital, or pharmacy records. All of them were followed because of the known increased risk of adenocarcinoma of the vagina in female offspring of women who were treated with DES during pregnancy (Herbst *et al.,* 1971) and because of DES-related vaginal or cervical abnormalities such as adenosis (glandular epithelium or its mucinous products in the vagina). Excluded were subjects of races other than Caucasian or socioeconomic status other than middle or upper class (since these were the most frequent demographic background variables of the entire DES clinic population); and subjects with mental retardation; severe congenital abnormalities and debilitating chronic diseases likely to interfere with everyday functioning; and carcinoma. Once target subjects were identified, we spent a great deal of effort in locating subjects who had changed their addresses and enrolling them in the study, even when they were living far away, in order to minimize volunteer effects and related selection biases. Originally, a total of 76 women with prenatal exposure to DES were sent invitations to participate. Of these, 2 could not be located, 2 did not respond to mail or phone contacts, 2 had to be excluded because they did not meet the criteria, 12 refused participation, 4 were undecided, and 54 agreed to participate. The current report concerns the first 30 DES women from this source who completed the research protocol. The data collection is continuing on the remainder of the sample.

Of the first 30 DES women, 15 had sisters who were not prenatally exposed to DES. One was excluded because of young age (10 years). We obtained permission to approach all other sisters for study participation. Where a DES subject had more than one unexposed sister, the one closest in age to the DES subject was selected. Twelve were interviewed. Two refused participation, one because she anticipated psychological stress, the other because of time pressures; both were never married and were younger than their DES sisters.

The DES subjects were to be compared to a second control group of women who were closely matched in age and socioeconomic background and who shared some of the gynecological procedures they had to undergo in adulthood and in the identification as having an increased cancer risk of the sexual or reproductive organs, but who differed in prenatal DES exposure. To obtain such a comparison sample, we selected from the same coauthor's (N.P.V.) private office population women who had been referred for a diagnostic workup because of an abnormal Pap smear (PAP subjects), using identical exclusion criteria. Initially, 104 PAP subjects were selected and contacted either by mail or in person. Of these, 14 could not be located, 17 failed to respond to repeated mailings and phone calls, and 12 had to be excluded on the basis of race or nationality. Of the remaining 61 PAP controls, 41 agreed to participate, 3 were undecided, and 17 refused. There were 32 PAP controls who were interviewed, and of these 2 had to be excluded, one because she failed to meet the socioeconomic criterion and the other because she believed she had cancer.

While definitive documentation of prenatal DES exposure existed for all DES subjects, the prenatal records varied greatly in the details of the information provided. Of 17 subjects on whom the records were sufficiently detailed, two were exposed to DES during the first trimester only, 11 during the first and second trimesters, 3 during the second and third trimesters, and 1 throughout the pregnancy. Of the remaining 13 subjects, 5 were definitely exposed during the first and possibly during the subsequent trimesters, 4 definitely during the second and possibly during the third, 1 definitely during the second and third and possibly during the first trimester. No specific trimester information was available for 3 subjects. Daily dosage was extremely variable and was often systematically increased during pregnancy; the range was 5-250 mg. Total dosage throughout pregnancy (minimum estimate) could be calculated with reasonable certainty for 13 subjects and ranged from 210 to 10,475 mg. The fact that 27 (90%) of our DES subjects had vaginal adenosis—a much higher percentage than is usually found in DES-exposed women (O'Brien *et al.*, 1979)—makes it likely that our sample was exposed to DES earlier, longer, and to higher dosages than DES women

in general. Of the DES subjects, 25 had been exposed to DES as the only hormone; additional hormones the subjects were exposed to were progesterone in 3 cases; anhydrohydroxy-progesterone (Prenone) in 1 case; and progesterone plus estradiol in 1 case. With regard to the 12 sisters, nonexposure to DES was documented by medical records for 10; the records of the remaining two could not be retrieved, but their mothers were sure that they had not been treated with DES or any other hormone during those pregnancies.

The 30 PAP subjects had undergone colposcopy and, if indicated, punch biopsy, rendering diagnoses of mild, moderate, or severe dysplasia in 22 cases. Many of these had been treated with cryosurgery or cone biopsy, depending on the severity of dysplasia, although some required no treatment at all. In 8 cases, colposcopic examination or biopsy did not reveal any dysplasia, and the abnormal Pap smear may have indicated an inflammation or an infection. Although documentation of prenatal hormone exposure could not be obtained for this sample, exposure to DES is rather unlikely since none of the subjects or their mothers knew about such exposure, nor did any of the PAP subjects show vaginal adenosis.

## Assessment Methods and Procedures

All subjects selected were contacted by their gynecologist (N.P.V.) with a letter explaining the purpose of the study as the psychological assessment of DES patients/PAP patients with the goal to ascertain general psychological development and specific emotional reactions to their medical history. A questionnaire (with return envelope addressed to the gynecologist) was included on which the patients were to indicate whether or not they were interested in study participation. Return of the questionnaire was rewarded with a $3 check. Subsequently, all interested patients were contacted for scheduling. Once a patient agreed to participate in the study, we asked her permission to contact her mother for participation in the project and, where available, the unexposed sister closest in age. Mothers and sisters were then contacted in the same way. Most subjects were evaluated in our research unit at Columbia; some preferred to be seen in the office suite of their gynecologist (N.P.V.), and some had to be tested and interviewed during a home visit. Subjects were reimbursed for their travel expenses and received an additional bonus of $35 for participation.

The 8-hour evaluation protocol included a variety of psychiatric and psychological assessment methods. Psychosexual development and functioning, the main focus of this report, were evaluated by the Sexual Behavior Assessment Schedule-Adult[4] (SEBAS-A; see Meyer-Bahlburg and

[4]Copies are available from the authors.

Ehrhardt, 1983), which covers psychosexual milestones (of romanticism and sexual behavior), sexual orientation, sexual activity level, and sexual dysfunctions. Sexual orientation in terms of imagery, attractions, and overt sociosexual behavior was assessed both for the 12 months preceding the examination and for the interval from puberty to the present ("lifelong"). Aspect-specific subscales as well as global scales were used and included the following variables: masturbation fantasies, masturbation erotica, romantic/sexual daydreams, romantic/sexual nightdreams, sexual attractions, imagery (a global rating encompassing all previously listed variables with the exception of masturbation erotica), sexual relations (with partners), and sexual responsiveness (a global rating incorporating all variables).

For each subscale and each global scale, the Kinsey Rating Scale (Kinsey *et al.,* 1948, 1953) was used with the following formulation: 0 = entirely heterosexual; 1 = largely heterosexual but incidentally homosexual (e.g., night dreams, masturbation fantasies); 2 = largely heterosexual but also distinctly homosexual; 3 = equally heterosexual and homosexual; 4 = largely homosexual but also distinctly heterosexual; 5 = largely homosexual but incidentally heterosexual; and 6 = entirely homosexual. Kinsey 2, or "a distinct" homosexual history was rated when the subject had an activity such as homosexual dreams over a period of at least 1 year recurring with some regularity rather than sporadically or incidentally. Whenever a subscale was rated 2, the corresponding global score could not be rated less than 2. In a few cases, global ratings were rated down to 0 when the highest subscale rating was a marginal Kinsey 1. Additional items concerned homosexual milestones, number of partners, frequency of heterosexual versus homosexual orgasmic experiences, etc.

All SEBAS-A interviews were conducted by female interviewers. Due to staff shortages, on the one hand, and frequent self-disclosure of the patients, on the other, only a fraction of the interviews could be conducted blindly. All interviews were audiotaped. For all subjects who were rated other than 0 on any of the Kinsey scales, all Kinsey-format items were independently and blindly co-rated from tape by three co-raters, and disagreements were resolved by discussion. Data analysis was based mainly on statistical case-control comparisons.

## RESULTS

### DES versus PAP Subjects

The sample characteristics are shown in Table I. At the time of interview, both groups were approximately the same age. Social status, as reflected in the Hollingshead Four-Factor Index (Hollingshead, 1975), was

**Table I.** Sample Characteristics of DES and PAP subjects

| Characteristic | DES ($N$ = 30) | | | PAP ($N$ = 30) | | | $t$ test ($p$, 2-tailed) |
|---|---|---|---|---|---|---|---|
| | $N$ | Mean | S.D. | $N$ | Mean | S.D. | |
| Age at interview (years) | 30 | 25.13 | 3.74 | 30 | 26.00 | 3.25 | n.s. |
| Subjects' Hollingshead 4F Index | 22[a] | 50.93 | 9.27 | 27[a] | 48.56 | 10.84 | n.s. |
| Parents' Hollingshead 4F Index | 29[b] | 49.78 | 9.89 | 30 | 45.13 | 10.28 | n.s. |
| WAIS-R Full IQ | 18[c] | 105.94 | 9.79 | 30 | 98.37 | 10.21 | $\leq .015$ |
| | $N$ | % | | $N$ | % | | $\chi^2$ ($p$, 2-tailed) |
| Current marital status | | | | | | | |
| Married[d] | 11 | 36.7 | | 19 | 63.3 | | |
| Divorced | 4 | 13.3 | | 3 | 10.0 | | $\leq .100$[e] |
| Never married | 15 | 50.0 | | 8 | 26.7 | | |
| Current religion | | | | | | | |
| Catholic | 5 | 16.7 | | 13 | 43.3 | | |
| Protestant | 2 | 6.7 | | 3 | 10.0 | | $< .100$[f] |
| Jewish | 20 | 66.7 | | 13 | 43.3 | | |
| Other | 3 | 10.0 | | 1 | 3.3 | | |

[a]The remaining subjects were still students and unmarried at the time of the interview

[b]This index could not be computed for one DES-exposed subject, due to missing information.

[c]One DES woman was not tested with either the WAIS or WAIS-R because she was familiar with both tests. This subject received the Primary Mental Abilities Test, instead; her full IQ from this test was 126. Eleven DES women were tested with the WAIS (Wechsler, 1955) before the WAIS-R (Wechsler, 1981) became available: Mean Full IQ = 113.00, S.D. = 6.50. According to Wechsler (1981), a WAIS Full IQ of 113 is approximately equal to a WAIS-R Full IQ of 105; thus, the two DES subgroups are comparable in level of intelligence.

[d]One of the married PAP women was separated.

[e]$\chi^2$ was calculated for married plus divorced vs. never married DES and PAP women.

[f]$\chi^2$ was calculated after collapsing "Protestant" and "Other."

also similar for both the subjects and their parents. The Index is based on education and occupation levels and takes marital status into consideration. The majority of both groups are of middle-class or higher socioeconomic background. With respect to the WAIS-R Full IQs, the DES-exposed group was about one-half S.D. above the PAP subjects, a significant difference. (The project was started before the WAIS-R was published. Therefore, the first subjects were tested with the older WAIS form.) Fewer DES women were married than PAP women, a nearly significant difference. There were relatively more Jewish women in the DES-exposed group, and Catholic women in the PAP sample.

The data on sexual orientation are listed in Table II and Appendix A. In comparison with the PAP women, the DES-exposed women had higher Kinsey scores on all items, with most of the differences of at least borderline ($p < 0.10$) statistical significance. Out of the ten variables, only one, masturbation erotica, failed to reach borderline significance, probably due to the relatively small number of women who were using erotic depictions or narratives for masturbatory arousal. The percentages of DES women with bisexual or homosexual responsiveness (Kinsey scale 2 through 6) was 21% (6 out of 29 women with data on this issue) for the preceding 12 months and 24% (7 women) for lifelong, as compared to 3% (one subject) for the preceding 12 months and none for lifelong among the PAP women (Fisher's Exact Test, one-tailed, $p < 0.05$ and $p < 0.01$, respectively).

Five of the 29 DES women, or 17%, had a Kinsey score of at least 3 (i.e., at least equally homo- and heterosexual) for overall sexual responsiveness during the preceding 12 months compared to none in the PAP sample,

**Table II.** DES vs. PAP Group Comparisons: Kinsey Scales (Mann-Whitney *U* test)

| Variable | *N* | | Direction of difference[a] | *p* (1-tailed) |
|---|---|---|---|---|
| | DES | PAP | | |
| Current (past 12 months) | | | | |
| Masturbation fantasies | 18 | 18 | ↑ | .020 |
| Masturbation erotica | 8 | 6 | ↑ | n.s.[b] |
| Romantic/sexual daydreams | 22 | 29 | ↑ | .063 |
| Romantic/sexual nightdreams | 13 | 21 | ↑ | .068 |
| Sexual attractions | 29 | 28 | ↑ | .003 |
| Global rating: imagery | 28 | 30 | ↑ | .094 |
| Sexual relations | 28 | 30 | ↑ | .017 |
| Global rating: sexual responsiveness | 29 | 30 | ↑ | .044 |
| Lifelong (since establishment of a sex life) | | | | |
| Sexual relations | 29 | 30 | ↑ | .018 |
| Global rating: sexual responsiveness | 29 | 30 | ↑ | .004 |

[a]↑, Kinsey scores of DES subjects are higher than those of controls.
[b]n.s., $p \geq .100$.

**Table III.** Sample Characteristics of Paired Subjects and Sister Controls

| Characteristic | DES ($N$ = 12) | | | Sisters ($N$ = 12) | | | Paired $t$ test ($p$, 2-tailed) |
|---|---|---|---|---|---|---|---|
| | $N$ | Mean | S.D. | $N$ | Mean | S.D. | |
| Age at interview (years) | 12 | 24.50 | 3.70 | 12 | 23.58 | 4.08 | |
| Subjects' Hollingshead 4F Index | 8[a] | 50.75 | 8.94 | 7[a] | 45.29 | 13.14 | |
| Parents' Hollingshead 4F Index | 12 | 53.88 | 9.24 | 12 | 53.88 | 9.24 | |
| WAIS-R Full IQ | 8[b] | 110.88 | 8.87 | 8[b] | 104.62 | 11.45 | |
| | $N$ | % | | $N$ | % | | $\chi^2$ (McNemar) ($p$, 2-tailed) |
| Current marital status | | | | | | | |
| Married[c] | 4 | 33.3 | | 8 | 66.7 | | $\leq .001$[d] |
| Divorced | 0 | 0.0 | | 0 | 0.0 | | |
| Never married | 8 | 66.7 | | 4 | 33.3 | | |
| Current religion | | | | | | | |
| Catholic | 2 | 16.7 | | 3 | 25.0 | | |
| Protestant | 1 | 8.3 | | 2 | 16.7 | | |
| Jewish | 7 | 58.3 | | 7 | 58.3 | | |
| Other | 2 | 16.7 | | 0 | 0.0 | | |

[a]The remaining subjects were still students and unmarried at the time of the interview.
[b]Three DES women were tested with the WAIS resulting in a mean Full IQ of 114.67, S.D. = 4.51, corresponding to a WAIS-R Full IQ of about 107. (An additional DES woman was tested with the PMA). The mean WAIS-R Full IQ of the three sister controls whose DES-exposed sisters were tested with the WAIS was 104.35. This difference is not significant, nor is the difference between the eight pairs of WAIS-R tested women.
[c]One of the married sister controls was separated.
[d]$\chi^2$ was calculated for married vs. never-married DES women and their sisters.

also a statistically significant result (Fisher's Exact Test, $p < 0.05$, one-tailed).

Other sections of the interview revealed that six of the seven DES women with a Kinsey score of 2 and higher for lifelong sexual responsiveness had had romantic friendships with other women but that only five had the experience of overt homosexual activities involving genital contact. During the 12 months preceding the examination, two were living with a homosexual partner. For one subject, with a Kinsey 3 rating, the bisexuality was limited to romantic and erotic imagery including attractions and crushes but had not been expressed in mutual romantic or sexual relationships with other women.

## DES Subjects and Sister Controls

Those DES-exposed subjects for whom DES-unexposed sisters were available were separately analyzed in comparison to their control sisters. The sample characteristics of the resulting 12 sibling pairs are listed in Table III. DES-exposed sisters and controls were not significantly different in terms of age, their own or their parents' socioeconomic status, or intelligence. However, significantly fewer of the DES subjects were ever married than subjects of the sister group.

Table IV and Appendix B show the results on sexual orientation. As in the case of the DES-PAP comparisons, the DES women had higher scores on all 10 Kinsey variables than their nonexposed sisters, although only a few of these (sexual attractions, global rating of sexual responsive-

**Table IV.** DES vs. Sister Group Comparisons: Kinsey Scales (Sign Test)

| Variable | $N$ (pairs) | Direction of difference[a] | $p$ (1-tailed)[b] |
|---|---|---|---|
| Current (past 12 months) | | | |
| Masturbation fantasies | 4 | ↑ | n.s. |
| Masturbation erotica | 2 | ↑ | n.s. |
| Romantic/sexual daydreams | 8 | ↑ | n.s. |
| Romantic/sexual nightdreams | 3 | ↑ | n.s. |
| Sexual attractions | 12 | ↑ | .062 |
| Global rating: imagery | 11 | ↑ | n.s. |
| Sexual relations | 11 | ↑ | n.s. |
| Global rating: sexual responsiveness | 12 | ↑ | .032 |
| Lifelong (since establishment of a sex life) | | | |
| Sexual relations | 12 | ↑ | n.s. |
| Global rating: sexual responsiveness | 12 | ↑ | .016 |

[a]↑, Kinsey scores of DES subjects are higher than those of controls.
[b]n.s., $p \geq .100$.

ness in the previous 12 months, and global rating of sexual responsiveness lifelong) reached statistical significance. Five of the 12 DES women (42%) had a sexual responsiveness rating of 2 or higher both for the 12 months prior to the examination and lifelong, as compared to one (8%) of the 12 nonexposed sisters ($p \leq 0.062$, 1-tailed, Binomial Test); and three of the DES-exposed women (25%) had scores of 3 and higher for the previous 12 months and two (17%) for lifelong, as compared to none of their nonexposed sisters ($p \leq 0.125$ and $p \geq 0.200$, respectively, 1-tailed, Binomial Test).

## DISCUSSION

The results of this study show a significantly increased rate of bi- or homosexuality in DES-exposed women compared to two independent control groups. There is a high consistency of this finding across a number of different Kinsey-scale rating categories, and the ratings were confirmed by multiple raters.

Before concluding that the findings on sexual orientation are related to prenatal DES, one has to consider a number of other possibilities. One issue to consider is sampling bias. It is unlikely that any systematic bias favoring bi- or homosexual individuals was introduced in the subject identification procedures, since they were based exclusively on the availability of documentation of DES exposure and on the demographic characteristics listed above. If there was a volunteer effect of the kind that people who were earlier or more easily enrolled in the study tended to be more frequently bi- or homosexual, these biases should have affected the two control samples in the same direction. On the other hand, are bisexual or homosexual individuals underrepresented in the control samples? For instance, in the case of the PAP group, one could speculate that this is a hyperheterosexual sample, since early onset of sexual experience and a history of multiple partners have been linked to an increased risk of developing abnormal Pap smears (Rotkin, 1973). The association between early onset of sexual intercourse and the development of cervical cancer or cervical dysplasia as diagnosed by an abnormal Pap smear is, however, mainly true for women of lower SES (Rent *et al.*, 1972), which does not apply to our sample. In order to get a population estimate of behavior frequencies of the various Kinsey scores, we combined the corresponding Active Incidence data for ages 25 and 30, separately for married and for single plus previously married females (Kinsey *et al.*, 1953, p. 499, Table 142) and applied these figures to all samples, calculating expected values separately for married and for divorced plus never married women, and then combining the figures for the

total sample. On this basis, the expected number of persons with a Kinsey score 2 to 6 in the PAP sample is 1.3, and in the sister sample 0.5; the expected value for Kinsey score 3 to 6 for the PAP sample is 0.8, and for the sister sample 0.3. The actual numbers of such people found in the control groups are very close to these expected values. Thus, these low rates by themselves do not indicate an underrepresentation of the bi- and homosexual spectrum. If one applies the same calculations to the DES sample, the expected numbers for Kinsey 2 to 6 individuals is 2.1, and for Kinsey 3 to 6 individuals, 1.3. The numbers of bi- or homosexual DES-exposed women found in our sample are considerably higher, corroborating the conclusion that bi- and homosexuality are truly increased in the DES sample. Moreover, the expected values calculated for the DES sample exceed those expected for the population at large (which has a lower percentage of singles in this age group than in our DES sample) so that our excess findings are very strong. Of course, the comparison to possible outdated population norms is problematic and has to be considered a crude approximation.

Sampling bias could also play a role if subject and control groups differ in some important demographic variable. However, the DES and PAP groups are very similar in socioeconomic levels and age at examination. The demographic group differences observed, such as in student status, Wechsler IQ, number of siblings, or religion are not large and are unlikely to account for the difference in sexual orientation, since there is little or no evidence in the literature of an association of these variables with sexual orientation in women. With regard to current religion, the seven bi- or homosexual DES women break down as follows: 4 are Jewish, 1 Protestant, 1 Catholic, and 1 other (raised Protestant). Both the PAP woman and the unexposed sister with Kinsey 2 scores are Protestant. Thus, in comparison to the distribution of religion in the total samples, there does not seem to be a particular religious bias associated with Kinsey 2-6 scores. It is also important to note that the demographic differences do not hold up for the DES women in comparison to their unexposed sisters.

If sampling bias is excluded as a confounding variable, a nonhormonal psychological explanation of our findings also has to be considered. The most plausible factor for the increased bisexuality might be the awareness of being a DES daughter. One could hypothesize that DES women might be at risk for being discouraged from heterosexuality because they are more likely to expect sexual and reproductive problems stemming from their history of DES exposure. We are not aware, however, of any systematic data supporting such an association for other medical syndromes. For instance, conditions with an extremely low rate of fertility, such as Turner syndrome, do not seem to be associated with increased bisexual or homosexual orientation. In our syndrome-specific interview schedules, we

asked about the impact of their respective medical conditions on various areas of life. Only two of the 30 DES women mentioned worries about their sex life, in one case continuing to the time of evaluation; both were heterosexual. Thirteen mentioned current concerns about their ability to bear children; two of these belonged to the group of seven bi- or homosexual women. None of the seven stated spontaneously that awareness of the DES condition was responsible for their bisexual development. A definitive answer to this issue cannot be given by our data, however, because none of the interview schedules addressed this particular issue in detail.

Another psychological theory (Storms, 1981) suggests that bi- or homosexual orientation is more likely to develop when puberty starts relatively early. This reasoning does not apply to our samples since there was no significant difference between DES and PAP women nor between DES women and their unexposed siblings, in the recalled age at menarche; on average, the DES women were slightly later than the controls (Meyer-Bahlburg *et al.*, 1984).

Concerning possible psychoendocrine interpretations of our findings, both adult and prenatal hormone conditions must be considered. Two recent endocrine studies of clinical samples of DES-exposed women (Peress *et al.*, 1982; Wu *et al.*, 1980) have demonstrated markedly increased rates of hirsutism and irregular menstrual cycles, both likely to be related to androgens. In fact, the same reports showed elevated testosterone levels in the DES women, especially in those with hirsutism. The testosterone levels were similar to those of hirsute women without a history of DES exposure. In this context, it is interesting to note that elevated androgen levels—although not characteristic of the majority—have been found in about one-third of the homosexual women on whom endocrine data have been published (Meyer-Bahlburg, 1979). Androgens have repeatedly been related to various aspects of female sexuality, and Sanders and Bancroft (1982) have suggested that a negative correlation of testosterone level and satisfaction with heterosexual relationships may lead some women with relatively high testosterone levels into adopting homosexual life-styles. Since, on the other hand, data on positive correlations of androgens and female sexual interest predominate, one can also hypothesize that increased sexual motivation may lead to an enhanced sexual responsiveness to a wider range of stimuli, including those of the same sex (via generalization), both in imagery and overt behavior. This reasoning, however, does not apply to our samples, since the DES women appear to be lower in sexual motivation than the PAP subjects (Meyer-Bahlburg *et al.*, in press). Hormone assays were not part of the present project; thus, we cannot ascertain the potential contributions of sex hormone level differences to our findings.

If data from animal research apply to the human condition, an endocrine contribution during very early (prenatal or perinatal) phases on sex

dimorphism of brain and behavior is more likely. Available data on adult women with prenatal androgen excess either only prenatally (Schwartz and Money, 1983) or pre- and postnatally (Ehrhardt *et al.,* 1968) demonstrate increased rates of bi- and homosexuality similar to the ones found in our DES sample. With regard to the question of which hormonal pathway might be involved in these presumptive brain effects (McEwen, 1983), our data suggest a participation of the estrogen pathway, which is not at variance with the findings on CAH women, since androgens may easily be aromatized to estrogens at the target organ level. The documentation of dosage available to us on these women does not allow us to attempt any statement on dose-response relationships.

A hormonal interpretation is also suggested by the fact that the seven DES women with Kinsey scores 2-6 were exposed to DES only, whereas the women who had DES plus progesterone exposure were all Kinsey 0-1 heterosexuals. A number of animal studies (e.g., Kincl and Maqueo, 1965; Dorfman, 1967; McEwen *et al.,* 1979) have shown that progesterone may protect against masculinizing/defeminizing hormonal effects—of both androgens and estrogens—during early development.

In CAH women, prenatal androgen excess is associated with marked tomboyish childhood play behavior (Ehrhardt and Meyer-Bahlburg, 1981), which may have some significance in their development of higher rates of bi- and homosexuality later in life. Preliminary analysis of the gender-role behavior data in the current study also shows differences between DES women and their controls, although not in regard to tomboyism. The DES women were less nurturant/maternal throughout their development. An interpretation of these data in relation to the development of sexual orientation is difficult.

From the viewpoint of rigorously controlled animal research, studies of human subjects with prenatal exposure to exogenous sex hormones are methodologically quite problematic. In our study, for instance, the DES subjects went through at least four preselection procedures. (1) Pregnancy treatment with DES was not administered in a random fashion but usually only to mothers with a history of miscarriage or acute pregnancy problems. (2) Our DES daughters were selected from a DES screening clinic. It is well-known that DES women who come to DES screening centers, either on their own or on referral by their physicians, typically have more numerous or more severe medical symptoms of DES exposure than DES women selected at random from pregnancy records (O'Brien *et al.,* 1979). (3) Selected for our study were DES daughters from a screening clinic population on whom prenatal documentation was available. (4) Some of those targeted could not be traced or refused to participate in the study. Steps 1 and 2 affect prevalence rates of medical symptoms. Whereas we are not aware of any evidence that suggests an influence of these selection steps on the preva-

lence rates of various degrees of sexual orientation, behavioral effects of some kind seem plausible. Steps 3 and 4 were shared between DES subjects and PAP controls, but steps 1 and 2 leave room for unforeseen confounding variables. Clearly, extreme caution in data interpretation is indicated, and replication in other samples with different control groups is required. Therefore, the findings presented here can only be considered suggestive and are not to be taken as proof of a hormonal contribution to the development of sexual orientation in humans. Even if hormones in general, and DES in particular, should have some influence on the development of sexual orientation, it is important to note that 75% of the DES-exposed women in our sample were exclusively or nearly exclusively heterosexual in spite of the DES exposure. Only one woman out of 30 was nearly exclusively homosexual from the establishment of her sexual life. This means that, at best, DES exposure may have contributed somewhat to the development of sexual orientation but does not have a strong and certainly not a determining influence. Moreover, findings on sexual orientation in individuals with hormonal abnormalities or particular hormonal treatment regimens during fetal life may not have any bearing on the etiology in bisexual or homosexual individuals without such medical histories (Meyer-Bahlburg, in press). Therefore, any conclusions from the data on the specific samples of our study to the development of sexual orientation in general seem unwarranted at this time.

## ACKNOWLEDGMENTS

Drs. Jean Endicott and Patricia Cohen were consultants on this project from its inception. Jane B. Gogan, Elizabeth W. Pierce, Paul G. Simeone, Neil P. Devins, Evan J. Elkin, and Roberta Stiel served as research assistants. Dr. Joel Becker participated in the co-ratings. Patricia Connolly and Dorothy Lewis provided secretarial services. We gratefully acknowledge everybody's contribution, as well as the efforts and cooperation of the women who participated as research subjects.

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**Appendix A.** DES Subjects ($N$ = 30) vs. PAP Subjects ($N$ = 30): Number of Subjects with Individual Kinsey Scores (K0-K6)

| Variable | DES | | | | | | | | PAP | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | K0 | K1 | K2 | K3 | K4 | K5 | K6 | NA[a] | K0 | K1 | K2 | K3 | K4 | K5 | K6 | NA[a] |
| Current (past 12 months) | | | | | | | | | | | | | | | | |
| Masturbation fantasies | 10 | 3 | 1 | | 1 | | 3 | 12 | 15 | 3 | | | | | | 12 |
| Masturbation erotica | 4 | 1 | 2 | | | 1 | | 22 | 3 | 3 | | | | | | 24 |
| Romantic/sexual daydreams | 14 | 3 | 1 | 1 | 1 | 1 | 1 | 8 | 23 | 5 | 1 | | | | | 1 |
| Romantic/sexual nightdreams | 8 | 2 | 1 | | 1 | | 1 | 17 | 17 | 4 | | | | | | 9 |
| Sexual attractions | 20 | 3 | | 2 | 1 | 1 | 2 | 1 | 27 | | 1 | | | | | 2 |
| Global rating: imagery | 17 | 5 | 2 | | | 2 | 2 | 2 | 22 | 6 | 2 | | | | | |
| Sexual relations | 24 | 1 | 1 | | | | 2 | 2 | 30 | | | | | | | |
| Global rating: sexual responsiveness | 17 | 6 | 1 | 1 | 1 | 1 | 2 | 1 | 24 | 5 | 1 | | | | | |
| Lifelong (since establishment of a sex life) | | | | | | | | | | | | | | | | |
| Sexual relations | 23 | 1 | 3 | | 1 | | 1 | 1 | 29 | 1 | | | | | | |
| Global rating: sexual responsiveness | 13 | 9 | 3 | 2 | 1 | 1 | | 1 | 22 | 8 | | | | | | |

[a]NA = Number of subjects for whom the particular variable is not applicable.

**Appendix B.** DES Subjects ($N$ = 12) vs. Sisters ($N$ = 12): Number of Subjects[a] with Individual Kinsey Scores (K0-K6)

| | DES | | | | | | | Sisters | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Variable | K0 | K1 | K2 | K3 | K4 | K5 | K6 | K0 | K1 | K2 | K3 | K4 | K5 | K6 |
| Current (past 12 months) | | | | | | | | | | | | | | |
| Masturbation fantasies | 2 | | 1 | | 1 | | | 3 | | | | 1 | | |
| Masturbation erotica | 1 | | | | | | 1 | 1 | | | 1 | | | |
| Romantic/sexual daydreams | 6 | | | 1 | | 1 | | 7 | 1 | | | | | |
| Romantic/sexual nightdreams | 1 | 1 | 1 | | | | | 3 | | | | | | |
| Sexual attractions | 8 | | | 2 | 1 | 1 | | 10 | 1 | 1 | | | | |
| Global rating: imagery | 8 | | 2 | | | 1 | | 9 | 1 | | 1 | | | |
| Sexual relations | 9 | 1 | 1 | | | | | 11 | | | | | | |
| Global rating: sexual responsiveness | 7 | | 2 | 1 | 1 | 1 | | 9 | 2 | 1 | | | | |
| Lifelong (since establishment of a sex life) | | | | | | | | | | | | | | |
| Sexual relations | 9 | | 3 | | | | | 12 | | | | | | |
| Global rating: sexual responsiveness | 6 | 1 | 3 | 2 | | | | 9 | 2 | 1 | | | | |

[a]For each variable, the frequency is based on the number of matched pairs where each pair member has a Kinsey rating; matched pairs where one or both members have an NA ("Not Applicable") rating are excluded.