Development and Familiality of Sexual Orientation in Females

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The development and familial clustering of sexual orientation were studied in 358 heterosexual, bisexual, and homosexual women. Sexual orientation, as measured by the Kinsey scales, was diverse yet showed statistical congruity and stability over a 1- to 1.5-year time span. Developmental patterns, as measured by retrospective reports on the ages of first sexual or romantic attraction and of self-acknowledgment of sexual orientation, were very similar in the heterosexual and lesbian subjects except for the difference in object choice. The bisexual subjects displayed intermediate patterns that were more similar to the heterosexuals' on most facets yet closer to the lesbian subjects' on other dimensions. Familial clustering of nonheterosexual orientation was significant. Using two criteria, elevated rates of nonheterosexuality were found in four classes of relatives: sisters, daughters, nieces, and female cousins through a paternal uncle. The current data are not sufficient to distinguish between genetic and shared environmental sources of this familial aggregation. We discuss the possibility of using developmental criteria to differentiate between inherited and cultural sources of variation in female sexual orientation.

KEY WORDS: Homosexuality; lesbian; bisexual; sexual orientation; familiality; development; heredity.

INTRODUCTION

Over half a century has past since Hirshfeld (1936) commented on the apparent familiality of both female and male homosexuality. However, systematic behavioral genetic studies designed to examine the sources of this familial aggregation have only recently been attempted.

Genetic studies of sexual orientation in women have been particularly sparse. Familial aggregation has been observed in two nuclear family studies in which homosexual women reported having more lesbian sisters than did heterosexual women (Pillard, 1990; Bailey and Benishay, 1993). However, the genetic and environmental contributions to this familial clustering have not been resolved by the four published twin studies, which have yielded somewhat contradictory results. Bailey et al. (1993), who analyzed the largest and most systematically collected series of subjects, estimated heritability ($h^2$) to be approximately 0.5 (range, 0.27 to 0.76) based upon concordance rates of 48% for monozygotic twins compared to 16% for dizygotic twins raised together. Similarly, Whitman et al. (1993) reported a 75% concordance rate for four pairs of female monozygotic twins. In contrast, Eckert et al. (1986) described four sets of female identical twins raised apart who were discordant for sexual orientation. King and McDonald (1992) also found low twin concordance rates for homosexuality in their combined study of female and male cotwins but did not break down the data according to sex.

The single published adoption study of female sexual orientation showed that the rate of homo-
sexuality was lower in the adoptive than in the biological sisters of lesbian probands (Bailey et al., 1993). Although this was interpreted to support a genetic hypothesis, some effect of shared environmental factors could not be ruled out because heterosexual probands were not included in the study. This highlights one of the difficulties with research in this area, namely, the uncertainty about the population incidence of nonheterosexuality in females; current estimates range from 0.6 to over 10%, depending on the criteria and sampling strategy employed (Kinsey et al., 1953; Bailey and Benishay, 1993; Johnson et al., 1994). Therefore it is prudent to establish background rates independently for each new protocol and group of subjects under study.

Several papers have addressed the question of whether the familialities of female and male homosexuality are etiologically independent or overlapping, but the results and interpretations are ambiguous. The prediction of the independent model is that female and male homosexuality will run in different families; that is, that female homosexual subjects will have an excess of lesbian sisters but not of gay brothers, whereas male homosexual subjects will show the opposite pattern. Pillard (1990) and Bailey and Benishay (1993) did find that lesbians had more homosexual sisters than brothers, a noteworthy finding since the population incidence of homosexuality is approximately half as high in women as in men; however, the differences between sisters and brothers were not statistically significant in the samples studied. Similarly, Pillard and Weinrich (1986), Bailey et al. (1991), Bailey and Pillard (1991), and Hamer et al. (1993) found that gay men had more homosexual brothers than sisters, but again, the differences were not significant. In contrast, Bailey and Bell (1993), who reanalyzed data collected by Bell et al. (1981a, b), found that both gay men and lesbians had more homosexual brothers than sisters. The prediction of the overlapping model is that elevated rates of homosexuality will be found in the opposite-sex siblings of both female and male homosexual subjects. Significant evidence in this direction has been presented by Pillard (1990), Bailey and Benishay (1993), Hamer et al. (1993), and Bailey and Bell (1993). Hence there are data to support both the independent and the overlapping models for the familial aggregation of female and male homosexuality, and it is possible that both hypotheses are partially correct.

A clear prediction for a genetically influenced trait is that it should appear at elevated rates in second and third degree lineages as well as within the nuclear family. Hamer et al. (1993) found that male homosexuals had higher than baseline rates of gay maternal uncles and male cousins through maternal aunts but not of gay paternal relatives. This led to the hypothesis of sex-linked transmission in selected families and the eventual identification of a linkage between DNA markers on a discrete region on the X chromosome known as Xq28 and male sexual orientation. Similar extended family and molecular studies of female sexual orientation have not been reported.

The clearest evidence linking genetics and sexual orientation in a subset of women comes from studies of female patients with congenital adrenal hyperplasia (CAH), a group of enzymatic deficiencies in cortisol biosynthesis transmitted by autosomal recessive genes. The most common form, accounting for 95% of the cases, is a deficiency in 21-hydroxylase activity mediated by the cytochrome P450c21 gene located within the human HLA major histocompatibility locus on the short arm of chromosome 6 (Higashi et al., 1986; Miller, 1988). Insufficient cortisol production results in an increased accumulation of androgens, causing a masculinization of the genitalia to varying degrees. Influences on the developing brain are also believed to occur but are not well understood. However, a "masculine" pattern of gender-role behavior has been reported for CAH patients in several studies (Money and Ehrhardt, 1972; Ehrhardt, 1979; Ehrhardt and Meyer-Bahlburg, 1981; Slijper, 1984). Furthermore, recent studies focusing on adult psychosexual development and sexual orientation indicate that females with CAH have significantly higher rates of homosexual orientation, behavior, and fantasy coupled with lower rates of heterosexual activity compared to their nonaffected sisters (Dittman et al., 1990a, b, 1992). These results suggest that excess prenatal androgens can predispose some women toward development of a homosexual orientation. However, because CAH is a relatively rare condition, and a majority of patients develops an apparent heterosexual orientation, it is evident that CAH plays only a minor role in the overall variability of female sexual orienta-