Neurohormonal Functioning and Sexual Orientation: A Theory of Homosexuality–Heterosexuality

Lee Ellis
Department of Sociology
State University of North Dakota at Minot

M. Ashley Ames
Indiana University at Bloomington

Following a historical sketch of attempts to explain homosexuality, we review evidence indicating that the process of determining human sexual orientation is fundamentally the same in all mammals. In this process, four phenotypic dimensions of sexuality develop from two more or less distinct sex genotypes. Studies are reviewed that indicate how phenotypic deviations from these two genotypes (called *sexual inversions*) can occur. The causes of sexual inversions are categorized as genetic-hormonal, pharmacological, maternal stress, immunological, and social experiential. From this evidence, we propose a theory of how the entire spectrum of human sexual orientation (vs. simply homosexuality) is determined.

A consistent preference for sexual relations with one's own sex (homosexuality), the opposite sex (heterosexuality), or varying degrees of ambivalence about the partner's sex (bisexuality) may be called *sexual orientation*. Homosexuality should not be confused with occasional homosexual experiences. Homosexual experiences are fairly common, especially early in adolescence (Chilman, 1983, p. 18; Kinsey, 1941) or in the absence of alternative sexual outlets (Aldridge, 1983; Groth & Burgess, 1980) and are no more indicative of homosexuality than occasional heterosexual experiences are indicative of heterosexuality. An individual's sexual orientation refers to distinct preferences consistently made after puberty in the presence of clear alternatives, whereas isolated instances of sexual behavior may or may not reflect one's sexual orientation (Gadpaille, 1972, p. 193).

History of Explanations for Sexual Orientation

Prior to this century, virtually all explanations of sexual orientation defied scientific verification. At least in the Western world, heterosexuality was attributable to what God had ordained as natural and good, and all deviations from it (along with all other nonprocreative sexual acts) were seen as the work of the devil or a sinful person's freely choosing to be evil (Allen, 1967; Greenberg & Bystryn, 1984). Throughout the twentieth century, scientists have tried to explain sexual orientation in more or less empirically testable terms. Most explanations have taken heterosexuality as a given and focused on explaining why a minority of individuals deviate from it. These explanations can be divided into those that focus on experiential and social

learning variables and those that emphasize genetic and physiological variables.

Experiential and Social Environmental Explanations

Early scientific attempts to explain homosexuality largely in environmentalistic terms were made by Freud (1905, 1953; see also Fenichel, 1945). Freud argued that homosexuality reflected a premature fixation of one's psychosexual development. Although he did not dismiss hereditary factors altogether, Freud thought that fixated psychosexual development was typically due to the presence of a domineering mother or the absence of a dominant father. Focusing almost entirely on male homosexuality, most contemporary psychoanalytic explanations continue to emphasize the theme of a romantic triad including a dominant mother, a weak father, and the mother's favorite son (Bieber & Bieber, 1979; Socarides, 1968). Some psychoanalytic explanations have also attributed homosexuality to seduction in early childhood by an older same-sex sibling or playmate, suggesting that this too could prematurely arrest psychosexual development (Cameron, 1963). Research supporting the psychoanalytic explanation primarily consists of evidence that male homosexuals have been reared by unusually protective mothers and/or detached and unloving fathers, although other interpretations of these findings are possible (discussed under the third hypothesis in the *Deductions* section).

Westermarck (1922) proposed another explanation for homosexuality. Noting that homosexuality appeared to be more prevalent in men than in women, he attributed it, at least in part, to the absence of eligible women, either because there were too few women or because of their excessive training in chastity.

Family environmental explanations, which emphasized inadequate parenting as the principal cause of homosexuality, were espoused in the 1940s, 1950s, and 1960s (Bakwin & Bakwin, 1953; Bene, 1965; T. V. Moore, 1945; Storr, 1964; West, 1959). These are sometimes referred to as *psychoanalytic explanations*, even though they downplayed Freud's assumption that sexual motives pervaded parent–child interactions. These theo-

rists shared with Freud the idea that homosexuality is an immature stage of psychosexual development, but focused much more broadly than Freud did on many aspects of family life, arguing that psychosexual development can be arrested in the homosexual stage by many factors, including unhappy and broken homes, inadequate parental and same-sex role models, as well as by dominant mothers and/or affectionless and weak fathers (Friedman & Stern, 1980).

Explanations that emphasized social environmental variables outside the home can be traced back to the 1940s. Without dismissing genetic involvement, East (1946), for example, attributed most homosexuality to confusion during the time one learns appropriate sex roles. East contended that if a person's appearance or mannerisms happen to resemble those of the opposite sex, especially if such traits are accompanied by an early homosexual seduction, the individual may elicit incorrect, or at least confusing, social reinforcement for his or her sex role behavior and thereby become a prime candidate for lifelong homosexuality. East's views were later elaborated by Kagan (1964), although Kagan also invoked the concept of self-labeling as both a response to and a reinforcement of others' impressions of the appropriateness of a person's sex role behavior (see also Plummer, 1981).

Another social environmental explanation focused outside the home was proposed by Kardiner (1963), who attributed male homosexuality to excessive societal demands on boys to be "masculine." Boys who felt inadequate in complying with those demands were believed to seek refuge in female roles.

Reinforcement learning principles inspired other learning explanations in the 1950s and thereafter. Kinsey, Reichert, Cauldwell, and Mozes (1955), Hacker (1957), James (1967), and Acosta (1975) all argued that homosexuality frequently resulted from the reinforcing nature of same-sex sexual encounters that happened to precede opposite-sex sexual encounters. Similarly, Gagnon and Simon (1973) maintained that sexual orientation was learned through varying schedules of reward and punishment. Assuming that homosexual experiences are fairly common during childhood and early adolesence, Gagnon and Simon reasoned that if these experiences were pleasurable and/or heterosexual encounters were distasteful (for whatever reason), a homosexual orientation was likely to become the dominant preference in adulthood (see also Akers, 1985, p. 195).

Imprinting theory, another social learning explanation of sexual orientation, was introduced in the 1950s. On the basis of ethological learning principles championed by Lorenz (1966), Smitt (1952) and Young (1961) argued that the first year or two of life are characterized by sexual neutrality, but that by the second or third year of life a person's sexual orientation is formed. Subtle, often accidental social encounters during this critical period cause sexual orientation to develop gradually, but irreversibly, toward a variety of ends. Imprinting theorists never spelled out in any detail the nature of the differences in experiences between those who develop a homosexual versus a heterosexual orientation.

A recent variant of a social learning explanation postulated a homosexual–heterosexual labeling concept (Robertson, 1977; Sagarin, 1975). According to this view, persons whose sexual experience happened to be with a member of the same sex would be inclined to label themselves as homosexual. Thereafter, the impression would most likely persist unless sufficient heterosexual experiences compensated for the homosexual label.

The most recent social learning explanation relied more on classical than operant conditioning principles. Working from evidence that male homosexuals reach sexual maturity somewhat earlier than male heterosexuals, Storms (1981; Wasserman & Storms, 1984) argued that early-maturing males are more likely to reach sexual maturity at a time when males are still largely interacting with one another, whereas later-maturing males are more likely to experience their first sexual interests at a time when societal forces have begun to encourage increased heterosexual contacts (e.g., school dances). Thus early-maturing males are more likely than later-maturing males to pair sexual awakening with males than with females.

Genetic and Physiological Explanations

The first serious attempt to explain sexual orientation in terms other than either social experiences or religious dogma seems to have been made by Von Kraft-Ebbing (1886/1965), and later elaborated on by H. Ellis (1915). Without being specific, they argued that homosexuality was "inborn." Their rejection of learning as a major cause of homosexuality came from their failure to detect any unusual social experiences common to homosexuals and from evidence that homosexual behavior apparently was not unique to humans.

With increased understanding of the endocrine system early in this century, hormonal influences on sexual orientation began to be explored. Hirschfeld (1920) and Forel (1924) conjectured that male and female hormones are rather delicately balanced and that certain unspecified imbalances could result in a homosexual orientation. Studies designed to detect endocrine imbalances in homosexual males proceeded from that point through the 1940s, but were hampered by the crude methods of measuring hormonal levels. Since then, little evidence has been found that circulating testosterone levels in male homosexuals are appreciably different from the average levels in male heterosexuals (see the fifth hypothesis in the *Deductions* section). Moreover, treating male homosexuals with supplemental testosterone did not alter their sexual orientation (Barahal, 1940). More complicated endocrinological differences seem to exist between many homosexual and heterosexual men, however (as discussed under the fifth hypothesis in the *Deductions* section).

The involvement of genetic factors in sexual orientation was studied by several researchers in the 1940s (Darke, 1948; Glass, DeVel, & Wright, 1940; Kallman, 1952; Lang, 1940; Jensch, 1941; Witschi & Mengert, 1942). The initial studies were based on statistical surveys rather than on laboratory investigations of genetic material per se. They examined twinning, sex of siblings, and other family pedigree features for detectable patterns in the transmission of homosexuality. Other investigators in the 1950s scrutinized the chromosomes themselves for abnormalities that might correlate with sexual orientation, but with few encouraging findings (Gentele, Lagerholm, & Lodin, 1960; Paré, 1956).

In the 1940s, two studies implicated brain functioning as a

cause of homosexuality (Kolarsky, Freund, Machek, & Polak, 1967; Silverman & Rosanoff, 1945). Kolarsky et al. theorized that the brain centers programming sexual orientation may be fairly easily disoriented by physical or chemical insults, especially during critical periods of childhood development.

In the late 1960s, new hormonal explanations of sexual orientation began to appear (Dorner, Docke, & Moustafa, 1968a, 1968b; Loraine, Ismail, Adamopoulos, & Dove, 1970; Saba, Salvadorini, Galeone, & Luisi, 1973). Unlike the theories in the 1920s, which concentrated on circulating levels of sex hormones after puberty, these newer explanations focused on perinatal hormone levels (i.e., hormone levels during gestation or soon after birth). A basic assumption underlying these explanations was that male and female brains are different, especially in those areas directly responsible for sexual behavior. Advocates of these explanations derived many of their arguments about the causes of homosexuality from evidence that lifelong homosexual behavior can be experimentally induced in nonhuman mammals if hormones are manipulated perinatally, and from human studies of genetic and drug-induced behavioral anomalies (discussed in the section on *Pharmacological Causes of Human Sexual Inversions*).

In this article, we argue that scientific evidence supports the view that hormonal and neurological variables, operating during gestation, are the main determinants of sexual orientation. This does not deny the involvement of experiential and social environmental variables, at least in the case of individuals who were exposed to intermediate levels of the requisite hormonal regimens (C. L. Moore, 1985, p. 39), but it does imply that very unusual postnatal experiences would be required to overcome strong predispositions toward either heterosexuality or homosexuality. Before confronting the question of how homosexuality (and, to a lesser degree, bisexuality) is determined, the neurohormonal determination of heterosexuality must be described.

Key Terminology

Concepts of Masculinity and Femininity

Before outlining crucial events in the typical patterns of sexual differentiation, the concepts of *masculine* and *feminine* must be considered. As they are commonly used, these terms often imply the opposite ends of a single continuum. Such a view can be misleading, however, both for humans (Reinisch, Gandelman, & Spiegel, 1979, p. 218) and for mammals in general (Kalcheim, Szechtman, & Koch, 1981; Van de Poll, de Bruin, van Dis, & van Dyen, 1978). For example, experiments with rats have identified certain hormonal regimens that cause males to sexually present to other males (typically a female behavior pattern among rats), even though these males also sometimes try to mount receptive females (typically a male behavior pattern; Dahlof, Hard, & Larsson, 1977; Whitney & Herrenkohl, 1977). Designating the sexual behavior of these animals as simply masculine or feminine obscures important features of their behavior.

At the neurological level, evidence indicates that more or less separate brain parts control masculine and feminine behavior (Arnold & Gorski, 1984, p. 436). Whereas the masculine brain areas normally develop at the expense of the feminine areas, this is not always the case. In fact, sometimes neither masculine nor feminine brain areas develop (McGivern, Claney, Hill, & Noble, 1984).

Terms for designating sexuality as not simply masculine or feminine are sorely needed. We use F/dM to refer to those traits that basically are feminine as well as demasculinized, M/dF for traits that are both masculine and defeminized, F/M for traits that are both feminine and masculine (i.e., ambiguous), and dF/dM for traits that are both defeminized and demasculinized.

Attributing Sexual Orientation to Nonhumans

Another issue requiring attention before we delve into the etiology of sexual orientation is whether the terms *homosexual* and *heterosexual* can be applied outside the human species. Without being able to deny or confirm that humans alone possess self-concepts of being homosexual or being heterosexual, our references to terms denoting variations in sexual orientation pertain only to sexual preferences inferred from overt behavior.

Presumably, in response to strong evolutionary pressure, most animals display a preference for sexually interacting with members of the opposite sex. This does not mean that they always choose members of the opposite sex, or that a minority does not predominantly choose members of the same sex (see Maple, 1977, p. 1174). Yet, because heterosexuals are more apt to reproduce, most members of all species (a) appear capable of discriminating one sex from the other, and (b) when allowed equal access to both sexes, usually choose to sexually interact with members of the opposite sex. We use the term *heterosexuality* to indicate such a preference, and we call individuals who display this preference *heterosexuals*, regardless of their species. Likewise, *homosexuals* refers to those animals who always, or nearly always, choose members of their own sex as sex partners. With these stipulations in mind, nonhuman animals can be said to have sexual orientations just as humans do.

Sexual Inversions

Sexual differentiation normally proceeds along four distinguishable phenotypic dimensions that all tend to complement the genetic sex (L. Ellis, 1982). A failure to differentiate in accordance with one's genetic sex in one or more of these four dimensions of sexuality is called a *sexual inversion*. If an individual with an XY karyotype tends to appear or function more in the F/dM range than in the M/dF range for a particular trait, a *male sexual inversion* is said to be present. In addition, if a genetic female tends to appear and/or function more in the M/dF range than in the F/dM range for one or more phenotypic dimensions, this is called a *female sexual inversion*. Before presenting examples of sexual inversions and discussing their causes, we should note that little consideration is given to sex karyotypes other than the usual XX or XY forms.

As we use the terms, sexual inversion and homosexuality are not entirely synonymous. Usually, homosexuality is a type of sexual inversion; but because an individual's sex normally is judged on the basis of morphological appearance, rather than

on a genetic basis (Money, 1969, p. 204), even this statement would have exceptions. An example—documented in humans (Ehrhardt & Meyer-Bahlburg, 1981), baboons (Bielert, 1984), and various rodents (Von Berswordt-Wallrabe, 1983)—involves genetic males who have an F/dM appearance (both genitally and in terms of secondary sex characteristics). They tend to prefer males as sex partners. Thus, both their appearance and their sex preferences are inverted relative to their genetic sex. Nevertheless, because people generally judge someone to be a homosexual if his or her "sex of appearance" is inconsistent with his or her preferences for sex partners, these examples would not be instances of homosexuality.

Five Dimensions of Sexuality

The present explanation of sexual orientation is based on current knowledge about sexual differentiation in general. One of the most important discoveries in this regard is that sexual differentiation develops in essentially the same fashion throughout the mammalian order (Ford, 1983; Grumbach, 1979; Loumaye, Thorner, & Catt, 1982). This means that knowledge gained from nonhuman mammalian experiments can be used to help understand human sexual differentiation, provided one allows for the varying time frames involved in human development and for a few minor species differences in biochemistry and the sequential timing of the synthesis and release of critical hormones. With this in mind, we outline how sexual differentiation normally unfolds within five dimensions. Four figures illustrate the sexual differentiation process, as follows. First, Figure 1 illustrates each of the five dimensions of sexuality and their primary interrelations from a causal standpoint. Figure 1 provides the most general picture of the process of sexual differentiation. Next, Figures 2 and 3 illustrate the main biochemical and sexual differentiation events throughout gestation for males and for females, respectively. Basically, these two figures provide a continuous account of how the second, third, and fourth dimensions of sexuality are organized. Last, Figure 4 illustrates the interrelation of the major sex hormones, and the various proteins and enzymes that cause them to convert into one another. We initially focus on the sex hormones themselves, but later cover the enzymes needed to convert the hormones into one another.

Genetic Dimension

The development of a mammal of either sex normally starts with one of two distinct genetic programs. The typical female genetic program consists of two concordant sex chromosomes (XX), and the male program consists of two disconcordant sex chromosomes (XY). A number of anomalous conditions result when deviations from these two basic sex karyotypes occur (see Kolata, 1986; Plomin, DeFries, & McClearn, 1980, p. 162), but we do not cover them in this article.

A key to the transcription of these two distinct genetic programs into male and female phenotypes largely involves genes on the Y chromosome, although the precise chemical code or codes involved still have not been identified (Kolata, 1986).

Genital Dimension

The genital dimension of sexuality entails all of the internal and external structures in and around a mammal's groin that are associated with reproduction. The basic appearance of these structures is determined fairly early in gestation, as Figures 2 and 3 show. Figure 2 identifies male differentiation of the genitals beginning during the 1st month of gestation and completed in the 5th month, with the bulk of changes occurring during the 3rd and 4th months. Normally, differentiation of the female genitals, especially of the internal structures, is delayed approximately 1 month compared with the male genitals (J. D. Wilson, George, & Griffin, 1981). Thus, Figure 3 shows differentiation of the female genitals beginning about midway through the 2nd month of gestation and completed soon after the start of the 6th month. Figure 5 illustrates the external genitals as they undergo sexual differentiation (Daly & Wilson, 1978; Martin & Voorhies, 1975). The main biochemicals responsible for these sexually dimorphic genital features are identified in the top portions of Figures 2 and 3. Starting with the substances shown at the bottom of the biochemistry section of both graphs, two trophic (or triggering) hormones are named: chorionic gonadotropin (CG), and luteinizing hormone (LH).

In humans, almost from conception, CG begins to cross the placental barrier of the fetus, regardless of the fetus's sex (Fishel, Edwards, & Evans, 1984). This maternal placental supply of CG peaks at the beginning of the 3rd month of gestation, declines slightly for 4–5 weeks, then begins a pronounced decline early in the 4th month of gestation. As the maternal placental levels of CG subside in the 3rd month, the fetus itself begins to produce the hormone (McGregor, Kuhn, & Jaffe, 1983). In addition, during the 3rd month of gestation, fetal production of LH becomes significant (Reiter & Grumbach, 1982). In the mother, CG may help prevent spontaneous abortion, apparently by promoting ovarian production of progesterone (Dalterio, Bartke, Brodie, & Mayfield, 1983). The function of CG in the fetus of either genetic sex is to induce the synthesis of various sex hormones. Later in gestation, LH is needed as well as CG for continued testosterone synthesis in the male fetus (Grumbach, 1979).

Soon after conception, genes on the Y chromosome trigger the synthesis of one or more chemicals that cause masculine variants on the basic female mammalian structures to begin to appear (Kolata, 1986). The first structure to undergo change is the gonadal primordium. Specialized cells (Leydig cells) start forming in the gonadal tissue that will soon begin synthesizing testosterone, a crucial hormone in sexual differentiation (Bloom & Fawcett, 1968). As Figure 4 shows, testosterone and all the other primary sex hormones are synthesized from cholesterol (Migeon, Amrhein, Keenan, Meyer, & Migeon, 1979). Before the newly formed Leydig cells can actually produce testosterone, they must be "switched on" by CG. As Figures 2 and 3 show, CG and LH levels are not significantly different for the two sexes. However, because females lack the Y chromosome, they fail to form Leydig cells, and thus no prenatal testosterone is produced. Testosterone is vital to the production of the male phenotype, and unless females are exposed to abnormally high levels of testosterone from an outside source (which could include the mother), their phenotype remains F/dM.
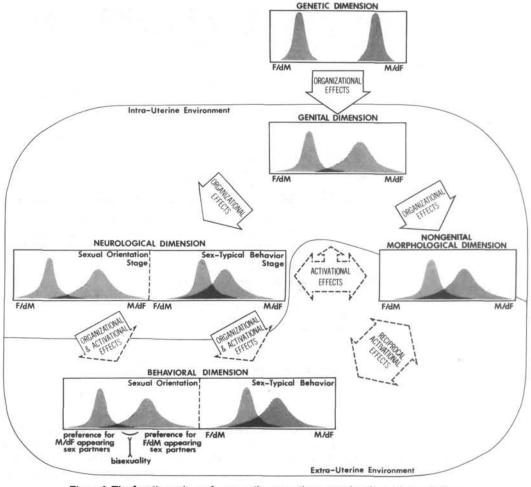
Figure 1. The five dimensions of mammalian sexuality and their primary interrelations. (F/dM = feminine, demasculinized. M/dF = masculine, defeminized.)

A second type of specialized cell also forms in male gonadal tissue: Sertoli cells. Sertoli cells become significant in number and function in the 2nd month of gestation. They primarily synthesize müllerian duct inhibitory factor. As the name implies, this substance prevents the formation of the structures that, in females, become the uterus and fallopian tubes (Shepherd-Look, 1982).
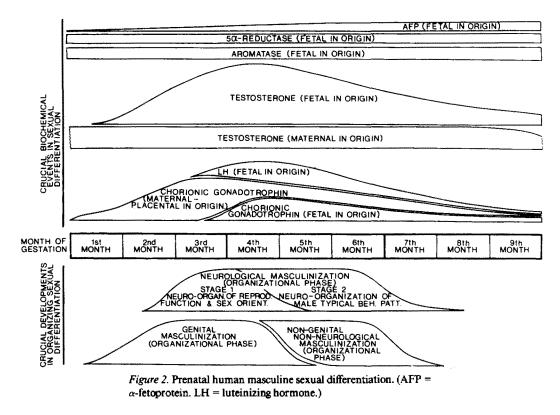
Some of the testosterone synthesized by the Leydig cells in the first 4 months of gestation is converted into an important metabolite of testosterone called dihydrotestosterone (DHT). This conversion is induced by 5α-reductase, as shown in Figure 3. Dihydrotestosterone is largely responsible for masculinizing the external genitalia.

For female differentiation the process is somewhat simpler. The ovaries begin to function at about the 4th month of gestation (see Figure 2F). The müllerian ducts form fallopian tubes and connect the ovaries to the uterus and vaginal cavity.

As Figure 5 shows, the sexes typically proceed along two recognizable routes in external genital appearance. However, the variability about these modal patterns in genital appearance is considerable.

Nongenital Morphological Dimensions

The development of secondary sex characteristics proceeds in two fairly distinct phases, which together constitute the nongenital morphological dimension of sexuality. For fetuses that are being masculinized, the first phase, the organizational phase, is the period in which testosterone produces most of its permanent effects. The biochemical nature of these alterations is complex and involves the formation of hormonal receptor sites within the cells. Receptor sites are proteins with an affinity for various specific hormones. In human males (Figure 2), this phase starts during the 4th month of gestation and lasts through the 7th month. The second phase, the activation phase, begins at puberty. During the second phase, the same hormones that were instrumental in establishing the receptor sites are produced in large quantities, and then bond to (and activate) the receptor sites, thereby altering the cell's functioning. If the receptor sites are not filled, only minor nongenital morphological differences appear between individuals who were exposed to high (vs. low) levels of testosterone during gestation (see Bardin & Catterall, 1981). Thus, the reason only slight sex differences

Figure 2. Prenatal human masculine sexual differentiation. (AFP = α-fetoprotein. LH = luteinizing hormone.)

in muscle mass and strength are present prior to puberty (Jacklin, Snow, & Maccoby, 1981), but then become quite noticeable afterward is that only a small percentage of the more abundant testosterone receptor sites in males are filled before puberty. For a small percentage of each sex, however, fairly substantial cross-sex secondary sex characteristics can be activated at puberty—that is, for those of either sex within the overlap of

the other sex's distribution in testosterone levels during a significant amount of the genital phase of sexual differentiation.

For the typical female, essentially no organizational phase of nongenital morphological sexual differentiation occurs. Commencing at puberty, female ovaries produce progesterone and estrogen. In the absence of testosterone receptor sites, these female hormones have little ability to enlarge muscle tissue. Nev-
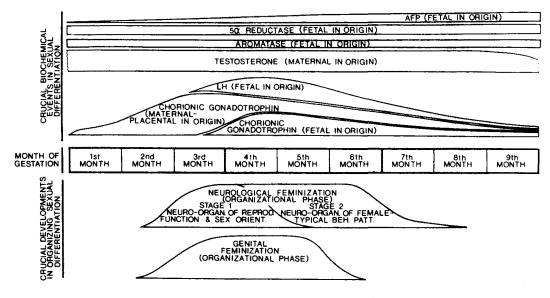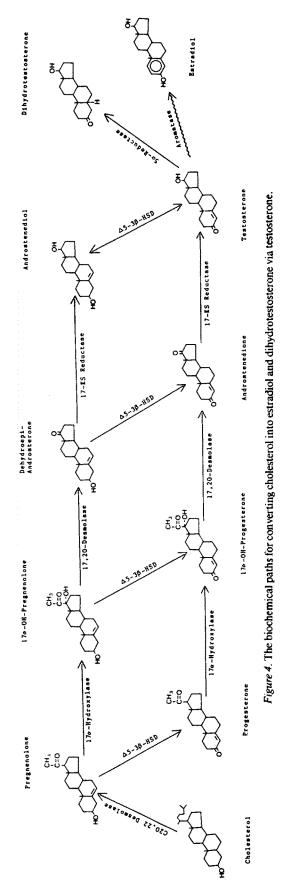

Figure 3. Prenatal human feminine sexual differentiation. (AFP = α-fetoprotein. LH = luteinizing hormone.)

Figure 4. The biochemical paths for converting cholesterol into estradiol and dihydrotestosterone via testosterone.

ertheless, female fetuses produce small quantities of testosterone on their own, and this is supplemented by testosterone produced by the mother that crosses the placenta (Geschwind, 1983, p. 36). To the extent that some females receive above average testosterone exposure and some males' testosterone production is below average, overlap in morphological appearance occurs both before and after puberty.

Neurological Dimension

That the nervous systems of human males and females are different is an issue with a long and controversial history (Sayers, 1982). Despite excesses in interpretation and sometimes even distortions of data to fit one's preconceptions, the evidence increasingly points toward the conclusion that there are many significant average sex differences in human brain structures and functions (e.g., de Lacoste-Utamsing & Holloway, 1982; Geschwind & Galaburda, 1985; Ojemann, 1983; Swaab & Fliers, 1985).

Figure 1 shows that the neurological dimension of sexuality may be thought of as occurring in two stages: the sexual orientation stage and the sex-typical behavior stage. As Figures 2 and 3 show, most of the neurological organization surrounding the first stage appears to occur during the 3rd and 4th months of gestation, whereas that for the second stage primarily occurs during the 5th and 6th months.

Although the nature and degree of sex differences in the brain are still being identified, three well-established differences that appear relevant to the first stage of neurological organization are all located in and around the hypothalamus. Specifically, the preoptic anterior nucleus appears to regulate masculine brain functions, such as tendencies to mount in response to various F/dM cues (Feder, 1984; Hart, 1974; McEwen, Davis, Parsons, & Pfaff, 1979; Van de Poll, de Bruin, van Dis, & van Dyen, 1978). Recent autopsies on humans revealed that, on average, this area of the brain was over twice as large in men as in women (Swaab & Fliers, 1985). A comparable sex difference has also been found in rats, except in those with experimentally altered androgen levels (Gorski, Gordon, Shryne, & Southham, 1978).

Two brain areas have been frequently implicated in producing F/dM brain functioning: the ventromedial nucleus (Pfaff, 1980; B. S. Rubin & Barfield, 1980) and the anterior nucleus (Dorner, Docke, & Moustafa, 1968a, 1968b; Whitney & Herrenkohl, 1977). The ventromedial nucleus has been associated most with regulating a cyclic, rather than a tonic, release of sex hormones; the anterior nucleus has been most implicated in controlling receptive responses to mounting attempts.

Behavioral Dimension

Similar to the neurological dimension, the behavioral dimension may be conceived of in terms of two overlapping components: the sexual orientation component and the sex-typical behavior component. The sexual orientation component refers to an individual's choice of sex partners, and the sex-typical behavior component refers to a variety of behavioral patterns that are more characteristic of one sex than the other. Although substantial evidence both in humans and other species indicates that sex-typical behavior patterns are inclined toward either a
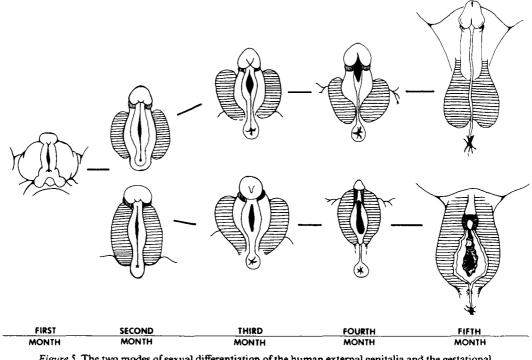
Figure 5. The two modes of sexual differentiation of the human external genitalia and the gestational timing of the differentiation process (adapted from Silber, 1981, p. 148).

feminine or a masculine style during neuro-organization, lifelong conditioning doubtless sometimes accentuates and sometimes blunts the differences resulting from neuro- organization (L. Ellis, 1986). However, we present evidence suggesting that conditioning normally plays even less of a role in determining sexual orientation.

In the next section, we discuss how differences in timing of the four phenotypic dimensions of sexuality and differences in the biochemistry required to induce each dimension specifically influence the expression of sexual orientation.

Experimental Induction of Sexual Inversions in Nonhumans

In the past 30 years, a large array of sexual inversions have been experimentally induced in laboratory animals. We focus here on sexual inversions that primarily involve behavior and the brain (for reports pertaining to morphological inversions, see Beach, Buehler, & Dunbar, 1983; Feder, 1981; Neumann, Elger, & Kramer, 1966; Stechler & Halton, 1982).

Five basic methods for inducing sexual inversions of a neurological-behavioral nature have been documented in laboratory animals: (a) direct perinatal androgen manipulation, (b) pharmacologically blocking or augmenting the perinatal effects of androgens, (c) maternal and neonatal exposure to androgen-depressing emotional stress, (d) the induction of immunity responses to androgens or other hormones involved in sexual differentiation, and, possibly, (e) sexual segregation during childhood. A description of each method follows.

Direct Perinatal Androgen Manipulation

A wide spectrum of inverted sexual behavior patterns have been induced by castrating male rodents at birth or within a few days after birth. The affected behavior patterns tend not to be fully exhibited until the onset of puberty. At that time, instead of mounting receptive females and behaving combatively toward other males, perinatally castrated male rodents show little or no sexual interest in females and often display feminine-like presenting postures when in the proximity of other males (Diehl, Godke, & Day, 1972; Dorner & Hinz, 1968; Ford & Schanbacher, 1977; Thomas & Gerall, 1969). Because the gonads are removed, this procedure requires artificial pubertal activation using a variety of sex hormones as activating agents. With some qualifications required for certain species, both testosterone (Dorner & Hinz, 1968) and estradiol (Baum, 1976) can trigger homosexual responses in these males. How both hormones work can be explained by noting that estradiol appears to be the main organizer and activator of sexual behavior in mammals, regardless of the mammal's sex or sexual orientation (Baum, 1976). However, because testosterone can be converted not only to estradiol, but also to DHT (see Figure 4), which organizes and helps to activate gonadal tissue (e.g., by increasing the sensitivity of the penis), testosterone is probably the best overall activator of both male and female sexual behavior (Ward, 1977).

In order to induce an inversion of the sexual behavior of males using castration, the testes must be removed within the 1st week after birth for most species of rodents (Ford, 1982, 1983; Shapiro & Goldman, 1979; Ward & Renz, 1972). For ani-

mals whose neuro-organization occurs entirely during gestation (e.g., primates), neonatal castration has no sexually inverting effects on behavior (Feder, 1984).

Because it is largely testosterone (which is aromatized to estradiol after entering the nervous system) that is required for an M/dF neuro-organization, castration of females (removal of their ovaries) during neuro-organization does not invert their sexual preferences. Postnatal female castration during the critical period of neuro-organization prevents only the full expression of F/dM behavior after puberty. To masculinize female behavior, testosterone levels must be in the male-typical range during a substantial proportion of neuro-organization. For species in which neuro-organization is still incomplete after birth (e.g., rodents), testosterone injections have been used to diminish and even eliminate postpubertal displays of F/dM receptive behavior in response to a courting male (Barraclough & Gorski, 1962; Beach & Kuehn, 1970; Carter, Clemens, & Hoekema, 1972; Edwards & Burge, 1971; Ward & Renz, 1972). In addition, fairly large doses of testosterone injected just before and/or just after birth (depending on the species) have resulted in female rodents mounting other females and resisting mounting attempts by males to a degree that is quite uncommon among most other females (Baum, 1976; Carter et al., 1972; A. A. Gerall & Ward, 1966; Manning & McGill, 1974; Morali, Carrillo, & Beyer, 1985; Sodersten, 1973). The predominant response these females make to an approaching male is dominating and aggressive, especially if the interactants are strangers.

Two failures to induce homosexual behavior in female rodents have been reported in postnatal testosterone injection experiments (Edwards & Burge, 1971; Whalen, Edwards, Luttge, & Robertson, 1969). However, these failures might be due to low dosages and/or not making injections during critical periods.

Exposure of a female fetus to high testosterone levels is usually accomplished by injecting the hormone into the mother rather than into the fetus. The mother readily transmits it to the fetus via the placenta linking their two blood systems. An extended series of experiments on inducing sexual inversions in rhesus monkeys has shown that, since primate neuro-organization occurs prior to birth, in order to completely invert a primate's behavior (including sexual orientation), intervention must occur prior to birth (Phoenix, 1974). In addition to inverting sexual orientation, primate experiments have disclosed that such sex-typical behavior patterns as dominance-related aggression also can be significantly inverted using similar procedures (Goy, 1978; Phoenix, Jensen, & Chambers, 1983). Specifically, prenatally androgenized genetic female monkeys play rougher and more competitively than do genetic females whose hormonal regimens have not been experimentally disturbed (Goy, 1978).

Blocking or Augmenting the Effects of Androgens With Drugs

Several neurologically active drugs can block testosterone's effects or otherwise interfere with the neuro-organizational actions of sex hormones. Examples include such antiandrogens as medroxyprogesterone acetate (Depo-Provera), flutamide, cimetidine, and cyproterone acetate. These drugs alter the syn-

thesis of androgens, the formation of androgen receptor sites, or the filling of androgen (and related hormonal) receptor sites in the brain (Anand & van Thiel, 1982; Clemens, Gladue, & Coniglio, 1978; Neumann & Steinbeck, 1974). When they are administered to a pregnant mother while her fetus' brain is being sexually organized, offspring are likely to have their postpubertal sexual behavior affected. The best documented effect is the tendency for various antiandrogens to have F/dM effects on the sexual behavior of male rats after the onset of puberty (e.g., presenting and elevating the rear to other males accompanying a failure to mount females; Neumann et al., 1970; Ward, 1972a).

Sex hormone priming at puberty is not required to activate homosexual behavior in these males because they have intact and functioning testes, and their external genitalia normally are entirely M/dF in appearance. Furthermore, their testosterone production appears to be well within the normal male range (Hull, Nishita, Bitran, & Dalterio, 1984, p. 1013).

Many drugs besides these antiandrogens appear to at least partially divert or block masculinization of the nervous system during neuro-organization. The list includes barbiturates, chlorimipramine, diazepam, diethylstilbesterol (DES), marijuana, pargyline, and reserpine (Dorner, 1981; Hull et al., 1984; Reinisch & Sanders, 1982). In addition, progesterone and drugs similar to it—variously called progestins and progestogens (Dorland's Medical Dictionary, 1965, p. 1225) as well as progestational steroids (Bardin, 1983, p. 135)—invert certain aspects of sexual differentiation, normally without affecting others (Diamond, Llacuna, & Wong, 1973; Ehrhardt & Meyer-Bahlburg, 1981, p. 1315; Hull, 1981; Hull, Franz, Snyder, & Nishita, 1980, p. 255; Snyder & Hull, 1980).

Generalizations about the types of sexual inversions produced by progestins, and how their effects occur, are difficult to make. Some of this difficulty derives from inconsistencies in the drugs' classification, which is understandable given the similarities among the various male and female sex hormones themselves (see Figure 4; for the chemical structures of various progestins see Mauvais-Jarvis, 1983, p. 10). In addition, most progestins are capable of binding not only to progesterone receptors, but also to androgen and estrogen receptors (Mauvais-Jarvis, 1983). Overall, the evidence from laboratory experiments has shown that both M/dF and F/dM sexual inversions can be induced with progestins.

Some progestins appear to inhibit the 5α-reduction of testosterone to DHT and/or to compete for androgen receptors, in which case demasculinization occurs (Von Berswordt-Wallrabe, 1983; Wright, Giacomini, Riahi, & Mowszowicz, 1983). Other progestins primarily have M/dF effects at least on genital structures (reviewed by Bardin, 1983). Besides variations among progestins, differences in dosage, timing, and variations in the hormones and enzymes with which progestins sometimes interact, and even the specific genetic makeup of the animal involved, may bear on whether the effect of progestins is primarily M/dF or F/dM (Blaustein & Brown, 1984; Sanders & Reinisch, 1985, p. 176).

According to Hull et al. (1984, p. 1013), any drug that alters dopamine levels in the brain probably affects the sexual differentiation of the nervous system, provided that it is administered

in fairly high dosages while the brain is undergoing neuro-organization (see also Dorner, Hecht, & Hinz, 1976).

The perinatal administration of at least one drug—pyridostigmine—appears to specifically facilitate M/dF neuro-organization (Dorner, 1981, p. 115). In addition, studies with rats indicate that some drugs, such as alcohol, have dF/dM effects on the brains (and, thereby, behavior) of both sexes (McGivern et al., 1984).

Maternal or Neonatal Exposure to Emotional Stress

Another method for inducing inverted sexual behavior patterns involves subjecting the neonate, or its mother while pregnant, to severe emotional stress. The most common stressing methods have involved repeatedly confining the mothers for several hours at a time to an intensely lighted enclosure while restraining her inside a narrow plastic tube.

One line of evidence that maternal stress during pregnancy might affect the behavior of offspring came from discovering that stress causes depressed testosterone production in a variety of species (Bernstein, Gordon, & Rose, 1983; F. L. Moore & Zoeller, 1985). Biochemically, this depressing effect is largely triggered by elevated levels of such stress hormones as adrenocorticotropic hormone (ACTH), corticosterone, cortisol, and epinepherine (Stechler & Halton, 1982; Ward, 1984), all of which appear to antagonize the synthesis of testosterone.

Because these stress hormones are carried in the blood, they cross the placenta, and thereby tend to antagonize fetal testosterone production. Ward (1972b) and her associates (Meisel, Dohanich, & Ward, 1979) have found that subjecting pregnant rats to stress during the third trimester results in at least partially inverted sexual orientations of the male offspring, usually with few significantly inverting effects on other sex-typical behavior patterns. Ward (1974, 1977) has produced both homosexual and bisexual male rats by exposing the mother to stress for about 1 week just before delivery, and similar results have been reported by Dahlof, Hard, and Larsson (1977), Whitney and Herrenkohl (1977), Gotz and Dorner (1980), and Rhees and Fleming (1981; for a failure to replicate, see Chapman & Stern, 1979).

Evidence that this and possibly other forms of sexual inversions are caused by the depressing effects of maternal stress on fetal testosterone production comes from experiments with rats showing that male fetuses whose mothers had been stressed just before birth had lower plasma testosterone levels at birth than male fetuses whose mothers had not been stressed during pregnancy (Dorner, 1979; Stahl, Gotz, Poppe, Amendt, & Dorner, 1978).

Also relevant is evidence that the sex drive of males born to mothers stressed during pregnancy was not depressed (Dahlof, Hard, & Larsson, 1977; Whitney & Herrenkohl, 1977). The only well-documented difference in their sexual behavior surrounds their consistent tendency to sexually present to other males and often to exhibit few or no attempts to mount females.

Finally, in regard to maternal stress, recent evidence indicates that female offspring also may have some aspects of their sexual behavior permanently altered as well, although probably to a lesser degree than males. The effect seems to incline females carried by stressed mothers toward somewhat greater sexual re-

ceptivity in general, although their orientation per se did not appear to be inverted (Politch & Herrenkohl, 1984).

Immunity Against Biochemicals Required for Sexual Differentiation

Immunity refers to the tendency for an organism's white blood cells to chemically attack foreign substances by building antibodies to those substances.

A recently developed method for inverting sexual orientation in laboratory animals involves the induction of an immune response to one or more of the biochemicals necessary for sexual differentiation of the brain. Either the mother's or the individual animal's own immune system is induced to regard one of the biochemicals required for sexual differentiation as a foreign substance. The immune system then prevents sexual differentiation from taking place (or at least from taking place completely) by breaking down the "foreign chemicals" required to carry it out. Antibodies are known to exist for LH, luteinizing hormone-releasing hormone (LHRH), CG, and nearly all of the estrogens and androgens (Breuer & Nieschlag, 1975).

We describe two examples of sexual inversions induced in laboratory animals with the aid of the immune system. First, Bidlingmaier, Knorr, and Neumann (1977) induced an immunity response to testosterone in female rabbits who were subsequently mated and impregnated. The male offspring of these mothers had genital structures, both internally and externally, that were distinctly F/dM, with the exception of the testes themselves, which were well formed and fully capable of producing male-typical levels of testosterone.

Apparently, antibodies to testosterone crossed the placenta from the mother while all of the genital structures other than the testes (which form first) were being formed; then these antibodies quickly saturated the fetus' blood system. It is noteworthy that the amount of testosterone in the blood of the F/dM genetic males at birth was much higher than for control males. The reason appears to be that the antibodies circulating in the blood were binding to the testosterone and thereby preventing the hormone from entering the brain.

Testosterone production is under hypothalamic-pituitary feedback control. When testosterone and other sex hormones fail to reach the hypothalamus in sufficient quantities, the hypothalamus dispatches LHRH to the pituitary; the pituitary then dispatches LH to the testes, resulting in increased testosterone production. Therefore, for these genetic males, above average male-typical quanitities of testosterone were being synthesized and emptied into the blood system, but none of the testosterone was feeding back to the hypothalamus because it was being bound to testosterone antibodies in the fetus' blood system. Because the hypothalamus was not receiving testosterone, it continued to send LHRH to the pituitary, and the pituitary continued to dispatch LH to the testes, resulting in extraordinarily high testosterone production in these individuals.

Unfortunately, nothing was determined about the sexual preferences of these rabbits, because they were killed shortly after birth so their internal sex organs could be examined. Nevertheless, the fact that the hypothalamus was not receiving testosterone makes it all but certain that these genetic males would have largely preferred sexually interacting with males when they

reached sexual maturity. Whether or not this constitutes an instance of homosexuality depends on one's definition. Even though they were genetic males, the fact that their genitals had an F/dM appearance means that they might be better classified as phenotypic females.

The other report of the use of the immune system to produce sexual inversions was by Kalcheim, Szechtman, and Koch (1981). They injected male rats during the first 3 days after birth with synthetic antibodies to LHRH. On reaching sexual maturity, these males both mounted females and exhibited lordosis responses to mounting efforts by other males to an unusual degree. Because LHRH provides an essential link in regulating testosterone production, and the brains of rats are still not completely sexually differentiated at birth, sexual orientation should be at least partially inverted. This would be a clear example of bisexuality, because the sexual orientation apparently was only partially inverted; in all other respects, these rats were males.

For two other reports of at least partial immunity-induced sexual inversions, see Goldman and Mahesh (1970) and Goldman, Quadagno, Shryne, and Gorski (1972).

Sexual Segregation

Studies among mammals have shown that a lack of social experience with peers during childhood nearly always results in sexual ineptitude in adolescence and adulthood, especially among males (Dunlap, Zadina, & Gougis, 1978; H. D. Gerall, Ward, & A. A. Gerall, 1967; C. L. Moore, 1985, p. 38). A key determinent of this ineptitude appears to surround the lack of childhood play opportunities during which animals gradually learn (a) not to fear, and at the same time, not to frighten other conspecifics when in close proximity to them; and (b) how to approach and fondle conspecifics in ways that elicit cooperative responses, instead of withdrawal or even attack responses. Sackett (1968) demonstrated that, despite their sexual ineptitude, male rhesus monkeys who had been reared in isolation still showed signs of being sexually aroused by the sight of female peers. This suggested that a heterosexual orientation per se was not disrupted by social isolation, but merely the social skills needed to express it.

Two recent experiments with monkeys have been reported in which inverted tendencies to mount and present appear to have been either induced or at least augmented by manipulating social environmental variables. Goldfoot, Wallan, Neff, McBrair, and Goy (1984) produced tendencies for male rhesus monkeys to present to other males and for females to mount other females prior to puberty by rearing them in unisex groups. When these unisex-reared monkeys were finally introduced into sexually integrated enclosures late in childhood, they rarely made sexual overtures to (in the case of males) or accepted sexual overtures from (in the case of females) members of the opposite sex. Goldfoot et al. did not report on postpubertal behavior, but subsequent research by D. A. Goldfoot (personal communication, May 1, 1986) revealed that with continued opportunity to interact with the opposite sex, most of these monkeys' sexual behavior is increasingly heterosexually oriented. The awkwardness of the sexual overtures and/or receptive postures of these monkeys compared to monkeys reared with peers of both sexes

may reflect a general uneasiness with being in close proximity to members of the opposite sex more than a lack of heterosexual orientation. Nevertheless, at this point, strict behavioral criteria of sexual orientation suggest that an experimental procedure may have been identified for inducing bisexuality in primates simply by manipulating social environmental variables after birth (for similar results with rats, see Jenkins, 1928).

While being careful not to confuse primate appeasement-submission gestures with sexually motivated presenting postures (Wickler, 1969), future studies of this phenomenon should try to determine what neurochemical events may be mediating these sexual inversions.

The second study is a complex experiment by Ward and Reed (1985) involving male rats whose mothers were stressed during pregnancy in ways similar to those Ward and her associates used in experiments discussed previously. In this new study, some maternally stressed (MS) males were reared exclusively with other MS males, some were reared exclusively with males whose mothers had not been stressed during pregnancy, others were reared exclusively with females, and still others were reared in isolation. The same four rearing conditions were applied to samples of male rats whose mothers had not been stressed during pregnancy, yielding a total of eight groups of male rats, presumably otherwise equivalent. On reaching puberty, these rats were all tested for displays of both masculine mounting–ejaculatory patterns and for lordosis responses to unfamiliar stimulus rats. Overall, the results supported earlier findings that maternal stress has sexually inverting effects on male rats. Nevertheless, they also indicated that social rearing conditions can have similar effects both independent of and especially in combination with maternal stress. Ward and Reed speculate that the tendencies for social rearing conditions to invert the sexual orientation of males may be mediated by a lowering of testosterone levels while they are still undergoing neuro-organization (which for rats appears to extend for about 4 weeks after birth).

In summary, all aspects of mammalian anatomy, physiology, and behavior can be sexually inverted. Most, if not all, of the methods for causing sexual inversion involve altering androgen and/or other sex hormone levels while the various parts of the body are sexually differentiating. As for sexual orientation, the crucial body part appears to be the brain.

Causes of Sexual Inversions in Humans

Even though sexual inversions are not induced in humans for experimental purposes, evidence has accumulated that many of the experiments conducted with laboratory animals have close parallels in humans. We show that at least four of the five methods used in laboratory animals to induce inversions of sexual orientation appear to have similar effects in humans. The evidence, summarized in Table 1, has been derived from both human and nonhuman research. Table 1 shows the most likely instigating causes of sexual inversions, the genetic sex affected, and the phenotypic dimension(s) affected.

By *instigating cause* we mean the first major event that interrupts the usual transcription of an XY karyotype into a masculine phenotype or an XX karyotype into a feminine phenotype. The hypothesized instigating causes of human sexual inversions have been grouped into the categories of genetic-hormonal,

Table 1
Types of Sexual Inversions Known or Suspected, Their Apparent Instigating Causes, and Their Main Phenotypic Manifestations

Inversion	Instigating cause(s)	Genetic sex affected	Phenotypic dimensional effects		Neuro-organizational and behavioral dimensions	
			Genital dimension	Nongenital morphological dimension	Sexual orientation	Sex-typical behavior patterns
Genetic-hormonal causes						
5α-reductase deficiency	Autosomal failure to synthesize 5α-reductase (which is required to convert testosterone to DHT (see Figure 3)	Male	F/dM–F/M before puberty, M/dF–F/M after puberty	F/M before puberty, M/dF–F/M after puberty	Preference for F/dM-appearing sex partners	M/dF–F/M
Androgen insensitivity syndrome	Autosomal failure to metabolize testosterone in the usual way due to an insufficient number of appropriate androgen/estradiol receptor sites	Male	Usually complete F/dM	Usually F/dM	Preference for M/dF–F/M-appearing sex partners	F/dM–F/M
Congenital adrenal hyperplasia syndrome	Autosomal mutation causes testosterone synthesis by the adrenal glands in the low male range	Female	M/dF externally, F/dM internally	F/M	Above average preference for F/dM–F/M-appearing sex partners	M/dF–F/M
Faulty testosterone synthesis	Various autosomal failures to synthesize one or more of the enzymes needed to convert cholesterol to testosterone	Male	Various forms of F/dM–F/M phenotypic traits			
Pharmacological causes Perinatal exposure to:						
Medroxprogesterone acetate, flutimide, cimetidine, and cyproterone acetate[a]	Reduced testosterone synthesis	Male	F/dM–F/M depending on timing and dosage	F/dM–F/M depending on timing and dosage	Above average preference for M/dF–M/F-appearing sex partners	F/dM–F/M
Various progestins, progestigens, etc.	Sometimes augments testosterone synthesis	Female	F/dM–F/M depending on timing and dosage	F/dM–F/M depending on timing and dosage	Above average preference for F/dM–F/M-appearing sex partners	F/dM–F/M
	Sometimes impedes testosterone synthesis	Male	Various forms of F/dM–F/M phenotypic traits depending on timing and dosage			
Diethylstilbesterol (DES)	Probably augments testosterone synthesis and/or functions as estradiol	Female	Unknown	Unknown	Above average preference for F/dM–F/M-appearing sex partners	Unknown

Table 1 (continued)

Inversion	Instigating cause(s)	Genetic sex affected	Phenotypic dimensional effects		Neuro-organizational and behavioral dimensions	
			Genital dimension	Nongenital morphological dimension	Sexual orientation	Sex-typical behavior patterns
Pargylin, reserpine, chlorimipramine, barbiturates, diazepam, or marijuana	Inhibits testosterone synthesis	Male	Varying degrees and forms of F/dM-F/M phenotypic traits depending on timing and dosage			
Alcohol	Probably interferes with the synthesis of all sex hormones	Both sexes	Varying degrees of dF/dM phenotypic traits depending on timing and dosage			
Stress-induced causes						
Perinatal exposure to prolonged and intense stress	Prevents synthesis of testosterone and its conversion to estradiol via fetal exposure to high maternal levels of stress hormones	Male	M/dF	M/dF-F/M	Preference for M/dF-F/M-appearing sex partners depending on extent and timing of stress	Unknown
Immunological causes						
Prenatal immunity to one or more sex hormones	Mother's and/or fetus' immune system synthesizes antibodies to one or more of the hormones needed for sexual differentiation	Male	M/dF	M/dF-F/M	Above average preference for M/dF-M/F-appearing sex partners	Unknown
Postnatal social environmental causes						
Childhood rearing in exclusive contact with one's own sex	Unknown	Male	M/dF	M/dF	Above average preference for M/dF-M/F-appearing sex partners	Unknown
	Unknown	Female	F/dM	F/dM	Above average preference for F/dM-F/M-appearing sex partners	Unknown

Note. F/dM = feminine, demasculinized; M/dF = masculine, defeminized; F/M = sexually ambiguous; and dF/dM = lacking sexual differentiation. DHT = dihydroxytestosterone.
a Cyproterone acetate and medroxyprogesterone are sometimes classified as progestins.

pharmacological, stressful, immunological, and social environmental.

Genetic-Hormonal Causes of Human Sexual Orientation

As Table 1 shows, four types of genetic mutations probably cause sexual inversions in humans. They all seem to involve errors in autosomal genetic programs, and three of the four affect only genetic males.

5α-reductase deficiency. One genetic-hormonal cause of male sexual inversions involves a failure to produce 5α-reductase. Although 5α-reductase is normally present in both sexes, its only known function is in males, where it converts testosterone to DHT (see Figure 5). Because DHT is required to masculinize the external genitals (Savage et al., 1980), when 5α-reductase is absent, the genitals develop an F/dM appearance no matter how much testosterone the testes produce.

Because DHT appears to play only a minor role in brain differentiation (see Hull et al., 1980), an inability to produce 5α-reductase does not greatly impede brain (and thereby behavioral) masculinization in these genetic males. In fact, the deficiency may facilitate brain masculinization to some degree by increasing the amount of testosterone available for crossing the blood-brain barrier and subsequently aromatizing to estradiol.

Genetic males who lack the ability to convert testosterone to DHT were first clinically identified in the 1970s (Fisher et al., 1978; Imperato-McGinley, Guerrero, Gauthier, & Peterson, 1974; Saenger et al., 1978; Savage et al., 1980).

As one would expect, these individuals, at least until recently, were reared as females because they usually had F/dM genitals. Despite their having been reared as females, however, virtually all of them exhibited a preference for female sex partners at puberty (Delozier & Engel, 1982; Imperato-McGinley, Peterson, Goutier, & Sturla, 1979; R. T. Rubin, Reinisch, & Haskett, 1981). In addition, some degree of masculinization of external genitals and of secondary sex characteristics occurs at puberty, presumably due to the direct action of testosterone, rather than DHT. The clitoris greatly enlarges at puberty, the vaginal opening sometimes partially fuses shut, the scrotal tissue sags to accomodate partially descended testes, and the breasts do not enlarge.

The 5α-reductase deficiency syndrome is an interesting example of sexual inversion. In nearly all cases, the sexual orientation of these individuals is consistent with their genetic sex (male), and not with their sex of rearing. However, because humans are accustomed to judging sex not on the basis of chromosome configuration, but on the basis of morphology—at least in the past—these individuals would likely be considered female homosexuals. One response to this confusion among villagers in the Dominican Republic, where this syndrome is often found, has been to call those affected *machihembra* ("man-woman").

Androgen insensitivity syndrome. Another genetically instigated cause of sexual inversions involving genetic males is the androgen insensitivity syndrome (or the testicular feminization syndrome). Persons with this syndrome develop testes that produce normal or above normal male quantities of testosterone, but they lack androgen receptor sites to bind to the hormone in

a normal way. The degree to which the syndrome is manifested depends on the quality and quantity of the available receptor sites. In the most extreme cases (called complete androgen insensitivity), affected children appear to be females, and are reared as such. At puberty, all of the usual secondary feminine sex characteristics appear except for menstruation. The determination that they are genetic males often is made for the first time when menarche fails to appear even quite late in adolescence. Longitudinal studies of affected individuals have noted a striking absence of most male-typical behavior and interests (Ehrhardt, 1975; Ehrhardt, Epstein, & Money, 1968; Money, 1969; Money, Ehrhardt, & Masica, 1968).

The androgen insensitivity syndrome also illustrates how the concept of heterosexuality–homosexuality occasionally is difficult to apply, as persons with this syndrome are genetic males with all F/dM phenotypic traits. By most conventional criteria, they would be considered heterosexual females (Carlson, 1977, p. 308).

What appears to be the same genetic condition has been found in rats and mice (Von Berswordt-Wallrabe, 1983, p. 111; Meaney, Stewart, Poulin, & McEwen, 1983) and in baboons (Bielert, 1984). As with humans, the affected animals tend to be largely F/dM in all phenotypic respects, except that they never menstruate or conceive.

Congenital adrenal hyperplasia (CAH) syndrome. So far the only genetically caused sexual inversion that has been well documented in genetic females is the CAH syndrome (also called the adrenogenital syndrome). Normally the adrenal glands produce only small quantities of androgens and other sex hormones, and instead mainly synthesize such stress hormones as adrenalin and cortisol. However, in persons with CAH, a genetically controlled enzyme deficiency causes testosterone production in male-range quantities by the fetal adrenal glands instead of cortisol (Money & Daléry, 1976). In genetic females, this results in varying degrees of genital masculinization (Reinisch, 1976). Despite surgical correction of the genitals at birth and rearing as females, longitudinal studies have found that the behavior patterns of persons with CAH tend to be unusually masculine for females. Compared to most females, they are more apt to prefer competitive sports, and are less likely to prefer playing with dolls and dressing in feminine clothing. They also report fewer fantasies about romance and marriage (Ehrhardt & Baker, 1974; Ehrhardt et al., 1968; Ehrhardt, Evers, & Money, 1968; Money & Ehrhardt, 1972; Shepherd-Look, 1982; Walker & Money, 1972). Their mannerisms and gestures also have been described as more M/dF than those of most females (Muller, Kraus-Orlitta, Dirlich-Wilhelm, & Forster, 1983).

The significant degree of overlap in the timing of genital masculinization and the first stage of neuro-organization (see Figure 2) leads to the prediction that, at puberty, the preference for sex partners by these genetic females will tend toward ambiguity. Consistent with that expectation, over one-third of those persons with CAH syndrome express a preference for sexual relations with females or at least with both sexes (Ehrhardt, 1978, p. 536; Money & Schwartz, 1977; Money, Schwartz, & Lewis, 1984). In addition, because high testosterone synthesis continues for these women on through the second stage of neuro-organization, it is not surprising that their sex-typical behavior is at least partially inverted as well (reviewed by Ehrhardt, 1978).

Faulty testosterone biosynthesis. This fourth genetic cause of sexual inversions affects only genetic males. It involves one or more of the genes that regulate the synthesis of the five enzymes needed to convert cholesterol to testosterone, as Figure 4 shows (Grumbach, 1979, p. 58). Four of these biosynthesis defects are transmitted as autosomal recessive traits, the fifth is apparently under the control of an X-linked recessive gene. The nature of the inversions caused by faulty testosterone biosynthesis depends on exactly which enzyme is not being produced. As more detailed clinical information is collected about these enzymic deficiencies, several separate syndromes may eventually be identified. For instance, if 17β-hydroxylase is not produced, the individual is likely to suffer a wide variety of medical maladies as well as some degree of genital inversion (Grumbach, 1979). Although reports of neurological, and thereby behavioral, inversions associated with faulty testosterone biosynthesis were not found, there is every reason to expect that they will be found.

Pharmacological Causes of Human Sexual Inversions

As already discussed for laboratory animals, the evidence that drugs can cause sexual inversions, including inverted sexual orientation, is strong, even though the detailed nature of those inversions is not yet clear (Ehrhardt, 1978). The list of drugs that can induce sexual inversions is already fairly extensive and is bound to lengthen as new drugs are developed and more is learned about how they affect various bodily processes.

In the absence of controlled experiments, it is difficult to demonstrate causal links between specific drugs and sexual inversions in humans. However, one set of drugs, the progestins (or progestogens), have been repeatedly implicated as having sexually inverting effects, even though the majority of human fetuses exposed to at least modest levels of these substances exhibit no evidence of sexual inversions (Ehrhardt & Meyer-Bahlburg, 1980, p. 184).

Maternal progesterone levels rise rapidly soon after conception and appear to play an important role in maintaining pregnancy (Riddick, Daly, Rosenberg, & Maslar, 1983; Rothchild, 1983). Especially in the 1950s, progestins were fairly widely prescribed to women at risk for spontaneous abortion (Ehrhardt, Meyer-Bahlburg, Feldman, & Ince, 1984, p. 458); however, their effectiveness in preventing miscarriages has been questioned in recent years (Ehrhardt & Meyer-Bahlburg, 1980, p. 186; Sureau et al., 1983, p. 247).

Most pertinent to the present discussion are reports of possibly higher than normal incidences of ambiguous external genitals in genetic females born to mothers taking various types of progestins during pregnancy (Hampson, 1965; Wilkins, 1960). Despite the fact that these children nearly always had their genitals surgically feminized in infancy and were reared as females, their sex-typical behavior patterns have been found to be unusually M/dF (Ehrhardt, 1969; Ehrhardt & Money, 1967; Money, 1969). Even as adults, these individuals have been found to have career interests and levels of self-assertiveness that are more similar to male patterns than to female patterns (Reinisch, 1977, 1981; see also Kolata, 1978). Nevertheless, postpubertal follow-ups have not revealed evidence of significant homosexu-

ality, or even bisexuality, among these women (Ehrhardt & Meyer-Bahlburg, 1980, p. 184).

Confounding the picture of the effects of at least synthetic progesterones on sexual inversions is evidence that some antiandrogen drugs that are sometimes classified as progestins or progestogens (e.g., medroxyprogesterone acetate) may have demasculinizing effects on the genitals of male fetuses (Aarskog, 1971; see also Ehrhardt & Meyer-Bahlburg, 1980, p. 185), particularly when taken in combination with the synthetic estrogen DES (I. D. Yalom, Green, & Fisk, 1973). Along similar lines, lesbianism was recently found to be more common among women whose mothers had taken DES during pregnancy (to lessen the risk of miscarriage) than among women whose mothers had not (Ehrhardt et al., 1985). Thus, just as in nonhuman mammals, synthetic (and possibly natural) progesterones appear to have both masculinizing and feminizing effects, depending on the dosage, timing of administration, and other substances (both natural and synthetic) with which they may interact. This unsettled state of affairs can be understood by again noting that progesterone is a necessary biochemical in the production of both androgens and estradiol (see Figure 4), and the fact that timing and dosage factors are likely to be key elements in determining their effects. Despite these complications, evidence from studies with humans and nonhumans indicates that manipulating these hormones can have sexually inverting effects on all four phenotypic dimensions.

Stress-Induced Causes of Human Sexual Inversions

As indicated earlier, laboratory animal research strongly supports the conclusion that stress during pregnancy can invert sexual orientation in male offspring. Two studies with humans point toward a similar conclusion, although the nonexperimental nature of their designs makes their results tenuous.

In the first study, Dorner et al. (1980) found that, among males born in Germany between 1934 and 1953, an unusually high proportion of homosexuals were born during and immediately after the Second World War (i.e., between 1941 and 1946). Without denying other post hoc explanations, Dorner et al. noted that this was a time of unusual stress for most German citizens, and perhaps especially so for pregnant women.

The second study by Dorner, Schenk, Schmiedel, and Ahrens (1983) involved asking a group of mothers of male homosexuals, bisexuals, and heterosexuals to recall any stressful episodes they may have experienced during pregnancy (e.g., deaths of close relatives, divorces, separations, traumatic financial or sexual experiences, feelings of severe anxiety). As the stress-induced hypothesis would lead one to expect, nearly two-thirds of the mothers of the male homosexuals, compared to one-third of the mothers of the bisexuals and less than 10% of the mothers of the heterosexuals, were able to recall such episodes.

Obviously, studies such as these can be criticized on a number of methodological grounds and would need to be carefully replicated and expanded before being considered as supporting a maternal stress explanation of male homosexuality in humans, but they are certainly suggestive in light of the experiments with rodents reviewed earlier.

Finally, before leaving the issue of stress-induced sexual inversions, it is important to underscore that, probably for genetic

reasons, animals vary considerably in their propensity to re-
spond to stress in ways that would significantly inhibit androgen
production. Thus, even if research confirms the stress-induced
hypothesis in humans, there probably is individual variation
among mothers in how much stress they can endure before their
offspring are significantly affected.

Immunity-Induced Causes of Sexual Inversions in Humans

No direct evidence of immunological causes of sexual inver-
sions in humans yet exists, despite the experiments discussed
earlier showing that at least partial sexual inversions, including
bisexuality, can be immunologically induced in laboratory ani-
mals. Nevertheless, there is indirect evidence that sexual inver-
sions (including those surrounding sexual orientation) could
occur in humans through immunological processes.

First, two recent reviews have concluded that sex hor-
mones—especially testosterone—interact with the immune
system in complex, and probably quite consequential, ways
(Geschwind & Galaburda, 1985; Grossman, 1985). In particu-
lar, a number of hormones normally secreted during pregnancy
seem to be vital for inhibiting the mother's immune system for
producing antibodies to the fetus' foreign cells (a classic exam-
ple being the Rh— syndrome). Presumably, both for genetic and
environmental reasons, some women may produce inadequate
amounts of these hormones, and thereby their immune systems
might chemically destroy some of the substances vital for sexual
differentiation.

Second, growing evidence implicates the presence of sub-
stances other than sex hormones that circulate in women in
higher amounts during pregnancy than at other times to help
suppress immune reactions to fetal cells (Muchmore & Decker,
1985). Maternal immune reactions to fetuses are increasingly
suspected as a cause of spontaneous abortions (Fainstat & Bhat,
1983), with some researchers suggesting that progesterone in
particular may partially suppress the mother's immune reac-
tions (Siiteri et al., 1977).

Another consideration is the fetus' sex. The more foreign the
fetus' cells are, the greater the risk that the mother's immune
system will produce antibodies to them. Male fetuses introduce
a wider range of foreign substances to which the mother may
gradually acquire immune responses than do female fetuses.
Because the mother's immune response may not be fully ac-
quired until the second or third pregnancy, later-born males
may be especially at risk.

Social Environmental Causes of Human Sexual Inversions

As noted earlier, two recent studies have shown that some
degree of homosexuality or at least bisexuality may result from
rearing rodents and possibly monkeys in unisex peer groups.
Whether or not a similar degree of restricted access to members
of the opposite sex could alter human sexual orientation re-
mains to be determined. It seems relevant to note that many
therapists who have tried to reorient male homosexuals have
emphasized the need to reduce their fear of approaching fe-
males in sexual ways (e.g., Feldman & MacCulloch, 1971, p.

15). However, if exclusive same-sex childhood rearing is re-
quired to invert sexual preferences, it is unlikely that these ex-
periments have much relevance to homosexuality outside the
laboratory, either in humans or in other animals.

To summarize the human evidence, although the kinds of
controlled experiments carried out with a variety of other
mammalian species have not been replicated in humans, "natu-
ral and inadvertent experiments" relevant to the process of sex-
ual differentiation have been documented. Recent studies of
these "experiments" point toward the same general conclusions
as have been reached for other mammals: Sexual orientation is
mainly the result of neurological factors that are largely deter-
mined prenatally, even though they do not fully manifest them-
selves until adolescence or adulthood.

A Gestational Neurohormonal Theory of Human Sexual Orientation

The following theory of sexual orientation postulates the in-
volvement of several interacting variables and purports to ex-
plain not only homosexuality and bisexuality, but also hetero-
sexuality. Its most basic premise is that human sexual orienta-
tion is determined in essentially the same way as in all other
mammals. As has been experimentally demonstrated, even
though mammalian sexual orientation tends to be guided by
complex genetic programs, the process is biochemically deli-
cate and can be diverted by a variety of environmental factors.

According to the present theory, sexual orientation in all
mammals is primarily determined by the degree to which the
nervous system is exposed to testosterone, its metabolite estra-
diol, and to certain other sex hormones while neuro-organiza-
tion is taking place. If the levels of these hormones are in the
typical female range during the first stage of neuro-organiza-
tion, on sexual maturity the individual will prefer sexually in-
teracting with conspecifics with an M/dF appearance. On the
other hand, if the level of these hormones in the brain is in the
typical male range during the first stage of neuro-organization,
the postpubertal preference will be for F/dM-appearing sex
partners.

For humans, sexual orientation appears to be primarily de-
termined roughly between the middle of the 2nd and the end
of the 5th month of gestation (Ehrhardt et al., 1984, p. 459).
Overlapping the latter part of that period and extending beyond
it for at least 2–3 more months is the time when neuro-organiza-
tion for a number of sex-typical behavior patterns seems to oc-
cur (this is identified as the second stage of neuro-organization
in Figure 1). *Sex-typical behavior* is behavior that is more com-
mon or intense in one sex than in the other, regardless of the
cause (Reinisch et al., 1979, p. 218). Several of the same sex-
typical behavior patterns that have been found to be nearly uni-
versal in humans have been documented in a wide variety of
mammalian species, and laboratory experiments have shown
that these sex differences usually can be eliminated by manipu-
lating neurohormonal factors, especially during neuro-organi-
zation (L. Ellis, 1986). For this reason, we hypothesize that
many sex-typical behavior patterns in humans substantially re-
flect the effects of neurohormonal factors. The mode of trans-
mission for sex-typical behavior patterns may involve fairly di-
rect programming of the brain to emit more or less automatic

responses to stimuli, or, more likely, merely programming varying neurological capacities, propensities, and preferences to learn some behavior patterns more readily than others. However, for sexual orientation, we are led to believe that learning plays much less of a determining role than for sex-typical behavior patterns generally, except in the sense of influencing the exact environmental contexts within which the orientation is expressed.

Deductions From the Gestational Neurohormonal Theory

This theory allows a number of testable hypotheses to be derived, several of which have already been tested fairly extensively.

Homosexuality should primarily be a male phenomenon. The theory predicts that homosexuality should be more common among males than females for two reasons. First, because all mammals are fundamentally female (Von Berswordt-Wallrabe, 1983, p. 110), it is only by inserting the Y chromosome into the mammalian genome that masculinity in any form is genetically possible. Even when the Y chromosome is present and functioning, however, its effectiveness is contingent on several normally inactive genes on autosomes interacting with genes on the Y chromosome to produce M/dF effects. This fundamental feature of mammalian sexual differentiation virtually insures that more inversions will be found in genetic males than in females.

Second, because only female mammals can gestate offspring, natural selection presumably has much more stringently favored females for their direct contributions to reproduction than males (Durden-Smith & deSimone, 1983, p. 116). Presumably, this has resulted in wider variability in most traits in males than in females, including those surrounding sexual orientation (see E. O. Wilson, 1978, p. 145).

The available evidence supports the idea that homosexuality is more common among males than among females, both in humans worldwide (Cory, 1951, p. 88; Davenport, 1965, p. 199; Hunt, 1974, p. 315; Hesnard, 1933, p. 189; Roth & Ball, 1964), and in all other mammalian species thus far studied (Gadpaille, 1972).

Homosexuals should have higher frequencies of other sexual inversions than heterosexuals. Neurohormonal events surrounding perinatal influences on sexual orientation can occur without affecting other aspects of sexual differentiation. However, because the determinants of sexual orientation—both in terms of the timing and the biochemistry involved—overlap organizational influences on the formation of muscle and skin tissue, as well as neuro-organizational foundations of sex-typical behavior patterns, the theory predicts a significant correlation between homosexuality (and, to a lesser degree, bisexuality) and other forms of morphological and behavioral inversions.

Considerable evidence supports this deduction. Many studies have found that, on average, F/dM mannerisms or interests are considerably more common in male homosexuals than in male heterosexuals, although, as the theory would indicate, some male homosexuals do not display a significant degree of F/dM mannerisms or intersts, and some male heterosexuals do (A. P. Bell, Weinberg, & Hammersmith, 1981; Blanchard, McCon-

key, & Steiner, 1983; Friedman & Stern, 1980; Green, 1979; Harry, 1983; Money & Russo, 1979; Saghir & Robbins, 1973; Sanders, Bain, & Langevin, 1985; Thompson, Schwartz, McCandless, & Edwards, 1973; Whitam, 1980; Zuger, 1984). Tendencies toward greater than average degrees of sexual inversions have been reported even in homosexual children prior to their manifesting a sexual orientation (Grellert, Newcomb, & Bentler, 1982; Whitam, 1977). Only two studies failed to find significant average differences, and in both cases, the differences still were in the predicted direction (Heilbrun & Thompson, 1977; Schlaich, 1976).

Studies of female homosexuals and bisexuals, likewise, have found their mannerisms and interests, on average, to be more M/dF than among female heterosexuals (Heilbrun & Thompson, 1977; Kaye et al., 1967; LaTorre & Wendenburg, 1983; Oldham, Farnhill, & Ball, 1982; Spence & Helmreich, 1978; Thompson et al., 1973).

Relationships between parents and homosexual offspring often may be strained and/or assume some cross-sex characteristics. Several studies have shown that not only do parents influence the behavior of their offspring, but, from birth onward, offspring influence the behavior of their parents (e.g., R. Q. Bell, 1968; R. Q. Bell & Harper, 1977; Friedrich & Boriskin, 1976; Reichler & Schopler, 1971). If so, and if the morphological appearance, mannerisms, and interests of homosexuals tend to be inverted even before puberty, parents are likely to respond somewhat differently to homosexual offspring than to heterosexual offspring, even before their orientation per se is manifested.

Presumably the differences in parental responses would be of two types: either they would treat a homosexual offspring as they would a member of the opposite sex or they would respond with irritation toward the inverted mannerisms and interests.

Evidence that parents—at least mothers—tend to treat male and female offspring differently comes from studies of humans (Kagan & Freeman, 1970; Lewis, 1972; Moss, 1967; Todd & Palmer, 1968) and several nonhuman species (Bixler, 1980; Jensen, 1969; Meier & Schutzman, 1968; Mitchell, 1968). In all cases, mothers tend to be more punitive toward, and less protective of, their male infants than their female infants. Our theory suggests that because of their partially inverted sex-typical behavior prior to puberty, offspring who will be homosexual as adults elicit less extreme sex-related differences in treatment from their mothers than do heterosexual offspring. Consistent with that view, studies have reported that the levels of protectiveness and punitiveness exhibited by mothers toward homosexual offspring tend to be intermediate to those exhibited toward heterosexual offspring during childhood, especially for male homosexuals (Bieber et al., 1962; Chang & Block, 1960; Evans, 1969; O'Connor, 1964; Saghir & Robins, 1973; Snortum, Gillespie, Marshall, McLaughlin, & Mosberg, 1969; Stephan, 1973; Thompson et al., 1973).

In humans, males tend to assume a more active role in child rearing than do males of most other primate species, and, in doing so, human fathers seem to be even more prone than mothers to respond differently toward male offspring than toward female offspring (Bronfenbrenner, 1967, p. 389; Mallen, 1983; Margolin & Patterson, 1975). For this reason, fathers might feel especially uneasy with inconsistencies between how

they expect their offspring to behave as males or females and what they actually observe. In this regard, several studies have found greater parental hostility toward homosexual boys than toward heterosexual boys, even during early childhood, especially by fathers (Bieber et al., 1962; Buhrich & McConaghy, 1978; Chang & Block, 1960; Evans, 1969; Mallen, 1983; Saghir & Robins, 1973; Sipova & Brzek, 1983; Snortum et al., 1969; Stephan, 1973; Thompson et al., 1973; for failures to replicate, see Greenblatt, 1967; Siegelman, 1974).

The present theory would explain these observations as reflecting parental responses to the partially inverted mannerisms, interests, and behavior of homosexuals relative to heterosexuals. This concurs with conclusions by Evans (1969) and Blanchard, McConkey, and Steiner (1983) that such parental responses are not a significant cause of sexual orientation, but largely the result of the partially inverted appearances and mannerisms during childhood that often correlate with an inverted sexual orientation in adulthood.

A further test of this deduction would involve comparing parent–child relationships among two groups of homosexuals— one in which the homosexuals exhibited little or no other evidence of sexual inversions, and the other in which there were substantial inversions besides homosexuality. If the causal order is as our theory indicates, homosexuals with no significant inversions of sex-typical behavior patterns will not differ from heterosexuals in terms of their relationships with their parents. Only those homosexuals with inverted sex-typical behavior patterns prior to puberty should have parent–child relationships distinguishable from heterosexuals. Relevant to this deduction, Sipova and Brzek (1983) reported that fathers of homosexual sons with the most effeminate mannerisms expressed fewer positive feelings toward their offspring than fathers of homosexual sons with the least effeminate mannerisms.

Homosexuality should reflect a significant degree of hereditability. If the theory is correct, homosexuality should be nonrandomly distributed along family lines. This is because even though environmental factors are involved in the etiology of sexual orientation, genetic factors also appear to be involved. Not only have specific genetic mutations already been found that cause homosexuality and other sexual inversions, but there are likely to be many genetic factors that merely increase the susceptibility to sexual inversions (see Durden-Smith & deSimone, 1983, p. 187).

Support for this deduction can be found in studies reporting considerably higher concordance rates for homosexuality among identical twins than among fraternal twins (reviewed by Cooper, 1978). In addition, several studies have found that close relatives of homosexuals have higher incidences of homosexuality than the general population (reviewed by Pillard, Poumadere, & Carretta, 1981). In a follow-up of this review article, the same authors (1982) found that nearly one-quarter of all brothers of male homosexuals also were homosexuals, a much higher rate than the 3–7% typically reported among human males generally.

Average neurohormonal differences should exist between homosexuals and heterosexuals in both sexes at comparable ages. Most studies have found that male homosexuals and male heterosexuals have similar levels of testosterone circulating in their blood after puberty (Birk, Williams, & Chasin,

1973; Brodie, Gartrell, & Doering, 1974; Doerr, Kockott, & Bogt, 1973; Pillard, Rose, & Sherwood, 1974; Tourney & Hatfield, 1973), with male homosexuals' levels possibly being slightly lower on average than those of male heterosexuals, but still well within the typical male range, compared to the typical female range (Kolodny, Masters, & Hendryx, 1971; Loraine et al., 1970; Pillard et al., 1974). Among females, lesbians appear to have circulating testosterone levels in the upper normal range for females generally, but still substantially below those found in virtually all males (Meyer-Bahlburg, 1979).

This failure to find major differences in circulating testosterone levels between homosexuals and heterosexuals has been thought to cast doubt on all neurohormonal explanations of sexual orientation (e.g., Robertson, 1977, p. 204). However, the lack of a relation (or at least a strong relation) between average postpubertal testosterone levels and sexual orientation is predicted by the present theory when one considers how mammalian sexual differentiation occurs. In humans, the gonads differentiate roughly during the first 4 months of gestation, whereas virtually all of the neuro-organization pertinent to sexual orientation appears to occur between the 3rd and 4th months. The main key to theoretically understanding inversions of sexual orientation is an inconsistency between what sex hormones do outside the nervous system (particularly in the genitals) and what they do inside the nervous system. In humans, the amount of testosterone produced by the testes after puberty primarily reflects how completely the testes were differentiated in the first 4 months of pregnancy; postpubertal testosterone production probably has only a slight relation to how much testosterone reached the brain during the first and second trimesters of pregnancy (when sexual orientation appears to be largely determined).

Nevertheless, for both sexes, the theory would predict that there should be neurohormonal differences between homosexuals and heterosexuals (with bisexuals, in most cases, being intermediate). The differences should have to do primarily with how the brain responds to sex hormone infiltration, rather than with how well the gonads produce sex hormones. According to two studies, one difference appears to have been identified. It relates to the fact that females tend to display an LH surge in response to estradiol injections, but males do not (Harris, 1964; MacKinnon, 1978). This female tendency is fundamental to the maintenance of monthly cyclicity in ovulation. The major events are as follows. As an increased amount of estradiol is released from the ovaries during the first half of the menstrual-estrus cycle, eventually an LH surge is triggered by the hypothalamic-pituitary network. This surge inhibits further estradiol production and induces ovulation and increased progesterone release, which, in turn, eventually cause estradiol production to also gradually rise, and thus the cycle starts over again. The entire process can be manipulated in most females merely by injecting high levels of estradiol. In the male, however, the hypothalamic-pituitary network is usually organized in such a way as to prevent major estradiol-induced LH surges.

Theoretically, one would expect many homosexuals to show at least partially inverted estradiol-induced LH surges relative to heterosexuals of their sex. Following evidence of an F/dM pattern of LH surges in response to estradiol injections among many male rats who had had their sexual orientations experi-

mentally inverted (Dorner, 1967, 1972; Kalcheim et al., 1981), Dorner, Rohde, Stahl, Krell, and Masuis (1975) reported similar tendencies in about one-half of a group of human male homosexuals. More recently, Gladue, Green, and Hellman (1984) also found that about one-half of the male homosexuals they tested exhibited an LH surge in response to estradiol injections, something that was found in almost no male heterosexuals.

Attempts to alter sexual orientation after birth should be minimally effective. From the standpoint of treatment, the present theory implies that efforts to change sexual orientation should be essentially confined to modifying where, when, and how sexual orientation is expressed; the orientation itself should not change. Theoretically, changing a homosexual's orientation should be just as difficult and as emotionally wrenching as changing a heterosexual's orientation.

Most therapeutic attempts to change sexual orientation have involved some type of counseling and/or conditioning procedures. We hypothesize that these methods should be only minimally effective, especially in any long-term sense. Basically, sexual orientation appears to be largely determined by hypothalamic-limbic system brain functioning, and most conditioning procedures, and certainly all counseling methods, gear their corrective efforts at neocortical functioning ("rational thought"). Although the neocortex's ability to learn ways to override and circumvent lower brain functioning should never be underestimated, basically a homosexual's neocortex would have to learn how to prevent hypothalamic-limbic areas of the brain from functioning as they were organized to function.

Concerning bisexuals, because their sex partner preferences tend to be ambiguous, one could reasonably expect a measure of success in changing their choices of sex partners to more or less exclusive heterosexuality.

Although there have been claims for many effective methods of treating homosexuality—even including the use of religious conversions (Pattison & Pattison, 1980) and exorcism (Ross & Stalstrom, 1979)—the reports do not seem to contradict our minimal-effect hypothesis. Specifically, claims of success primarily have come from those studies using various forms of psychotherapy, group therapy, and aversive conditioning. Success rates for psychotherapy (Bieber et al., 1962; Beiber & Beiber, 1979, p. 416; Coates, 1962; A. Ellis, 1965; McLeish, 1960), for group therapy (Schwartz & Masters, 1984), and for aversive conditioning (reviewed in Adams & Sturgis, 1977) typically are in the 30–60% range. The criteria for success often have been either vague or considerably less than exclusive heterosexual behavior, and the follow-up periods typically have been no more than 1 or 2 years. In addition, one finds that in virtually all of these clinical studies, nearly all of the clients voluntarily sought help and expressed a desire to change (Allen, 1953; Hatterer, 1970; McLeish, 1960; Schwartz & Masters, 1984, p. 173). Because our theory contends that a homosexual would be as adverse to sexually interacting with a member of the opposite sex as a heterosexual would be contemplating sexual intimacy with a member of the same sex, most of those seeking treatment are in fact probably bisexuals. Support for that deduction comes from reports that the best single predictor of which "homosexuals" are most likely to respond to treatment is the amount of heterosexual experience prior to treatment (Coates, 1962; Feldman & MacCulloch, 1971, p. 15; Mendel-

sohn & Ross, 1959). In fact, some have concluded that successful treatment was impossible for clients who have had no pleasurable heterosexual experiences prior to program entry (Acosta, 1975, p. 19; Marciano, 1982, p. 152).

Conclusion

Recent surveys in the United States indicate that about one-quarter of the adult population believes that innate factors cause homosexuality; the remainder largely attribute the phenomenon to various childhood and adolescent experiences (Gallup, 1977, p. 10; Leitenberg & Slavin, 1983, p. 342). The literature review at the beginning of this article suggests that a majority of behavioral scientists also favor experiential explanations for sexual orientation.

We have tried to review and organize the available scientific evidence into a fairly comprehensive theory. This theory holds that sexual orientation is a fundamental component of mammalian sexual differentiation, and that inversions of sexual orientation are not unique to the human species. Without delineating all of the combinations of factors that could induce sexual inversions, we identified four categories of causes that are independent of experiential processes (from the standpoint of the individual whose sexual orientation is affected). These are (a) direct genetic-hormonally induced inversions, (b) drug-induced inversions, (c) inversions due to maternal stress during pregnancy, and (d) inversions caused by immunity factors. The one social experiential factor that seems to invert sexual orientation involved segregation of an individual from all members of the opposite sex throughout most of childhood. Whether or not real-life parallels to this experimental procedure exist remains to be seen.

According to our theory, complex combinations of genetic, hormonal, neurological, and environmental factors operating prior to birth largely determine what an individual's sexual orientation will be, although the orientation itself awaits the onset of puberty to be activated, and may not entirely stabilize until early adulthood. The involvement of learning, by and large, only appears to alter how, when, and where the orientation is expressed. For humans, the crucial timing appears to be between the middle of the 2nd month of gestation and about the middle of the 5th month, during which time the hypothalamic-limbic regions of the male's nervous system are permanently diverted away from their otherwise-destined female phenotype.

Either because of unusual features in the genetic programs that control the biochemical processes of masculinization and feminization or because of environmental interference with these biochemical processes, sexual inversions of varying degrees occur fairly frequently. These inversions can involve any of the four phenotypic dimensions of sex: the genital dimension, the nongenital morphological dimension, the neurological dimension, and the behavioral dimension. Most important to our theory are the latter two dimensions. Theoretically, the behavioral dimension of sexuality primarily is a manifestation of the neurological dimension, and the neurological dimension may be conceived of in terms of two stages. The first stage establishes essentially permanent differences in the hypothalamic-limbic region, wherein sexual orientation basically is determined. The second stage, occurring during the latter half of human gesta-

tion, and apparently involving more diverse and recently evolved brain parts, pertains to behaviors that tend to complement sexual orientation, that is, sex-typical behavior patterns.

Finally, to those interested in preventing homosexuality, any use of our theory to do so at this point would be reckless. Even if the essential accuracy of the theory were to become established, careful thought should be given to the desirability and potential hazards of intervention. Several decades of intense research may be required to adequately test the theory, and, if it is basically confirmed, to identify precisely where and when intervention might be feasible. Also, before attempting intervention, moral issues should be addressed. Although morality can never be directly derived from a scientific theory, our theory, at the very least, challenges those who are intolerant of homosexuals (see Plummer, 1975, p. 102) and those who support the retention of laws against their expressing themselves sexually (see A. P. Bell & Weinberg, 1978, p. 187). The increasing public acceptance of homosexuality apparent in recent surveys (Glenn & Weaver, 1979, p. 114; M. Yalom, Estler, & Brewster, 1982, p. 150) is in tune with the evidence reviewed. Ultimately, the theory implies that, were it not for delicately balanced combinations of genetic, neurological, hormonal, and environmental factors, largely occurring prior to birth, each and every one of us would be homosexual.

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