2 Biological Perspectives on Sexual Orientation

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The question of "biological" influences on human sexual orientation remains immensely controversial (see Barinaga 1991). This stems, in part, from the inconclusive nature of the empirical evidence; however, the ambiguity of the scientific answers is only part of the problem. The question "Is homosexuality 'biological'?" has been subjected to many interpretations, often not clearly specified. Thus this chapter has two main goals: first, to clarify different meanings that have been attached to "biological" in the context of research on human sexual orientation and, second, to summarize research findings for the most pertinent meanings.

Before we consider alternative meanings of "biological," it is important to specify what is meant by sexual orientation and related terms such as *heterosexual* and *homosexual*. I use the term *sexual orientation* to refer to one's pattern of sexual attraction, to men or to women. Thus men who are sexually attracted to women and women who are sexually attracted to men are "heterosexual" in their sexual orientation. Men who are attracted to men and women who are attracted to women have a "homosexual" orientation. Those who are attracted to both men and women have a "bisexual" orientation. Note that these are psychological rather than behavioral definitions. Thus, for example, the behaviorally heterosexual man who is attracted to both his wife and her brother is considered bisexual, and the teenage male prostitute who sells his body only for the purpose of buying gifts for his female love interest is heterosexual in orientation.

This is not the only possible meaning of sexual orientation. Some men who are sexually attracted to other men call themselves heterosexual. Some

women who are less sexually attracted to women than to men call themselves lesbians. They are using these words to label what I call their "sexual identity," which I understand to be the identity they desire for reasons other than the relative intensity of the sexual feelings for men versus women. For instance, a man who prefers sex with men may still identify himself as heterosexual, because he prefers heterosexual marriage or other aspects of lifestyle more common to heterosexuals (e.g., rearing children). A woman who prefers sex with men may adopt a lesbian identity as an expression of her emotional and political solidarity with lesbian feminists. In these cases *sexual orientation* and *sexual identity*, as these terms are used herein, are discordant. Some writers have emphasized the frequency of discordance between sexual identity, sexual orientation, and sexual behavior (Klein 1990). In my research experience, however, men and women who label themselves "heterosexual" have almost always admitted to far greater sexual feelings toward and activity with the opposite sex. The opposite pattern has been true for those who call themselves "homosexual," particularly regarding recent patterns of feelings and behavior (as opposed, e.g., to feelings and behavior during adolescence). Nevertheless, these observations are based only on my own research experiences. The degree of concordance between sexual orientation, sexual identity, and sexual behavior is an important question that deserves far more systematic attention that it has received. I chose a psychological definition of sexual orientation for this chapter because it seems likely that sexual attraction is more closely linked to potential biological mechanisms than either sexual identity or sexual behavior, which are more susceptible to social processes (Le Vay 1993). It also seems plausible that sexual orientation is more longitudinally stable than either sexual identity or sexual behavior; however, this remains an uninvestigated empirical question.

Alternative Construals of the "Biological" Question

Biological Determinism Versus Free Will

The argument over whether homosexuality is "biological" or "freely chosen" is perhaps the most common and least productive version of the biology debate. It is common because participants on both sides believe that crucial moral answers hinge on its outcome. For instance, they argue that if homosexuality is biologically determined, and hence homosexuals could not have chosen heterosexuality, then it is unfair to judge their sexual behavior

morally. The argument is unproductive for at least two reasons. First, its resolution is primarily a philosophical rather than an empirical matter, and scholars who have considered it at length generally (and legitimately) take a strong position independent of evidence (e.g., Money 1988; Le Vay 1993). Second, the rational link between the position that homosexuality is biologically determined and a sympathetic view of homosexuality is much more tenuous than commonly assumed. This is because all behavior is biologically determined, in one fundamental sense. Thus if homosexuality (or heterosexuality) is excused on the grounds that it is biologically determined, all behavior must be excused, including behavior that should not be excused, such as dishonesty, theft, homophobia, or even genocide. These behaviors are also biologically determined, in the sense I now elaborate.

Most scientists are both (strict) determinists and materialists. Determinism, in its strict sense, implies that all present events (including mental states and behaviors) are completely caused by past events. Equivalently, given a configuration of events at Time A, there can be exactly one configuration of events at later Time B. Materialists believe that all causes and effects obtain in the material world, as opposed, for instance, to a nonmaterial "soul." Thus a materialistic determinist acquainted with modern neuroscience believes (as I do) that all behavior is most proximately caused by brain states, and thus behavioral differences must be caused by brain differences. This is true even for socially acquired traits. For instance, there must be relevant brain differences between the group of people who have learned the quadratic theorem and the group who has not yet acquired this knowledge, though of course those brain differences are undoubtedly subtle. Future recitation of the theorem depends on activating the brain's "representation" of it. Thus all behaviors are "biologically determined" in the sense that all events are caused, and behavioral events are caused by brain states, which are "biological." By these assumptions, the mere fact that Le Vay (1991) found a brain difference between homosexual and heterosexual men was unsurprising; such a difference has to exist. Because we are biological organisms, everything about us is traceable to biology. As John Money (1988) incisively noted: "The postnatal determinants that enter the brain through the senses by way of social communication and learning are also biological, for there is a biology of learning and remembering. That which is not biological is occult, mystical, or, to coin a term, spookological" (p. 50). To be sure, there are some more meaningful and interesting construals of the question "Is homosexuality 'biological'?"—some of which are considered in the

following. To encourage more responsible usage, I recommend referring to "biological" causes, influences, theories, or explanations (i.e., with quotation marks). This draws attention to the problematic term that has both numerous connotations and an uninformative literal meaning.

Innate Versus Acquired

Most people participating in the "biology" debate have this version of the issue in mind, at least part of the time. As Lehrman (1970) noted, there are at least two interpretations of "innate." The interpretation of innate as genetic or heritable is the more restrictive of the two, and a discussion of that sense is deferred to the next section. A behavior is also said to develop innately to the extent that it develops in a uniform or fixed pattern without being learned. In this sense, innate signals an independence from, or perhaps a resistance to, psychosocial influences.

Studies in comparative psychology and ethology have shown that behaviors cannot simply be divided into those that are innate versus those that are acquired. For instance, Mineka et al. (1984) showed that rhesus monkeys acquired a fear of snakes by observing other monkeys react fearfully to a snake, and thus the fear is acquired. However, another study (Cook and Mineka 1989) demonstrated that the monkeys did not acquire a fear of flowers, even though they had observed monkeys reacting fearfully to them (through videotape manipulation). They suggested that because snakes are dangerous to monkeys and flowers are not, rhesus monkeys have become "evolutionarily prepared" to learn fear of snakes easily through observation. Furthermore, since snakes are common in the monkeys' natural habitat, they are virtually guaranteed to acquire snake fear. Thus, in a sense, snake fear in rhesus monkeys is highly innate.

Given these complications, it is tempting to dismiss discussion of whether a characteristic such as homosexuality is "innate" or "acquired" as misguided and, instead, to focus on elaborating the process of development. While it is certainly true that obtaining a full picture of development is more illuminating than determining the degree of innateness, the latter goal is also useful for organizing research to accomplish the former. Thus diverse processes of acquisition, such as operant and classical conditioning, imitation, and persuasion, can be theoretically and empirically pitted against such innate processes as heredity and prenatal neuroendocrine development.

Genes Versus Environment

The question "Is homosexuality 'biological'?" is often asked in the form "Is homosexuality genetic?" That the question is often not meant literally was recently evidenced on an American talk show in which a gay man with a heterosexual identical twin argued emphatically that sexual orientation is "genetic," seemingly oblivious to the contradictory nature of his own personal evidence. In fact, many people say "genetic" when they mean "innate," a problematic equation. For instance, if massive androgen injections given prenatally to a female fetus altered her sexual orientation, this would be an innate influence, but it would be entirely environmental. Conversely, there are conceivable developmental routes that involve the genes but that most people would not consider innate. For instance, suppose a gene existed for feminine beauty and, furthermore, that boys with this trait were relatively likely to be treated in a way that fostered homosexuality. A quantitative genetic analysis would find homosexuality to be heritable, but the necessary developmental step would be psychosocial. A phenotypic (observable) difference between organisms is genetic, or heritable, to the extent that it is attributable to genetic differences between them, regardless of the intervening steps from genotype to phenotype.

Essentialism Versus Social Constructionism

During the past decade perhaps the most contentious version of the "biology" debate has been whether sexual orientation is a category universal (in some way) to every culture or merely an arbitrary categorization that says more about the observer (or constructor) than the observed (e.g., Halperin 1989). Social constructionists make much of historical accounts of Greece and Rome, in which sexual acts between men appear to have been much more common than they are in contemporary Western societies. Furthermore, they argue that there was no equivalent categorization in the ancient societies. In contrast, those labeled by the social constructionists as essentialists argue that there have probably been people whom we could identify as homosexual, bisexual, and heterosexual in all times and places. Boswell (1980, 1989, 1990) has argued persuasively that these categories have been recognized throughout the recorded history of Western civilization.

The essentialism-constructionist debate is fueled, in part, by the different ways that the two sides use "homosexuality." Social constructionists empha-

size cultural variation in incidence of homosexual behavior and in the way sexuality is treated linguistically. These issues are actually more pertinent to the social construction of sexual identity and sexual behavior than that of sexual orientation. Boswell (1990), however, has generally emphasized homosexuality and bisexuality as psychological attraction patterns.

But even granting the social constructionist premise that homosexuality (or, for that matter, heterosexuality) occurs in only some societies, it does not follow, as some constructionists believe (e.g., De Cecco 1990), that "biological" investigations into sexual orientation are misguided. Given a society that has constructed the sexual categories "heterosexual" and "homosexual," there is still the question of why people may adopt one or the other label (or are so labeled by others). The categories "priest," "Sumo wrestler," and "Fortune 500 executive" are surely more socially constructed than "homosexual" or "heterosexual," but within any society in which they are meaningful, there are probably "biological" (i.e., innate or genetic) factors that contribute to the likelihood that one will be categorized within any one of them.

"Biological" Explanations of Sexual Orientation: The Empirical Evidence

The Neuroendocrine View

Background The most influential "biological" theory of sexual orientation is motivated by the observation that homosexuals have a sexual orientation identical to that of opposite-sex heterosexuals. Gay men and heterosexual women are sexually attracted to men; lesbians and heterosexual men are sexually attracted to women. The neuroendocrine theory of sexual orientation then, in simplistic and general form, is that the brains of gay men have something in common with those of heterosexual women, and similarly for lesbians and heterosexual men. Furthermore, according to this view, the relevant brain differences between men-preferring and women-preferring individuals are relatively innate, depending less on postnatal experience such as parental socialization than on patterns of hormonal exposure.

Before examining the neuroendocrine theory in detail, let us consider its plausibility on a priori grounds. First, children who become homosexual adults appear to display some behaviors more typical of the opposite sex. Gay men frequently (but not invariably) remember being relatively gender nonconforming in childhood — for example, being teased for being "sissies,"

preferring female playmates, and shunning rough sports (Bell et al. 1981; Grellert et al. 1982; Harry 1983; Whitam 1977). Prospective studies have shown that this association is not because of memory bias. A majority of extremely gender atypical boys become gay or bisexual adults (Green 1987; Zuger 1978), a far higher proportion than would be expected by chance. Similarly, lesbians recall being more masculine during childhood compared to heterosexual women (Bell et al. 1981), though there have unfortunately been no prospective longitudinal studies of tomboys. For both men and women, the association between sexual orientation and (recalled) childhood gender nonconformity is strong (though somewhat less so for women) (Bailey and Zucker 1993). This supports the idea that homosexuals have been subject to some influences more typical of the opposite sex and is thus consistent with a neuroendocrine hypothesis.

Doubtless some readers will cringe at the implication that a sexual orientation difference mirroring a sex difference is consistent with a "biological" theory. Are human sex differences in behavior not caused by socialization differences? In fact, an immense body of research describes differences in the ways that boys and girls are socialized by parents, other adults, and peers. However, the vast majority of these studies cannot claim to show more than that boys and girls are treated differently. They cannot claim to show that this differential treatment makes *any* difference, much less that it makes *all* the difference. (The human sex differences literature is limited primarily by the difficulty of doing definitive research on etiological questions about sex differences, since it is rarely possible to separate "biological" and social influences; e.g., typical females are both "biologically" female and treated as females.) My own intuition is that both social and "biological" factors will be found necessary to account for many behavioral sex differences. In any case, advocates of neither nature nor nurture can honestly claim to have excluded the other side's explanation for any behavioral sex difference. Hence the possibility of innate sex differences remains viable.

Indeed, sexual orientation may be an especially strong candidate for "biological" causation. This is because the most familiar social influences cannot plausibly be operating. Homosexuals are attracted to members of their own sex despite their (usually) heterosexual parents' example and despite the punishment that they endure from peers and many other enforcers of social norms. Furthermore, prehomosexual boys are often gender nonconforming despite being socialized to the contrary and despite the punishment that often follows such behavior in males. Although some psychological theories circum-

vent these problems by emphasizing subtle aspects of parenting (e.g., Lidz 1968), these theories have generated remarkably little empirical support (Bell et al. 1981; Siegelman 1981). Moreover, insofar as such theories have garnered support, the direction of causation is ambiguous. For instance, consistent with Freudian theory, homosexual males do appear to have poorer childhood relationships with their fathers than do male heterosexuals (Bell et al. 1981). However, as Bell et al. have pointed out, it is possible that the fathers are reacting to the atypical childhood behaviors of the prehomosexual boys.

The Theory The neuroendocrine theory of sexual orientation (Byne and Parsons 1993; Ellis and Ames 1987; Meyer-Bahlburg 1984) posits that the sexual differentiation of brain structures affecting sexual orientation proceeds roughly analogously to the differentiation of morphological structures such as the external genitalia. Both male and female embryos start development identically. Sexual differentiation begins when the undifferentiated gonads develop into either ovaries or testes; male development is triggered by the sex determination gene on the Y chromosome. Later, the testes of the male fetus secrete two hormones that further masculine differentiation. Müllerian inhibiting substance (MIH) prevents the growth of the uterus and related structures, and in this sense is a defeminizing substance. In contrast, testosterone and other closely related substances (generally speaking, androgens) masculinize relevant structures, forming both the internal male sex organs and the external genitalia. For the most part, masculine development requires androgens, and without the action of androgens, feminine development occurs. This is evidenced most dramatically in 46, XY androgeninsensitivity syndrome, in which genetic males lack a gene needed to utilize androgens effectively, despite normal androgen levels. Individuals with this syndrome are evidently typical females, both anatomically and psychologically, with the exception of the internal reproductive organs, whose formation was blocked by MIH (Money 1988).

The neuroendocrine theory of homosexuality hypothesizes that there are brain structures that sexually differentiate during prenatal and possibly early postnatal development and that these structures determine sexual orientation toward males or toward females. Presumably, masculinization of the relevant brain structures in heterosexual men and homosexual women occurs because of relatively high levels of androgens, whereas development in a feminine direction requires a relative dearth of androgens (or relatively low sensitivity to androgens). The neuroendocrine view stresses the role of organizational, as opposed to activational, hormones; that is, androgens are

hypothesized to affect the sexual differentiation of brain structures during critical periods of development. For instance, one cannot necessarily predict that in a homosexual man the differentiation of a brain structure in a feminine direction will be associated with a low level of circulating testosterone during adulthood. Indeed, that hypothesis is untenable given a large number of studies that show otherwise (Meyer-Bahlburg 1984). It should be noted that the neuroendocrine theory makes rather strong predictions about the existence of relevant neural structures affecting sexual orientation in men and women. While the account of morphological differentiation provided above is generally accepted, the causes and extent of sexual differentiation of the human brain remain speculative.

Fortunately the neuroendocrine theory does not merely rest on the analogy with morphological sexual differentiation. Four general areas of research have been used to support a neuroendocrine view of homosexuality: studies manipulating the sexual behavior of nonhuman animals, studies of humans with unusual patterns of hormone exposure, studies relating sexual orientation to traits thought to be innately sexually dimorphic, and direct neurophysiological studies of human sexual orientation.

Studies of Nonhuman Animals The study of other species, particularly rodents, has been immensely important in the development of a "biological" view of homosexuality (Adkins-Regan 1988; Byne and Parsons 1993; Meyer-Bahlburg 1984). Perhaps the most influential animal model has been the rat. Typical female rats exhibit a posture called "lordosis" during sexual receptivity in response to appropriate tactile stimulation; lordosis allows male rats to achieve intromission and ejaculation. Typical male rats, in contrast, show high rates of mounting behavior. However, genetic males can be made to display lordosis by (surgically or chemically) castrating them prenatally or perinatally and then administering appropriate hormones during adulthood to activate the behavior. Similarly, genetic females can be made to exhibit a male pattern of sexual behavior by the perinatal administration of androgens and subsequent replacement of sex hormones in adulthood. The sexual differentiation of these behaviors has been shown to involve the preoptic, anterior, and ventromedial portions of the hypothalamus.

There is an important limitation of the rat findings as support for a neuroendocrine model for human sexual orientation (Atkins-Regan 1988; Byne and Parsons 1993; Meyer-Bahlburg 1984). Human homosexuals do not clearly display a pattern of copulatory behavior typical of the opposite sex, with the exception of their sexual orientation. Homosexual men do not

appear to show decreased mounting behavior nor homosexual women an increase in mounting behavior, compared to their heterosexual counterparts. Conversely, the large majority of the rat studies have failed to assess preference for males versus females. Thus the rat studies have focused on a dimension of behavior that is not clearly relevant to human sexual orientation. As Byne and Parsons (1993) have noted, when a neonatally castrated rat displays lordosis in response to mounting by another male, it is the mounted animal that has provoked the interest of psychoneuroendocrinologists, not the animal that initiated the contact. Yet if rat sexual behavior were directly analogous to human sexual orientation, the male who mounted the treated animal would be equally worthy of an explanation. There have, in fact, been experimental studies of the origins of preference for males versus females in rats (Brand et al. 1991, 1992) and ferrets (Martin and Baum 1986; Stockman, Callaghan, and Baum 1985; see also the review of this issue by Adkins-Regan 1988). These studies have explored the consequences of either blocking the effects of androgens in male animals or administering androgens to female animals, prenatally or perinatally. In general, treated adult animals spend less time with animals of the opposite sex and more time with animals of the same sex. Though the behaviors in these studies appear to have more relevance to human sexual orientation than do studies of mounting or lordotic behavior, they are still uncomfortably distant from establishing a consistent interest in sexual contact with same-sex conspecifics.

What, then, has been the value, if any, of neuroendocrine studies of rodents and other nonhuman species? As Ruse (1988) has noted, these studies have had immense heuristic value in the specification of models for human sexual orientation. It was largely results of animal work that led researchers such as LeVay (1991) to focus on the anterior hypothalamus as the most promising area of the human brain to be causally related to sexual orientation. On the other hand, some scientists argue that some researchers have assumed too close a correspondence between the sexual behavior and related brain organization of rats and humans, thus leading themselves (and the field) astray (Gooren 1990; Byne and Parsons 1993). The ultimate gauge of the value of animal models for human sexual orientation will be the number of theoretically interesting, replicable findings they generate, using human subjects.

Studies of Prenatal Influences in Humans For obvious reasons experimental studies of the effects of prenatal hormonal manipulations on humans are impossible. However, in some rare circumstances humans have been

inadvertently exposed to unusual patterns of hormones in utero, as a result of either medical intervention or genetic anomalies. These "natural experiments" are potentially informative regarding the effects of hormones on the development of sex-dimorphic behavior such as sexual orientation.

Perhaps the most extensively studied condition has been congenital adrenal hyperplasia (CAH). CAH is a genetic autosomal recessive condition that prevents the production of sufficient quantities of cortisol to inhibit the release of adrenocorticotropic hormone (ACTH) and subsequent adrenal steroid synthesis. Affected individuals are thus exposed to high levels of androgens. The level of androgens is sufficient to cause some degree of masculinization of genitals in most females with the condition, enough so that the sex of the child is frequently ambiguous at birth. Some of these females have been reared as males, particularly before the 1950s, before corrective surgery was available or because diagnosis of the condition was late. However, the large majority of such women are now assigned as females and given early surgery to feminize their genitals and ongoing hormonal therapy to prevent virilization.

A neuroendocrine theory of sexual orientation is supported by, and indeed seems to require, a finding of increased homosexuality among CAH females reared as women. There have been several studies of this issue. Ehrhardt, Evers, and Money (1968) reported that as many as half of their twenty-three CAH female subjects were bisexual (depending on the criteria). None was exclusively homosexual. This study was problematic because it included late-treated patients, who were notably masculine in appearance. In contrast, a study of eighteen late-treated CAH women found no reports of homosexual fantasy or experience (Lev-Ran 1974). However, subjects in this study were from the Soviet Union, where intolerance of homosexuality was particularly high, possibly making subjects less open. Money et al. (1984) studied thirty women with early-treated CAH and found results similar to those of Ehrhardt et al. Of those for whom sexual history data were available, 48 percent were bisexual with respect to fantasy or behavior, significantly higher than a control group. The largest study to date on sexuality among CAH women, by Mulaikal et al. (1987; this paper contains some subjects studied by Money et al. 1984), found a 5 percent (4/80) rate of self-identification as "homosexual" or "bisexual." Unfortunately these authors did not report the incidence of homosexual attraction, as distinct from behavior. Furthermore, fully 38 percent of the women in this sample gave insufficient data to ascertain sexual orientation. It is possible that those with homosexual feelings may have been overrepresented in that group. Ditt-

mann et al. (1992) found increased homosexual versus heterosexual interest in thirty-four female CAH patients, compared to fourteen control sisters. Finally, in a recent abstract Zucker et al. (1992) reported that a sample of twenty-nine CAH women had significantly less attraction to men and significantly more attraction to women than a control group consisting of their female relatives.

There is another body of CAH research which, though not directly concerned with sexual orientation, is quite relevant. This is the study of gender atypicality of female children with CAH. Since gender atypicality or gender nonconformity is a strong predictor of adult homosexuality, a finding that girls with CAH are, for instance, more tomboyish would provide indirect support for a neuroendocrine theory of homosexuality. Several studies have provided results suggesting that CAH girls are tomboyish in certain respects, particularly regarding play patterns (Berenbaum and Hines 1992; Ehrhardt et al. 1968; Ehrhardt and Baker 1974). The most methodologically rigorous of these studies, by Berenbaum and Hines, compared CAH girls and boys to young male and female relatives on a free play paradigm, in which "feminine," "masculine," and "neutral" toys were equally available. CAH girls and boys and unaffected control boys were all much more likely to play with "masculine" toys and less likely to play with "feminine" toys than were control girls.

The literature regarding CAH and sexual orientation is inconclusive. Although most of the studies have found some evidence for increased homosexuality in CAH women, the largest study (Mulaikal et al. 1987) found relatively low rates. The link between CAH and some aspects of childhood gender nonconformity is more compelling, but this provides only indirect evidence for a neuroendocrine view of homosexuality. Furthermore, some have argued that even if CAH women have higher rates of homosexuality (or childhood gender nonconformity), a neuroendocrine interpretation is unnecessary (Byne and Parsons 1993; Bleier 1984). This is because CAH females are often born with masculinized genitals that might very well affect parental attitudes or self-concept in important ways. However, it should be noted that Berenbaum and Hines found the degree of masculine toy preference in their CAH girls to be unrelated to the degree of genital virilization reported at diagnosis. Furthermore, parents of CAH and normal girls did not differ in their reports of behavior toward their daughters. Although studies of CAH have provided some promising results, and therefore may eventually provide definitive data on the question, they cannot now be invoked as conclusive evidence for either nature or nurture. For more conclusive findings, it will be necessary to study large samples of CAH females (as

Mulaikal et al. did), comparing them to large control samples on detailed measures of sexual attraction (as some of the smaller studies did).

I have focused on CAH because it has been studied relatively frequently and because, of all the hormonal anomalies, it is closest to the neuroendocrine model of homosexual etiology. Other conditions have been mentioned as relevant for theories of sexual orientation, including androgen insensitivity, 5-alpha reductase deficiency, and prenatal exposure to synthetic hormones with androgenizing effects. However, androgen insensitivity and 5-alpha reductase deficiency are far less convincing than CAH as "natural experiments," because they do not clearly separate hormonal and experiential influences (Byne and Parsons 1993; Money 1988). For instance, androgen-insensitive XY individuals are effectively female in their hormonal influences. They are also raised as females, thus confounding social and "biological" influences. Female offspring of hormonally treated pregnancies are less problematic in this interpretive respect, but the results of relevant studies have been highly inconclusive (Byne and Parsons 1993).

Sexually Dimorphic Traits and Sexual Orientation Another strategy for studying neuroendocrine hypotheses has been to compare homosexuals and heterosexuals on traits that are sexually dimorphic. Findings that homosexuals are somewhat intermediate between heterosexual men and women on these traits provide some support for a neuroendocrine theory of homosexuality, particularly if the relevant traits are plausibly thought to be innately sexually dimorphic. The rationale, often unstated, is that a pattern of hormonal influences causing a brain to differentiate homosexually is likely to have more general effects. These should result in a more gender atypical pattern of neural organization in some other respects, as well.

One characteristic that seemed promising in this respect was the luteinizing hormone (LH) response. Female rats show a surge of LH following secretion of estrogen, which triggers ovulation. In contrast, estrogen inhibits LH secretion in male rats. In rats this sex difference has been found to depend on the organizational effects of prenatal androgens (Gorski 1966). Humans also show a sex difference in LH secretion following estrogen injections. Thus great excitement initially greeted two reports that homosexual (but not heterosexual) men show a partial LH surge to the administration of estrogen (Dörner et al. 1975; Dörner 1988; Gladue et al. 1984). These findings supported the possibility that male homosexuals have a "feminine brain."

However, it now appears that the LH data may be problematic as support for a neuroendocrine theory of homosexuality. Other studies have failed to

replicate the finding of a partial LH surge in male homosexuals (Gooren 1986a; Hendricks et al. 1989). These failures, by themselves, are not a fatal blow to the LH data, because they were all small studies, with insufficient statistical power to guarantee replication. More problematic is the work of Gooren (1986a, 1986b), who has demonstrated that the human sex difference in LH release is unlikely to reflect differences in neural organization. In an elegant series of experiments, Gooren studied male-to-female and female-to-male transsexuals, before and after hormonal therapy and sex reassignment surgery. He found that these individuals showed a pattern of LH response appropriate to their hormonal and/or gonadal sex; thus LH response appears to be a function of circulating androgens; that is, pretreatment female-to-male and post-treatment male-to-female transsexuals showed the LH surge, whereas pretreatment male-to-female and post-treatment female-to-male transsexuals did not. Indeed, the same individuals showed two different patterns of LH response in two different phases of their treatment. Gooren thus demonstrated that LH response patterns are unlikely to provide information about the masculine or feminine neural organization of homosexuals or heterosexuals. Because two independent studies found an association between sexual orientation and LH response, and since no one has convincingly explained away these findings, they cannot be entirely rejected. Nor, on the other hand, can they be considered strong evidence for a neuroendocrine theory of homosexuality.

A second line of research has focused on sexually dimorphic characteristics thought to be related to cerebral lateralization (Geschwind and Galaburda 1985), including spatial ability and handedness. Lateralization refers to the tendency of certain brain functions to be specialized in either the right or left cerebral hemisphere. Males are more lateralized than females, and right-handers are more lateralized than left-handers, on average, for both verbal and spatial functions (McGlone 1980). Somewhat paradoxically, males are more likely than females to be left-handed, but this is thought by some scientists to result from a sex difference in timing of cerebral development (Geschwind and Galaburda 1985). Men tend to have higher spatial abilities relative to verbal abilities, and this pattern has been hypothesized to be related to sex differences in lateralization. It is also noteworthy that CAH has been associated with both increased left-handedness (Nass et al. 1987) and higher spatial scores (Resnick et al. 1986) among women.

Several studies have reported homosexual men to have a higher incidence of left-handedness than heterosexual men (McCormick et al. 1990; Lindesay 1987; Götestam et al. 1992). Relatedly Watson (1991) found an

increased rate of left-handedness among male-to-female transsexuals, and Gooren (1991) obtained similar results in a combined sample of male-to-female and female-to-male transsexuals. On the other hand, two recent reports on large samples both failed to find an increase in left-handedness among homosexual men (Satz et al. 1991; Marchant-Haycox et al. 1991). McCormick et al. (1990) also found an increased rate of left-handedness among homosexual women, the only report to focus on women to date, and Tkachuk and Zucker (1991) found a higher incidence of left-handedness in a combined sample of homosexual males and females. Thus there is some indication that both male and female homosexuals have an increased rate of left-handedness. (Because high levels of fetal testosterone are hypothesized to be associated with left-handedness, it is counterintuitive that male homosexuality is associated with sinistrality. James [1989] offers an intriguing explanation of this apparent paradox, suggesting that male homosexuality may arise from a different pattern of timing of prenatal androgen surges, with high androgen levels occurring when handedness is affected and low levels occurring when sexual orientation is affected.)

Regarding spatial ability, several studies have suggested that homosexual men score lower than heterosexual men on spatial tests (Sanders and Ross-Field 1986; Gladue et al. 1990; McCormick et al. 1991; Tkachuk and Zucker 1991). Similarly gender-nonconforming boys, who are likely to become homosexual men, also have been found to perform less well than controls on spatial tests (Finegan et al. 1982; Grimshaw et al. 1991). The one study focusing on women, however, found lesbians to obtain lower scores than heterosexual women (Gladue et al. 1990), a finding difficult to reconcile with a neuroendocrine theory.

Thus a reasonable number of studies suggest that there may be lateralization differences between homosexuals and heterosexuals, particularly among men (who have been studied more often). Nevertheless, they cannot be considered definitive proof for a neuroendocrine theory of sexual orientation. This is partly because of the mixed findings. It is troubling, for instance, that the largest studies of handedness found no association with sexual orientation. But, more important, neuroendocrine theories of both sexual orientation and lateralization are currently insufficiently specified to allow strong predictions, and hence confirmations, of either. Furthermore, the causes of the sex difference in spatial ability, particularly, remain controversial, with both "biological" and psychosocial explanations being offered (Linn and Petersen 1985).

Human Neuroanatomical Studies Potentially the most persuasive type of evidence for a neuroendocrine theory of sexual orientation is finding neuroanatomical differences between homosexuals and heterosexuals in areas of the brain hypothesized to be involved in sexual or related behavior. Studies of nonhuman animals have implicated the hypothalamus as the most likely site of interest. However, any part of the brain that is sexually dimorphic is of interest, given the likelihood that an influence affecting sexual differentiation of the hypothalamus would affect other areas of the brain as well.

Careful, systematic research on sex differences in the human brain has only recently begun, and studies of neuroanatomical correlates of human sexual orientation are rare indeed. Before this literature is evaluated, it is useful to put it into a methodological context. Neuroanatomical studies are typically enormously painstaking enterprises. Because of this, a research team usually investigates several brain locations of interest. Sometimes a difference is discovered after the researchers, looking at one part of the brain, notice a potential pattern elsewhere. Both these factors increase the probability of type 1 error, that is, the possibility that a difference occurs merely as a result of chance sample fluctuations. Hence, while replication in science is always important, replication of neurophysiological findings is especially so. By the same token, failures to replicate with small samples should not be considered definitive disconfirmation of initial findings, since small samples are associated with a high type 2 error rate.

Three highly publicized reports of brain structures are related to sexual orientation. The first, by Swaab and Hofman (1990), found the suprachiasmatic nucleus of the hypothalamus to be 1.7 times larger in homosexual than in heterosexual men. This nucleus is thought to be involved in the regulation of circadian rhythms and, as such, is a surprising location in which to find a sexual orientation difference. These authors also examined a nucleus that their research group (Swaab and Fliers 1985) had previously found to be sexually dimorphic, but found no difference between homosexuals and heterosexuals. The sexual orientation difference in the suprachiasmatic nucleus has not yet been replicated.

The most noted finding of a brain difference between homosexuals and heterosexuals was reported by LeVay (1991). LeVay investigated two hypothalamic nuclei that had previously been reported to be sexually dimorphic (Allen et al. 1989). He studied the brains of eighteen homosexual men, all of whom had died of acquired immune deficiency syndrome (HIV/AIDS), a comparison group of sixteen men whose sexual orientations were unknown

but presumed to be heterosexual, and a group of six women. One of the nuclei was not even found by LeVay to be sexually dimorphic. However, the third interstitial nucleus of the anterior hypothalamus (INAH-3) was less than half as large in the women as in the heterosexual men, replicating the previous researchers' findings. Furthermore, the nuclei of the homosexual men were also less than half the size of the heterosexual men's, and were indistinguishable from those of the women.

LeVay's findings have been subjected to intense scrutiny. For instance, it has been noted that the findings could be owing to the effects of AIDS rather than sexual orientation. However, LeVay demonstrated that his findings were robust even when the analysis was restricted to those heterosexual men who had died of AIDS. Another criticism has been that LeVay did not know for certain that his "heterosexual" group contained no homosexuals. However, any misclassification of subjects would diminish the obtained effect size relative to the true effect size. Finally, it should be emphasized that LeVay was studying a nucleus that had been found *twice* (counting LeVay's own demonstration) to be sexually dimorphic. Thus the a priori justification for his search was strong. Like all important findings, LeVay's should be replicated. The INAH-3 remains the most promising road to confirmation of a neuroendocrine theory of sexual orientation.

The most recent brain study, by Allen and Gorski (1992), demonstrated sex and sexual orientation differences in the anterior commissure (AC) of the corpus collosum, with heterosexual women's ACs being larger than heterosexual men's, but with homosexual men's the largest of all. Their search was motivated by a previous finding of a sex difference in the AC (Allen and Gorski 1991) and thus was well justified. Though apparently sexually dimorphic, the AC is not thought to be involved in sexual behavior. So this finding may reflect the generalized effects of neuroendocrine influences that also affect the areas of the brain that directly regulate sexual orientation.

Current Status of the Neuroendocrine View The neuroendocrine theory of sexual orientation is currently the most influential etiological theory of sexual orientation. Its empirical support, however, is largely indirect. The most careful and definitive studies of hormonal influence on sexually dimorphic sexual behavior have used nonhuman animals whose species-typical mating behaviors do not map directly onto ours. Rats do not seem to have a sexual orientation in the same way humans do, and it is controversial whether the behaviors that have been studied (primarily lordosis and mounting) are relevant at all. The most replicated relevant finding in humans, that sexual

orientation is related to childhood gender atypicality, is not clearly a biological phenomenon, though it does suggest that homosexuals and opposite-sex heterosexuals have been subject to similar influences, as predicted by a neuroendocrine view. Individuals who have been exposed to atypical levels of hormones are, in principle, quite relevant to the neuroendocrine theory; however, results of research on such persons have been mixed. Studies of sexually dimorphic traits related to brain lateralization such as spatial ability and handedness have also provided a complicated empirical picture. Furthermore, the neuroendocrine theory does not make strong predictions about the relationship between these traits and sexual orientation. Perhaps the most promising findings have been the demonstrations of neuroanatomical differences between heterosexual and homosexual men. If these findings are replicated (and extended to women), they may provide the long sought-after proof of neuroendocrine routes to human sexual orientation.

Genetics and Sexual Orientation

Human behavior genetics has produced evidence for substantial genetic factors in a wide variety of behavioral traits (Plomin 1990), from different types of psychopathology to personality and intelligence, even to characteristics such as religiosity (Waller et al. 1990). Indeed, failures to find significant heritability for well-measured traits in large sample studies have been exceedingly rare. Viewed from this perspective, it is hardly daring to hypothesize that sexual orientation may be heritable as well. However, sexual orientation is significantly different from the aforementioned characteristics. Homosexuals have presumably always been at a reproductive disadvantage compared to heterosexuals. (With respect to recent history, this disadvantage is demonstrably severe. Bell and Weinberg [1978; Tables 17.1 and 17.13] showed that both male and female homosexuals reported less than one-quarter of the number of children as same-sex heterosexuals.) If sexual orientation is somewhat heritable, this means that some genes predispose individuals to homosexuality. How have those genes resisted elimination by the inevitable engine of natural selection? Even at its lowest estimated base rates, homosexuality occurs far more frequently than the highest known mutation rates; thus mutation alone cannot account for the persistence of "gay genes," if they exist. The paradox of relatively high incidence and low fertility of homosexuals makes sexual orientation a likely candidate for low or zero heritability.

It is genuinely surprising, therefore, that the available evidence is more consistent with moderate to high heritability for both male and female sexual orientation (though relevant evidence is more plentiful for men). Before reviewing this evidence, it will be useful to explicate some basic genetic concepts.

Heritability If a trait is at least partially genetic, then it should be familial. Therefore the first step in a behavioral genetic investigation is generally to find if the trait of interest runs in families. Although necessary, familiality is not sufficient to justify a genetic conclusion, because traits can be familial for genetic or environmental reasons. Therefore more sophisticated approaches are needed subsequently, and these might be termed "heritability studies," because they have one common goal of providing a heritability estimate for the trait.

Heritability is the proportion of phenotypic variance that is explicable by genetic variance. Represented by a number ranging from 0 to 1, heritability is estimated in several ways, though the most intuitive is the intraclass correlation of monozygotic (MZ) twins who were reared separately in environments assigned at random. This method of estimating heritability is used infrequently, because of the extreme rarity of MZ twins who were reared apart. There are, for instance, only six separated MZ pairs with homosexuality (i.e., at least one twin is homosexual) in the literature (Eckert, Bouchard, Bohlen, and Heston 1986).

Perhaps the most widely used design for estimating heritability of human behavioral traits is the classical twin study, in which MZ and dizygotic (DZ) twins are compared for their degree of phenotypic similarity. The rationale is as follows: MZ twins are genetically identical (with rare exceptions), but DZ twins are not. MZ and DZ twins were both reared together and thus had equally similar rearing environments. Thus if MZ twins are more similar than DZ twins for a trait, it must reflect the increased genetic similarity. An important assumption on which the classical twin method depends is the "equal environments assumption," that the relevant environments are no more similar for MZ than for DZ twins. Although this assumption has been criticized (e.g., Lewontin et al. 1984), it seems to be accurate for traits studied so far (Plomin et al. 1989). Unfortunately it has not been specifically examined within the context of sexual orientation.

Because heritabilities may be different for different populations (e.g., Asians versus whites), and because heritability estimates typically have substantial standard errors around them, they should be viewed as approxima-

tions. I use the following very rough scale: less than .25 is low, .25 to .50 is moderate, and greater than .50 is high heritability. Note, however, that even a "low" heritability of .16 implies a correlation of .40 between genotype and phenotype. Furthermore, provided the assumptions required to compute heritability are valid, this correlation may be interpreted causally, that is, individual differences in genotype cause individual differences in phenotype. Given our present ignorance about causes of sexual orientation, one would be delighted to find a causal connection so large. On the other hand, if heritability were .16, environmentality would be .84, implying a correlation between relevant environment and phenotype of nearly .92. In this case environmental causes would dwarf genetic ones, though even the latter would be important enough to be interesting.

Familiality of Sexual Orientation Hirschfeld (1936) remarked more than fifty years ago that homosexuality appeared to run in families. However, rigorous confirmation of Hirschfeld's informal observations has taken a long time. In a landmark study Pillard and Weinrich (1986) demonstrated the plausibility of doing family genetic studies of homosexuality. Using newspaper advertisements they recruited fifty-one homosexual male and fifty heterosexual male probands, who were blind to the purpose of the study. During the interview they asked about the sexual orientation of all siblings. Furthermore, they asked (and received, to a large degree) permission to contact siblings. This was generally done through the mail, but some phone interviews were necessary. In the sibling questionnaires or interviews, several questions were asked about sexual orientation. Pillard and Weinrich showed that probands predicted their siblings' orientations with a high degree of accuracy. They also found substantially more gay brothers among gay male probands than among heterosexual male probands, 20 to 4 percent, respectively. This is roughly consistent with another family study of male homosexuality. Bailey et al. (1991) obtained estimates of the number of brothers "known" to be homosexual from heterosexual male and female probands, as well as homosexual male probands, finding a 10 percent rate among the brothers of homosexual men compared to a 2 percent rate among the brothers of heterosexual men and women. Although brothers were not contacted to verify their sexual orientations, the results of Pillard and Weinrich suggest that this is unnecessary.

Is female sexual orientation also familial? There have now been two reasonably large studies of this question. Pillard (1990) found a 25 percent rate of homosexuality or bisexuality among sixty sisters of bisexual or homosexual

female probands, compared to a rate of 11 percent for sisters of fifty-three heterosexual female probands. It is interesting that they also found a marginally significant elevation of homosexual brothers among their homosexual female probands. If replicated, this finding would have important implications for theories of sexual orientation: a common mechanism in the development of male and female homosexuality. Bailey and Benishay (1993) also reported significant familiality. Of the ninety-nine sisters of the homosexual probands, 12 percent were homosexual compared to 2 percent of the eighty-three sisters of the heterosexual probands. Although a slightly higher percentage of brothers of the homosexual probands were also homosexual (7 to 1 percent), this difference was not significant. Both Pillard (1990) and Bailey and Benishay (1993) obtained verification of probands' reports of their siblings' sexual orientation in the majority of cases, and both found such reports to be highly accurate.

Genetic Studies The first noteworthy genetic study of sexual orientation was done by Kallmann (1952a, 1952b), who found a 100 percent concordance rate for thirty-seven male MZ twin pairs compared to a 15 percent rate for twenty-six dizygotic male DZ pairs. Kallmann's study has been justifiably criticized for its methodology, particularly its reliance on sampling from correctional and psychiatric institutions, its lack of detail regarding zygosity diagnosis, and its anomalous findings. Most important, results of several case studies and small twin series (reviewed by Rosenthal 1970) suggest that the true MZ concordance rate, although appreciable, is substantially less than 100 percent. Because of these problems, the study is generally held in low regard. Nevertheless, no one has offered a plausible alternative to genetic influence to account for Kallmann's strikingly different concordance rates.

A fascinating report of MZ twins raised separately (Eckert et al. 1986) was consistent with a high heritability for male sexual orientation. Both the two male pairs included were concordant for homosexual feelings (though one of the twins classified himself as a heterosexual anyway). In contrast, in the same study none of four female pairs was concordant. Obviously the sample size of this study was too small to justify strong conclusions.

Given the promising results obtained by Kallmann, it is somewhat surprising that almost forty years passed before the question of heritability was again investigated using large samples. Seemingly by coincidence, four reasonably large studies have been reported in the last year. All four obtained samples through advertisements in gay publications. For instance, the first and largest, by Bailey and Pillard (1991), used advertisements that asked for

gay men with either male twins or adoptive brothers. (Adoptive brothers are biologically unrelated males reared as siblings to the probands.) Eligible and interested subjects called the investigators and were interviewed, usually in person, about their sexual orientations and related traits. They were also asked about their brothers' sexual orientation and, finally, for permission to contact their co-twins or adoptive brothers. As Pillard and Weinrich (1986) found, sibling reports were highly related to proband reports. The rates of homosexuality (including bisexuality) among the relatives were 52 percent (29/56) for MZ co-twins; 22 percent (12/54) for DZ co-twins; and 11 percent (6/57) for adoptive brothers. This pattern is consistent with moderate to strong heritability for male sexual orientation. Under varying assumptions, heritability estimates ranged from .31 to .74. The lowest estimates came from models that assumed a base rate of 10 percent for homosexuality. Recent reports suggest that the lower base rate of 4 percent is more appropriate (Johnson et al. 1992; ACSF 1992). All heritabilities computed assuming a 4 percent rate exceeded .50. One anomalous finding of the study was a lower than expected rate of homosexuality among the biological non-twin brothers of 9 percent. Genetic theory predicts that this rate should be equal to that for DZ twins and higher than that for adoptive brothers.

Because I have defined "sexual orientation" as sexual attraction rather than either identity or behavior, it is important to address one methodological issue of this study. Both probands and relatives were classified according to sexual identity, that is, whether they called themselves "gay/bisexual" or "heterosexual." This was done for three reasons. First, this was the easiest way to write the advertisements used to recruit probands. Second, in both groups sexual identity was closely related to both sexual behavior and sexual attraction patterns, as measured by the seven-point Kinsey scale (Kinsey et al. 1948). Third, probands' ratings of their relatives were used when relatives' self-ratings were unavailable, and it was believed that the concordance between probands' ratings and relatives' self-ratings would be higher for the broader categories of sexual identity than for the more specific Kinsey scores. Indirectly supporting this belief was the finding that although probands were almost perfect at predicting whether their relatives would identify as "heterosexual" versus "gay/bisexual," they did poorly at predicting whether a nonheterosexual relative would call himself "gay" or "bisexual." This method of classifying sexual orientation was also used in the female family study by Bailey and Benishay (1993) mentioned previously and in the genetic study of female sexual orientation by Bailey, Pillard, Neale, and Agyei (1993) discussed later.

The second genetic study, restricted to twins, was reported by King and

McDonald (1992). Using recruitment methods similar to those of Bailey and Pillard, they found a sample of forty-six homosexuals with twins (thirty-eight male; eight female). The reported concordances, 25 percent for MZ twins compared to 12 percent for DZ twins, appear to conflict with the higher rates obtained by Bailey and Pillard. However, King and McDonald's sample was considerably smaller, so that the difference might largely be owing to sampling error. Furthermore, it is unclear how zygosity was diagnosed in this study, nor were co-twins contacted to verify orientations, nor were results reported separately for men and women. Finally, it is noteworthy that five of seven respondents considered their co-twins entirely heterosexual despite an apparently prolonged incestuous homosexual relationship. The third study, by Whitam et al. (1992) found even higher concordances than did Bailey and Pillard—66 percent for MZ versus 30 percent for DZ pairs.

A fourth study (Bailey et al. 1993) focused exclusively on female sexual orientation. The methodology was identical to that of Bailey and Pillard's (1991) genetic study of males. Probands were homosexual or bisexual women with twins or adoptive sisters. Of the relatives whose sexual orientation could be confidently assessed, 48 percent (34/71) of MZ co-twins, 16 percent (6/37) of DZ co-twins, and 6 percent (2/35) of adoptive sisters were homosexual. Probands also reported that 14 percent (10/73) of their non-twin biological sisters were homosexual, a rate quite similar both to the DZ twin rate and to the rate found by Bailey and Benishay (1993) in their family study of female sexual orientation. Heritabilities were significant under a wide range of assumptions about the population base rate of homosexuality and ascertainment bias, and they were of the same order as those obtained by Bailey and Pillard for male sexual orientation (1991).

Thus the available genetic evidence suggests that both male and female sexual orientations are moderately heritable. However, the limitations of this literature must be recognized. All the genetic studies discussed herein used a method of subject ascertainment that may be susceptible to serious biases; that is, if gay men with gay co-twins are more likely than gay men with heterosexual co-twins to volunteer for such studies, the concordance rates will be artifactually higher. Bailey and Pillard (1991), however, demonstrated that this kind of bias cannot lead to spurious findings of heritability unless it is greater for MZ than for DZ twins. Given the general consistency of the picture obtained from family studies, the small study of twins reared apart (at least for men), and the four large twin studies, all of which found higher MZ than DZ concordance, it seems likely that the heritability findings are robust. However, large population-based twin studies (for a small version, see Buhrich et

al. 1991) or systematically obtained (and hence representative) samples of gay twins are necessary to address such methodological issues.

One striking result of the more recent genetic studies is the high rate of discordance among the MZ twins. Except for Kallmann's study, concordances have been well under 100 percent. This shows that environment exerts an influence on sexual orientation. It should be remembered, however, that "environment" refers simply to all nongenetic influences, biological or social. There is presently no good candidate for an environmental factor not shared by MZ co-twins that might affect sexual orientation.

Even if it could be demonstrated with certainty that sexual orientation were substantially heritable, the question would remain of what, exactly, the relevant genes are doing. Although most genetic researchers have been influenced by the neuroendocrine theory, a finding of nonzero heritability is merely consistent with it. Such a finding does not provide direct support for a neuroendocrine view because alternative genotype-to-phenotype routes involving social environmental factors are imaginable (see, e.g., Byne and Parsons 1993). Only if either a specific gene affecting sexual orientation or a genetic marker associated with it were identified could genetic data strongly confirm a neuroendocrine theory.

The Paradox of "Gay Genes" Revisited I have delayed discussion of possible explanations for the persistence of genes for sexual orientation until a consideration of the genetic data. While ultimately inconclusive, the genetic data seem strong enough to justify serious consideration of mechanisms by which "gay genes" might be maintained.

Several explanations have been proposed. A necessary feature of any such explanation is that, although the relevant genes detract from a gay individual's reproductive output, they facilitate reproduction in other carriers. One possible model, proposed by Hutchinson (1959), is that of heterozygote superiority, of which sickle-cell anemia is an example. Heterozygote superiority occurs when individuals homozygous for either of two alleles at a genetic locus (i.e., those who have two copies of either gene) have decreased reproductive success compared to heterozygotes (who have one copy of each). This model explains the persistence of genes for sickle-cell anemia, because heterozygotes are better protected against malaria than individuals without any sickling genes. Although plausible, this model is not very useful without a specification of the alleged advantage for heterozygotes.

The second explanation considered here is an application of an immensely important concept in evolutionary theory, namely, kin selection

(Hamilton 1964; Wilson 1978). Applied to sexual orientation, kin selection theory suggests that while homosexuals do not themselves reproduce at high rates, their sacrifice enables kin (most likely siblings and parents) to reproduce more than they otherwise would. Kin selection requires that this increased benefit be substantial: Siblings or parents must total at least two extra children for every one foregone by the homosexual individual. What is the specific mechanism by which homosexuals aid kin in reproduction? It has been proposed that homosexuals may be particularly likely to aid in rearing and to invest resources in their nieces and nephews (Weinrich 1987; Wilson 1978). This theory could be supported either by demonstrating increased fertility of relatives of homosexuals or by examining specific behaviors of homosexuals toward their relatives. Unfortunately no empirical studies of this theory have been attempted, so it remains unfounded speculation.

Related Phenotypes: Bisexuality and Transsexualism

Bisexuality In "biological" studies of sexual orientation, there are generally two approaches to bisexuals: They are either classified with homosexuals or they are excluded. Which approach is more justifiable?

From the vantage of a neuroendocrine theory, bisexuality need not be particularly problematic. Attraction to women means that sexual orientation has been masculinized. However, some unknown physiological process has prevented behavioral defeminization, and thus attraction to men occurs simultaneously. In this view it makes most sense to treat bisexuals as intermediate between heterosexuals and homosexuals. Until a neuroendocrine account of bisexuality garners scientific support, however, the etiological relationship between bisexuality and either homosexuality or heterosexuality remains an open, empirical question. Relevant evidence consists of whether bisexuals are more like homosexuals or heterosexuals with respect to relevant variables. For instance, Bell et al. (1981) found bisexual women to be intermediate between heterosexual and homosexual women in recollections of childhood gender nonconformity. No such pattern was reported for men. Consistent with the null finding for men, Freund (1974) found that men who self-labeled as bisexual were aroused to homosexual but not heterosexual erotic stimuli.

One empirical strategy, as yet untapped, would be to see whether patterns of familiality differed between bisexuals and homosexuals. For example, do bisexuals and homosexuals differ in their rates of homosexual siblings? Do

MZ co-twins of bisexual probands have a different distribution of sexual orientation from MZ co-twins of homosexual probands? One problem with doing such studies is that bisexuals appear to be somewhat rare, comprising a minority of nonheterosexuals in most samples. Bailey and Pillard (1991) obtained results supporting the hypothesis that homosexuality and heterosexuality may be more categorically than dimensionally distinct, with bisexuality a relatively rare occurrence. Verifying this observation could have important implications for understanding the development of sexual orientation.

Transsexualism Transsexualism is a rare phenotype characterized by a persistent dissatisfaction with one's anatomical sex (gender dysphoria) and a desire to change it. Blanchard (1987, 1989) has shown that there are two independent types of transsexualism, which he distinguishes as homosexual and heterosexual transsexualism. The former is characterized by attraction to the same sex, an early history of childhood gender nonconformity, early onset of gender dysphoria, and a fairly even sex ratio. The latter type of transsexuals are heterosexual without a history of childhood gender nonconformity and are nearly always male. Heterosexual transsexuals usually have a history of fetishistic cross-dressing (transvestism), which, in adulthood, evidently transforms into the desire to change sex. It is homosexual transsexualism that seems likely to be etiologically related to homosexuality. Besides the obvious similarities of a homosexual orientation and childhood gender nonconformity, Blanchard and colleagues have demonstrated other nonobvious similarities in homosexuality and homosexual transsexualism (Blanchard and Sheridan 1992a), including a sibling sex ratio biased toward males and a relatively late average birth order. On the other hand, they failed to find a high percentage of homosexual siblings among homosexual transsexuals (Blanchard and Sheridan 1992b).

Future Directions

There is much work to be done in articulating the "biological" mechanisms involved in the development of sexual orientation. Indeed, even most researchers who are engaged in, or otherwise sympathetic to, a biological research program freely admit that neuroendocrine or genetic hypotheses about sexual orientation have not been supported to a degree of certainty that would justify their acceptance. Nor can critics of these hypotheses rea-

sonably claim that they have been adequately falsified. There are roughly three broad programs of research that would further illuminate both the strengths and weaknesses of "biological" theories of sexual orientation: research on basic nonbiological questions of interest to homosexology in general, research designed to replicate the most important "biological" findings, and, given the results of the replication attempts, research aimed at elaborating promising "biological" findings.

All researchers studying sexual orientation are impeded by the absence of knowledge about some very basic facts. Foremost are incidence figures for sexual feelings, identification, and behavior. It is symptomatic of the sorry present state of knowledge that we still look to Kinsey's (1948, 1953) data to estimate these figures, despite the facts that those data are decades old, had severe sampling biases that Kinsey acknowledged at the time, and are cited by different writers to support a wide range of estimates. For example, many people are fond of citing a 10 percent figure for homosexuality in the general population; this figure derives from Kinsey's data. Gebhard (1972), however, used Kinsey's data to estimate the incidence of female homosexuality at 1.5 percent or less. Although these differences are partly explicable by use of different criteria for "homosexuality," Kinsey's data cannot yield trustworthy, specific current estimates. Successful future studies will attempt representative sampling, obtain high cooperation rates, and ask specific and detailed questions about both sexual feelings and behavior. Though a hostile political environment has impeded progress, at least three large relevant surveys are in progress (one American, one British, and one French). These studies were most immediately motivated by the need to obtain information relevant to the epidemiology of sex practices likely to transmit HIV. Let us hope, however, that the need to focus on specific sexual behaviors will not prevent the surveys from inquiring about sexual orientation and other psychological issues as well.

To illustrate the importance of knowing incidence figures more precisely, let us consider two examples. First, suppose one interviewed a large cohort of gay men and found the rate of homosexuality among their brothers to be 12 percent. If one accepts the high estimate of 10 percent for the base rate of homosexuality in the general population, then one will conclude that familial factors make only a trivial contribution to the development of sexual orientation in men. This is because brothers of gay men, in this case, do not have much of an increased rate of homosexuality. They are only 1.2 times more likely to be gay than is a man sampled randomly from the general population. In contrast, if one accepts Gebhard's (1972) estimate of 4 percent, then this is triple the expected rate, a fairly substantial increase. As a

second example, consider the possibility suggested by unsystematic research (e.g., Friday 1991) that homosexual fantasies may be quite common among women who identify themselves as heterosexuals. If such fantasies were similar in nature, frequency, and intensity to those of homosexually identified women, then it would seem unlikely that sexual identity merely reflected sexual feelings, per se. Why would only a relatively small minority of the women capable of homosexual arousal call themselves "lesbian" and mate more or less exclusively with other women? I emphasize that this example is hypothetical, and many of us who take a biological approach believe that heterosexual and homosexual women's sexual feelings differ in important respects. But we do not know this.

Another kind of research that is needed in homosexology, generally, is a systematic study of homosexuality across many diverse cultures. The research with which I am most familiar (e.g., Bailey and Pillard 1991; Bailey et al. 1993) primarily used white Americans as subjects, because they volunteered most frequently. Cross-cultural differences in the causes of sexual orientation are certainly conceivable and could illuminate the role of the social environment. Alternatively, it is possible, as Whitam and Mathy (1986) have suggested, that homosexuality develops similarly across cultures that seem to differ in important respects. If so, this would provide more support for a "biological" view.

The second broadly defined research program previously endorsed concerns replication. Many of the findings considered important for "biological" research were obtained in studies that had important methodological limitations. I have attempted to note the most serious limitations in the relevant sections of this paper. My purpose here is merely to emphasize that it is crucial to demonstrate beyond a reasonable doubt the validity of findings on which a theory rests. As Zubin (1987) pithily expressed it, "It ain't ignorance that causes all the trouble. It's knowing things that ain't so!" The most important study to replicate, in my view, is LeVay's (1991). If possible, it would be highly desirable to examine brains of lesbians as well. Furthermore, areas with mixed findings, such as the CAH literature, could greatly benefit from a large, controlled, and, one would hope, definitive study.

Provided that careful studies replicate the most promising findings supporting the neuroendocrine and genetic theories of sexual orientation, such as LeVay's neuroanatomical study and Bailey and Pillard's genetic study of male sexual orientation, the next phases of research should attempt to elaborate their deeper meanings. Thus, for example, does the INAH-3 affect sexual orientation, or is it noncausally associated with sexual orientation but masculinized along with causally relevant brain structures? A number of

techniques from contemporary neuroscience may be useful in answering such questions, including immunohistochemistry, hybridization histochemistry, positron emission tomography (PET), and magnetic resonance imaging (MRI). Assuming that careful studies replicate the heritability findings, the questions of which genes are involved and what they are doing remains. Methodologies such as linkage analysis are useful in theory for the identification of genes affecting a trait, and there is reason to hope that they will someday illuminate sexual orientation. As our knowledge of the genome progresses exponentially, so will the power of these techniques. However, to date they have not been very useful in studying behavioral characteristics, perhaps because these characteristics are typically etiologically complex (Plomin 1990). Even so, behavior genetics methods can be used to study genetic and environmental mechanisms indirectly.

For example, as I have noted previously, the high rate of discordance among the MZ twins shows that environment must exert an influence on sexual orientation. Furthermore, the effective environment for sexual orientation largely appears to include aspects of experience (social or biological) that differ between MZ twins who have been reared together. No current theories of sexual orientation would predict frequent discordance between MZ co-twins. Given the possibility of prenatal influences on sexual orientation, it would be useful to know whether aspects of the prenatal environment such as hormonal exposure often differ between MZ co-twins. The equivalent question on the psychosocial side is whether parental treatment of twins differs in ways likely to foster differences in sexual orientation.

Genetic mechanisms can be illuminated by identifying differences between probands of concordant MZ pairs and those of discordant MZ pairs. Probands of concordant pairs should have relatively high genetic loadings compared to those of discordant pairs. Bailey and Pillard (1991), for example, found that male MZ probands who had been gender atypical during childhood were neither more nor less likely than other probands to have gay co-twins. Hence childhood gender atypicality does not appear to be a marker of genetic influence on sexual orientation. This finding should be replicated, and other candidate markers should be investigated in this manner.

Future research should also attempt to integrate different "biological" approaches. For example, neuroanatomical studies of MZ twins discordant for homosexuality could illuminate the nature of environmental influences, while analogous studies comparing MZ homosexual probands from concordant versus discordant pairs could provide valuable information about the routes by which genes exert their influence.

Conclusion

The present state of biological research on biological influences on sexual orientation is one of inconclusive complexity. My own view is that the general area is an exciting and promising one. A theory can be considered promising for only a limited time, however. Without conclusive results, it will eventually be dismissed as disappointing, having failed to fulfill its original promise. The time is ripe for biological theories of sexual orientation to fulfill theirs.

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