

## What Causes Homosexuality?

This is the first and perhaps the most basic question about homosexuality. In order to understand the phenomenon of same-sex sexual relations, we must first explore what the research shows about the origins of such attractions.

There are two main theories as to what causes homosexual attractions. One is that a homosexual orientation is essentially dictated by genetic and or biological factors—put simply, that people are “born gay.” The other theory is that homosexual attractions develop as primarily as a result of psychological and environmental influences and early experiences. In the public square, the latter theory has appeared to be in decline and the former gaining favor in recent decades. But what does the research show? Let’s look at these two theories in turn.

### Are People “Born Gay?”

While the research of the infamous sex researcher Alfred Kinsey is often used by those seeking the moral approval of homosexuality, there is one point on which he is seldom quoted: his rejection of a biological origin for homosexuality.

- Kinsey’s colleague and biographer, Wardell Pomeroy, reports: “By the end of 1940 he had recorded more than 450 homosexual histories, enough to convince him that the psychologists were making matters worse by starting with the assumption that homosexuality was an inherited abnormality which could not be cured simply because it was inherent. Kinsey was convinced that there was absolutely no evidence of inheritance.”

Wardell B. Pomeroy, *Dr. Kinsey and the Institute for Sex Research* (New York: Harper & Row, 1972), 76.

## *Alleged evidence of the biological origin of homosexuality*

A handful of studies published during the 1990s have claimed to offer evidence in favor of a biological or genetic cause for homosexuality. Three of these in particular—a study of brain structure by Simon LeVay, a study of twins by J. Michael Bailey and Richard C. Pillard, and a study of “gene linkage” and “gene markers” by a team led by Dean H. Hamer—attracted considerable media attention and are largely responsible for the popular belief that a “gay gene” has already been found. Let’s look at these in turn.

### The Brain Studies of Simon LeVay

Some researchers have theorized that the sexual preferences and behavior of homosexuals may be dictated by the structure of the brain—particularly if the brains of homosexual men, for example, can be shown to resemble those of heterosexual women more than they resemble those of heterosexual men.

One highly publicized study that purported to demonstrate this was conducted in 1991 by former Salk Institute researcher Simon LeVay. LeVay studied the brains of cadavers, including 18 men known to have been homosexual and one known to have been bisexual. He compared them with the brains of another 16 men and six women whom he presumed to have been heterosexual. This is what LeVay claimed to have found:

- “INAH 3 was more than twice as large in the heterosexual men as in the women. It was also, however, more than twice as large in the heterosexual men as in the homosexual men. This finding indicates that INAH is dimorphic with sexual orientation [i.e., shows a difference in structure between homosexuals and heterosexuals], at least in men, and suggests that sexual orientation has a biological substrate.”

Simon LeVay, “A Difference in Hypothalamic Structure Between Heterosexual and Homosexual Men,” *Science*, 253: 1034 (August 1991).

### *Weaknesses of LeVay’s study*

- LeVay’s study, however, suffered from serious methodological errors, including the failure to adequately identify a control group. LeVay made questionable assumptions regarding the orientation of the “heterosexual” cadavers. He assumed that they were all heterosexual,

even though a number of the allegedly “heterosexual” subjects had died of AIDS, a disease that remains far more common among homosexual men than among heterosexuals: “Sixteen subjects were presumed to be heterosexual men: six of these subjects died of AIDS and ten of other causes.”

LeVay, “A Difference in Hypothalamic Structure Between Heterosexual and Homosexual Men,” 1035.

- Another anomaly of LeVay’s study was the fact that three of the “heterosexuals” had brain clusters smaller than the mean size for the homosexuals. On the other hand, three of the homosexuals had larger clusters than the mean size for “heterosexuals.” Thus, LeVay was forced to admit, “The existence of ‘exceptions’ in the present sample (that is, presumed heterosexual men with small INAH 3 nuclei, and homosexual men with large ones) hints at the possibility that sexual orientation, although an important variable, may not be the sole determinant of INAH 3 size.”

Ibid.

- LeVay, in fact, admitted that his claim of a *correlation* between this brain structure and sexual orientation could not prove *causation*, or even the direction of influence, noting that “[T]he results do not allow one to decide if the size of INAH 3 in an individual is the cause or consequence of that individual’s sexual orientation, or if the size of INAH 3 and sexual orientation co-vary under the influence of some third, unidentified variable.”

Ibid.

- All 19 of his homosexual subjects had died of AIDS, and LeVay noted that another “problem” was “the possibility that AIDS patients constitute an unrepresentative subset of gay men, characterized, for example, by a tendency to engage in sexual relations with large numbers of different partners or by a strong preference for the receptive role in anal intercourse,” both of which are major risk factors in acquiring human immunodeficiency virus (HIV) infection.

Ibid.

- A related issue is that the allegedly smaller brain clusters might not have caused homosexuality, but instead could have resulted from sexual activity or AIDS-related brain damage. “[T]here is the

possibility that the small size of INAH 3 in the homosexual men is the result of AIDS or its complications and is not related to the men's sexual orientation." He further allowed that until "tissue from homosexual men dying of other causes becomes available, the possibility that the small size of INAH 3 in these men reflects a disease effect that is peculiar to homosexual AIDS patients cannot be rigorously excluded."

Ibid., 1036.

### *Other researchers reject LeVay's findings*

- William Byne and Bruce Parsons, writing in *Archives of General Psychiatry*, also raised the question of how AIDS could have impacted LeVay's subjects, concluding that it is possible to "hypothesize a plausible mechanism by which human immunodeficiency virus infection" could account for a selective reduction in the volume of INAH3 in the homosexual men.
- Byne and Parsons also challenge LeVay's use of animal studies "to support the notion that the INAH3 is crucial to the 'generation of male-typical sexual behavior.'"
- Finally, they conclude that "LeVay's study can be faulted for a number of technical flaws, such as a variable method of tissue fixation, inadequate sexual histories, and small sample sizes."

William Byne and Bruce Parsons, "Human Sexual Orientation: The Biologic Theories Reappraised," *Archives of General Psychiatry*, 50: 235 (March 1993).

## Other Brain Studies

### *Theories concerning the anterior commissure*

Since LeVay, researchers have examined other areas of the brain to see if there are differences between homosexuals and heterosexuals.

- One study by L. S. Allen and R. A. Gorski (1991) reported that an area of the brain known as the anterior commissure (AC) "was

larger in homosexual as opposed to heterosexual men, a finding that was interpreted as support for the hypothesis that sexual orientation reflects the sexually differentiated state of the brain.”

- However, after reviewing the evidence, researchers Mitchell S. Lasco, et al., reported: “We examined the cross-sectional area of the AC in postmortem material from 120 individuals, and found no variation in the size of the AC with age, HIV status, sex, or sexual orientation.”

Mitchell S. Lasco, Theresa J. Jordan, Mark A. Edgar, Carol K. Petito, and William Byne, “A Lack of Dimorphism of Sex or Sexual Orientation in the Human Anterior Commissure,” *Brain Research*, 936 (2002): 95.

- Byne and Parsons add that even if Allen and Gorski’s findings *could* be replicated, “the size of the AC alone would tell us nothing about an individual’s sexual orientation because the overlap of AC size between homosexual and heterosexual men was tremendous (i.e., the size of the AC of 27 of 30 homosexual men fell within the range established by 30 heterosexual men). Because these authors relied heavily on the brains of the subjects with acquired immunodeficiency syndrome and provide little clinical history, their study is subject to many of the same interpretive difficulties as LeVay’s study of the hypothalamus.”

Byne and Parsons, “Human Sexual Orientation: The Biologic Theories Reappraised,” 235.

### *The suprachiasmatic nucleus*

Byne and Parsons also reject as unsubstantiated a report indicating that the size of another hypothalamic nucleus, the suprachiasmatic nucleus (SCN), is larger in homosexual than heterosexual men:

- “Again, however, this study has not been corroborated, and few studies of this sort have proved to be replicable in the past. But even if corroborated, this finding would not support the prenatal hormonal hypothesis, because in humans the size of the SCN does not vary with sex. Furthermore, existing evidence does not suggest a primary role for the SCN in the regulation of sexual behaviors.”

Ibid.

## *The corpus callosum*

Byne and Parsons mention yet another attempt to prove that the brain structure of homosexuals differs from that of heterosexuals:

- “There has also been recent speculation that the morphology of the corpus callosum may be found to be female-typical in homosexual men (LeVay. *New York Times*. October 7, 1991: letter). Such speculation is premature as the 23 studies that have sought sexual dimorphism [i.e., differences between men and women] in the corpus callosum have yielded conflicting results. Although the initial study...concluded that the splenium of the corpus callosum is larger ( $P=.08$ ) and more bulbous in women than in men, none of the 22 subsequent studies replicated the sex difference in splenial size. Furthermore, while some researchers did replicate the finding of a more bulbous splenium in women, others found it more bulbous in men and still others found no sex difference. As described by Byne, some of the negative studies have been unfortunately misinterpreted as successful replications.”

Byne and Parsons, “Human Sexual Orientation: The Biologic Theories Reappraised,” 235.

## Conclusion: Brain Studies

- Byne and Parsons conclude: “In summary, three as yet uncorroborated reports suggest that the size of three different brain structures may vary with sexual orientation in men. These reports must be viewed cautiously while replication studies are pending.” The authors note further that even if these inconclusive findings were consistently replicated, “we will not know whether the anatomic correlates are a cause or a consequence of sexual orientation.”

Ibid., 229, 235.

## The Bailey and Pillard Study of Twins

- Writing in the *Archives of General Psychiatry*, J. Michael Bailey and Richard C. Pillard claim to have found a higher rate of homosexuality among identical (“monozygotic”) and fraternal (“dizygotic”) twins than among adoptive siblings. They reported that “of the

relatives whose sexual orientation could be rated, 52 percent (29/56) of monozygotic cotwins, 22 percent (12/54) of dizygotic cotwins, and 11 percent (6/57) of adoptive brothers were homosexual...” The authors concluded that “the pattern of rates of homosexuality by type of relative was generally consistent with substantial genetic influence...”

J. Michael Bailey and Richard C. Pillard, “A Genetic Study of Male Sexual Orientation,” *Archives of General Psychiatry*, 48 (December 1991): 1089, 1094.

The findings of Bailey and Pillard, however, are not entirely consistent with a genetic theory.

### *Methodological deficiencies of Bailey and Pillard*

- Bailey and Pillard themselves alluded to problems with their sampling method: “The sampling method employed in this study falls short of the ideal genetic epidemiological study, which would involve systematic sampling from a well-specified population. In particular, although all recruiting advertisements stated that [subjects] were desired regardless of the sexual orientation of their relatives, there is no guarantee that volunteers heeded this request.”

Bailey and Pillard, “A Genetic Study of Male Sexual Orientation,” 1094.

- Byne and Parsons confirm that Bailey and Pillard did not employ “a systematically ascertained sample of twins. Subjects...were recruited through advertisements placed in homosexual-oriented periodicals and, therefore, may not be typical of the homosexual population at large.”

Byne and Parsons, “Human Sexual Orientation: The Biologic Theories Reappraised,” 230.

### *Twin study fails to support genetic hypothesis*

- As Byne and Parsons explain, in Bailey and Pillard’s study, “the concordance rate for homosexuality in nontwin biologic brothers was only 9.2 percent—significantly lower than that required by a simple genetic hypothesis, which, on the basis of shared genetic material, would predict similar concordance rates for dizygotic twins and nontwin biologic brothers. Furthermore, the fact that the concordance rates were similar for nontwin biologic brothers (9.2 percent) and

genetically unrelated adoptive brothers (11.0 percent) is at odds with a simple genetic hypothesis, which would predict a higher concordance rate for biologic siblings.”

Byne and Parsons, “Human Sexual Orientation: The Biologic Theories Reappraised,” 229.

- Bailey and Pillard themselves admit that the rate of homosexuality among nontwin biological siblings, as reported by the subjects, was “significantly lower than would be predicted by a simple genetic hypothesis and other published reports.”

Bailey and Pillard, “A Genetic Study of Male Sexual Orientation,” 1089.

- In their analysis, Byne and Parsons point out that the evidence actually suggests an environmental rather than a genetic cause for homosexuality, arguing that “we must at least consider the possibility that the higher concordance rate for homosexuality in dizygotic twins compared with nontwin biologic brothers is due to increased similarity of the trait-relevant environment in the former. This is because dizygotic twins and full biologic siblings share the same proportion of genetic material. Thus, any difference in the true concordance rates would be attributable to environmental rather than genetic factors.”

Byne and Parsons, “Human Sexual Orientation: The Biologic Theories Reappraised,” 229–30.

### *Other twin studies fail to support the genetic theory*

- A study in *The Journal of Sex Research* examined monozygotic and dizygotic twins in the Minnesota Twin Registry. While the study claimed to find “significant genetic effects” for the sexual orientation of women, no such effects were found for men: “For men, no significant genetic effects were found for number of opposite- and same-sex sexual encounters, nor for sexual orientation.”

Scott L. Hershberger, “A Twin Registry Study of Male and Female Sexual Orientation,” *The Journal of Sex Research*, 34 (2): 212 (1997).

- The study concluded that environmental factors were a primary component of the formation of sexual orientation: “Special sibling environment effects were found for self-identified sexual orientation for male and female MZ [monozygotic] twins and opposite-sex

female DZ [dizygotic] twins.” The authors concluded, “Environmental effects were also important for sexual orientation, in fact, more important in the aggregate than genetic effects...”

Hershberger, “A Twin Registry Study of Male and Female Sexual Orientation,” 220, 221.

- Bailey and Pillard themselves note other twin studies that were unable to demonstrate a genetic cause: “Buhrich et al reported a twin study of sexual orientation and related behaviors...They found a strong familial resemblance, but had insufficient power to determine whether that correlation was due to genetic or environmental factors or both.”

Bailey and Pillard, “A Genetic Study of Male Sexual Orientation,” *Archives of General Psychiatry*, 1090.

- Miron Baron, writing in the *British Medical Journal*, also questioned the results of the twin studies that have been conducted: “Most of these results are uninterpretable because of small samples or unresolved questions about phenotypic classification, the selection of cases, and the diagnosis of twin zygosity or because they make the untested assumption that monozygotic and dizygotic twins have similar environmental experiences such that any difference in concordance rate would be genetic in origin.” Baron concluded: [T]he finding that the adoptive brothers of homosexual twins are more prone to homosexuality than the biological siblings suggests that male homosexuality may well be environmental.”

Miron Baron, “Genetic linkage and male homosexual orientation,” *BMJ*, 307: 337 (7 August 1993).

- If homosexuality were a trait determined entirely by a person’s genes, one would expect 100 percent of the identical (monozygotic or MZ) twins of homosexuals to also be homosexual. Yet this is not the case; indeed, “what is most intriguing” about the twins studies to Byne and Parsons “is the large proportion of MZ twins who were discordant for homosexuality despite sharing not only their genes but also their prenatal and familial environments. The large proportion of discordant pairs underscores our ignorance of the factors that are involved, and the manner in which they interact, in the emergence of sexual orientation.”

Byne and Parsons, “Human Sexual Orientation: The Biologic Theories Reappraised,” 230.

## Gene Studies of Dean Hamer

A flurry of media reports in 1993 indicated that scientists had at long last discovered a “gay gene.” The reports were based on the work of geneticist Dean Hamer of the National Cancer Institute. Hamer, however, never claimed to have found a gene that inevitably determines that a person will be homosexual. Rather, he claimed to have located a genetic component to some instances of male homosexuality.

Dean H. Hamer, *et al.*, “A Linkage Between DNA Markers on the X Chromosome and Male Sexual Orientation,” *Science* 261 (1993): 321–327.

- Hamer writes: “The role of genetics in male sexual orientation was investigated by pedigree and linkage analyses on 114 families of homosexual men....The goal of our work was to determine whether or not male sexual orientation is genetically influenced. We used the standard techniques of modern human genetics, namely pedigree analysis and family DNA linkage studies.”

Hamer, *et al.*, “A Linkage Between DNA Markers on the X Chromosome and Male Sexual Orientation,” 321.

Of the families Hamer interviewed that had more than one son who was homosexual, a significantly larger number had a maternal uncle or a maternal aunt’s son who was also homosexual. By comparison, the links with paternal linkage were weaker. This would suggest a maternal linkage for male homosexuality in some cases. Finding homosexual brothers who had homosexual maternal uncles would indicate that the gene determining homosexuality was transmitted through the mother’s family line.

After studying 40 pairs of brothers who were homosexual, Hamer hypothesized that a certain genetic marker on the X chromosome was at least partially responsible for their homosexuality. Since men have an X and a Y chromosome, and they inherit their X chromosome from their mothers, Hamer theorized that the mother may be the carrier of the gene determining homosexuality in their sons. Homosexual behavior would not be manifested in the mothers’ lives, but they would pass that gene on to their sons.

Hamer’s study is known as “linkage” study, where researchers isolate traits found in an extended family and then looks for a common DNA segment, or marker, on a particular chromosome. If the same marker is present consistently in the family members who have that trait, it is theorized that the

marker may be the gene that causes—or “codes”—for that trait. Linkage studies have successfully located genes that cause Huntington’s disease, cystic fibrosis, and muscular dystrophy. However, to date linkage studies have not found genes that code for complex behaviors.

### *Hamer’s findings*

- Hamer claimed: “We have now produced evidence that one form of male homosexuality is preferentially transmitted through the maternal side and is genetically linked to chromosomal region Xq28....[I]t appears that Xq28 contains a gene that contributes to homosexual orientation in males.”

Hamer, *et al.*, “A Linkage Between DNA Markers on the X Chromosome and Male Sexual Orientation,” 325.

### *What did Hamer not find?*

- Hamer did not claim to have found that homosexuality is directly inherited, like eye color: “Although the observed rates of homosexual orientation in the maternally derived uncles and male cousins of gay men were higher than in female and paternally related male relatives, they were lower than would be expected for a simple Mendelian trait.”
- In addition, Hamer did not claim that all cases of homosexuality could be explained by the presence of this gene marker: “[T]here was a substantial number of families in which lesbians or paternally related gay men were present. This could be explained if some instances of homosexuality were male-limited and maternally inherited whereas others were either sporadic, not sex-limited, or not maternally transmitted.”

*Ibid.*, 322.

- In fact, Hamer did not even attempt to estimate what proportion of the instances of homosexuality could be linked to this gene marker: “At present, we can say nothing about the fraction of all instances of male homosexuality that are related or unrelated to the Xq28 candidate locus...”

*Ibid.*, 325.

- He furthermore admitted the influence of environmental factors: “Given the overall complexity of human sexuality, it is not surprising that a single genetic locus does not account for all of the observed variability. Sib-pairs that are discordant at Xq28 should provide a useful resource for identifying additional genes or environmental, experiential, or cultural factors (or some combination of these) that influence the development of male sexual orientation.”

Hamer, *et al.*, “A Linkage Between DNA Markers on the X Chromosome and Male Sexual Orientation,” 325–26.

### *Linkage studies of other traits give reasons for caution*

- Baron summarizes: “There are lessons too from other studies—for example, of the hypothesis that another behavioural trait, manic depressive illness, is X linked. Support for this hypothesis was initially furnished by segregation patterns consistent with X linked transmission and reports of linkage to chromosomal region Xq27-28. In some studies the statistical support for these findings far exceeded the significance levels reported by Hamer *et al.* Moreover, the evidence from twin and adoption studies for a genetic component in manic depressive illness was far more compelling than that for homosexuality. Unfortunately, non-replication of the linkage findings by other investigators, as well as extension and reevaluation of the original data, has resulted in diminished support for this hypothesis. This outcome underscores the uncertainties in linkage studies of complex behavioural traits.”

Miron Baron, “Genetic linkage and male homosexual orientation,” *BMJ*, 307: 338 (7 August 1993).

- George Rice, et al., writing in *Science*, notes that “the evidence for X linkage has been questioned on theoretical and empirical grounds (8, 9). Most would agree that male homosexual orientation is not a simple Mendelian trait. There would be strong selective pressures against such a gene. Hamer’s identification of a contribution from a gene near Xq28 to homosexuality in some families that were selected for X-linked transmission of that trait might be fraught with type 1 (false positive) error. This is important to consider, given the irreproducibility of many linkage reports for complex behavioral traits.”

George Rice, Carol Anderson, Neil Risch, and George Ebers, “Male Homosexuality: Absence of Linkage to Microsatellite Markers at Xq28,” *Science*, 284: 666 (April 1999).

### *Hamer's findings have not been replicated*

- The 1999 study in *Science* by Rice, et al., attempted without success to duplicate Hamer's findings. "Sharing of alleles at position Xq28 was studied in 52 gay male sibling pairs from Canadian families. Four markers at Xq28 were analyzed....Allele and haplotype sharing for these markers was not increased over expectation. These results do not support an X-linked gene underlying male homosexuality."

Rice, *et al.*, "Male Homosexuality: Absence of Linkage to Microsatellite Markers at Xq28," 665.

- The authors write: "It is unclear why our results are so discrepant from Hamer's original study (6). Because our study was larger than that of Hamer *et al.*, we certainly had adequate power to detect a genetic effect as large as was reported in that study. Nonetheless, our data do not support the presence of a gene of large effect influencing sexual orientation at position Xq28."

*Ibid.*, 667.

### *A key problem: If homosexuality were genetic, it would have died out*

- Baron explains a key objection to "gay gene" theories: "Support for a genetic hypothesis is further complicated by cultural and evolutionary considerations....Sexual patterns are to some extent a product of society's expectations, but it would be difficult to envisage a change in the prevalence of a genetic trait merely in response to changing cultural norms. Also, from an evolutionary perspective, genetically determined homosexuality would have become extinct long ago because of reduced reproduction. Thus the purported linkage stands in apparent contradiction to the flimsy genetic and epidemiological evidence....[A] single gene or a particular genetic mechanism is unlikely to explain most of the variance in a phenomenon as complex as sexual orientation. Whether or not this sample is truly representative of familial homosexuality is an open question."

Miron Baron, "Genetic linkage and male homosexual orientation," *BMJ*, 307: 337 (7 August 1993).

- Bem also discusses the theory that homosexuality is "an evolutionary anomaly," and asks the question: "How do lesbians and gay men manage to pass on their gene pool to successive generations? Several

hypothetical scenarios have been offered....Although these speculations have been faulted on theoretical, metatheoretical, and empirical grounds (Futuyma & Risch, 1983/84), a more basic problem with such arguments is their circularity. As Bleier has noted about similar accounts, this logic makes a *premise* of the genetic basis of behaviors, then cites a certain animal or human behavior, constructs a speculative story to explain how the behavior (if it were genetically based) could have served or could serve to maximize the reproductive success of the individual, and this conjecture then becomes evidence for the *premise* that the behavior was genetically determined. (1984, p. 17)”

Daryl J. Bem, “Exotic Becomes Erotic: A Developmental Theory of Sexual Orientation,” *Psychological Review*, 103 (2): 328–29 (1996).

## Theories that Hormone Levels Influence Sexual Orientation

The theory that homosexuality was the result of a deficiency of male sex hormones in male homosexuals and, conversely, excessive levels of testosterone in lesbians, was repeatedly raised from the 1940s through the late 1970s.

- Byne and Parsons note that such ideas persisted “despite the failure of hormone treatments to influence sexual orientation and despite the fact that most studies failed to find any association between adult hormone levels and sexual orientation.”

Byne and Parsons, “Human Sexual Orientation: The Biologic Theories Reappraised,” 228.

### *Prenatal hormone levels*

When research data failed to support this theory, attention turned from adult hormonal levels to the levels of hormones in the womb.

- Bem explains that, reasoning from research on rats, “some researchers hypothesized that human males who are exposed prenatally to substantially lower than average amounts of testosterone and human females who are exposed to substantially higher than average amounts of testosterone will be predisposed toward a homosexual orientation in adult life.”

Bem, “Exotic Becomes Erotic: A Developmental Theory of Sexual Orientation,” 329.

However, other researchers questioned comparing hormonal levels in rats with humans.

- Byne and Parsons explain: “The problems inherent in extrapolating from mating behaviors and postures in rodents to psychological processes in humans are complex...Motivated sexual behaviors in humans are unlikely to be under such rigid endocrine control. Thus, the suitability of...behavior in rodents as a model for motivated sexual behavior in humans is questionable....It is difficult to imagine that the gamut and plasticity of human sexual behavior can be reduced to factors as simple” as the way in which a female rat responds to a male.

Byne and Parsons, “Human Sexual Orientation: The Biologic Theories Reappraised,” 231.

- Other researchers claimed to find a link between prenatal hormonal levels and lesbianism by interviewing women who have congenital adrenal hyperplasia (CAH), a chronic endocrine disorder that exposes them to abnormally high levels of androgen during the prenatal period—levels comparable to those received by males during gestation. According to Bem, “CAH women have now reported more bisexual or homosexual responsiveness than control women.”

Bem, “Exotic Becomes Erotic: A Developmental Theory of Sexual Orientation,” 329.

However, other researchers question any direct link between prenatal hormones to sexual orientation:

- Bem notes that both boys and girls who were exposed to abnormally high levels of androgen during gestation exhibited “increased aggression later in childhood (Reinisch, 1981), and girls with CAH have shown stronger preferences for male-typical activities and male playmates in childhood than control girls.” Thus, according to Bem, “the major reason for expecting CAH girls to be disproportionately homoerotic in adulthood is that they are overwhelmingly likely to feel different from other girls. Not only are they gender nonconforming in their play activities and peer preferences, as most lesbians are during their childhood years, but the salience of the CAH status itself aids and abets their perception of being different from other girls on gender-relevant dimensions.”

Ibid., 329–30.

- Byne and Parsons summarize the available evidence: “[I]f the pre-

natal hormonal hypothesis is correct, then one might expect to find homosexuality in a large proportion of males with syndromes involving prenatal androgen deficiency or insensitivity, and also in females with syndromes involving androgen excess. However, extensive reviews of the literature suggest that this is not the case.” They conclude: “Currently, data pertaining to possible neurochemical differences between homosexual and heterosexual individuals are lacking.”

Byne and Parsons, “Human Sexual Orientation: The Biologic Theories Reappraised,” 230–32.

## Genetic Characteristics May Play an Indirect Role in Sexual Orientation

To date, all theories regarding the existence a “gay gene” remain unsubstantiated. However, some researchers suggest that genetics may play an indirect role through the presence of certain temperamental traits that increase the likelihood that certain individuals will experience same-sex attractions or come to identify themselves as homosexual.

- Byne and Parsons explain: “For example, if a gene influenced some factor, such as temperament, in a manner that would increase the probability of homosexual development in a particular environment, that gene could be called a gene for homosexuality with reduced penetrance.” However, the authors caution: “Such terminology, however, would minimize the overriding importance of environment in such a scenario.”

*Ibid.*, 230.

- Bem agrees that “biological factors influence sexual orientation only indirectly, by intervening earlier in the chain of events to determine a child’s temperaments and subsequent activity preferences.... [C]orrelation between a biological factor and sexual orientation is more plausibly attributed to its influence in early childhood than to a direct link with sexual orientation.”

Bem, “Exotic Becomes Erotic: A Developmental Theory of Sexual Orientation,” 327.

Much of the confusion about what research has actually shown regarding a

genetic influence on the development of homosexuality has to do with a misunderstanding of the difference between a trait that is “heritable” (that is, one that runs in families) and one that is “inherited” (that is, one which is directly determined by genes). The difference, and the multiple pathways that could lead to homosexuality, were acknowledged by Bailey and Pillard of the twin studies:

- “Heritability is not informative regarding the development of sexual orientation (or, for that matter, of any trait). That is, given any heritability estimate, there are a variety of possible developmental mechanisms. For instance, these data are consistent with heritable variation in prenatal brain development or in some aspect of physical appearance that, by way of differential parental treatment, leads to differences in sexual orientation....[O]ne assumption of the heritability analyses presented above is that *there are no major genes for homosexuality...*” [emphasis added].

J. Michael Bailey and Richard C. Pillard, “A Genetic Study of Male Sexual Orientation,” *Archives of General Psychiatry*, 48: 1095 (December 1991).

### *Biological theories of homosexuality*

- Byne and Parsons conclude that the biologic theory remains unproven, and note that “the appeal of current biologic explanations for sexual orientation may derive more from dissatisfaction with the present status of psychosocial explanations than from a substantiating body of experimental data. Critical review shows the evidence favoring a biologic theory to be lacking. In an alternative model, temperamental and personality traits interact with the familial and social milieu as the individual’s sexuality emerges. Because such traits may be heritable or developmentally influenced by hormones, the model predicts an apparent nonzero heritability for homosexuality without requiring that either genes or hormones directly influence sexual orientation per se.”

Byne and Parsons, “Human Sexual Orientation: The Biologic Theories Reappraised,” 228.

- Bem concurs that “a theoretical rationale for a direct path between the genotype and sexual orientation has not even been clearly articulated, let alone established.”

Bem, “Exotic Becomes Erotic: A Developmental Theory of Sexual Orientation,” 328.

## The Political Agenda Behind Promoting the “Gay Gene”

- Revealingly, Byne and Parsons note: “Finally, political arguments have been offered in favor of biologic causation. It has been suggested that if sexual orientation is largely a biologic phenomenon, ‘society would do well to reexamine its expectations of those who cannot conform’; and, writing in the ‘Opinions and Editorials’ pages of the *New York Times* (December 17, 1991: 19), Bailey and Pillard stated: ‘If true, a biological explanation is good news for homosexuals and their advocates.’ However, political arguments have no impact on biologic realities, including the extent of genetic or hormonal influences on the emergence of sexual orientation.”

Byne and Parsons, “Human Sexual Orientation: The Biologic Theories Reappraised,” 236.

### *How the media distort the issue*

- Bem refers to the role that the media play in distorting the scientific evidence and misleadingly assuming that there exists a “gay gene”: “Like all well-bred scientists, biologically oriented researchers in the field of sexual orientation dutifully murmur the mandatory mantra that correlation is not cause. But the reductive temptation of biological causation is so seductive that the caveat cannot possibly compete with the excitement of discovering yet another link between the anatomy of our brains and the anatomy of our lovers’ genitalia. Unfortunately, the caveat vanishes completely as word of the latest discovery moves from *Science* to *Newsweek*. The public can be forgiven for believing that research is but one government grant away from pinpointing the [sexual] preference gene.”

Bem, “Exotic Becomes Erotic: A Developmental Theory of Sexual Orientation,” 330.

## Do Upbringing, Experience, and the Social Environment Contribute to the Development of Homosexuality?

In 1973 the American Psychiatric Association removed homosexuality from its list of mental disorders. That decision did not come as a result of new research. Ronald Bayer, author of the most exhaustive treatment of the 1973 decision, has described what actually happened:

- “A furious egalitarianism that challenged every instance of authority had compelled psychiatric experts to negotiate the pathological status of homosexuality with homosexuals themselves. The result was not a conclusion based on an approximation of the scientific truth as dictated by reason, but was instead an action demanded by the ideological temper of the times.”

Ronald Bayer, *Homosexuality and American Psychiatry: The Politics of Diagnosis* (Princeton, NJ: Princeton University Press, 1987), 3.

Prior to 1973 an extensive literature existed on the role of upbringing and experience in the development of homosexuality. Yet one of the unfortunate effects of the APA decision was to largely stifle further research on the psychological origins of homosexuality.

In the remainder of this chapter we will examine parts of that large body of work showing the key developmental influences, as well as looking at more recent research supporting developmental theories of homosexuality.

### *Early childhood developmental factors*

The causes of same-sex attraction are many and varied. Prior to 1973 many researchers focused on the early childhood years:

- A 1969 study in the *Journal of Consulting and Clinical Psychology* concluded: “We found a remarkable number of conditions and circumstances that may result in homosexuality...What happens after the child is born is complicated by many factors; there are not only inner biological and emotional factors, parental and familial surroundings, social and cultural circumstances; but the various pressures and expectations shift as the child grows and hardens as he establishes his ways into his eventual adult character structure.”

Ralph H. Gundlach, “Childhood Parental Relationships and the Establishment of Gender Roles of Homosexuals,” *Journal of Consulting and Clinical Psychology*, 33 (April 1969): 137.

- Similarly, Psychiatrists Byne and Parsons, writing in *Archives of General Psychiatry*, state that “it seems reasonable to suggest that the stage for future sexual orientation may be set by experiences during early development, perhaps the first 4 years of life.” The authors conclude: “The inadequacies of present psychosocial explanations do not justify turning to biology by default—especially when, at present,

the biologic alternatives seem to have no greater explanatory value. In fact, the current trend may be to underrate the explanatory power of extant psychosocial models.”

William Byne and Bruce Parsons, “Human Sexual Orientation: The Biologic Theories Reappraised,” *Archives of General Psychiatry*, 50 (March 1993): 236.

- Back in 1968 Ralph R. Greenson, clinical professor of psychiatry at UCLA, offered the following developmental theory, which focuses on the need of boys to “dis-identify” from their mother: [T]he male child, in order to attain a healthy sense of maleness, must replace the primary object of his identification, the mother, and must identify instead with the father. I believe it is the difficulties inherent in this additional step of development, from which girls are exempt, which are responsible for certain special problems in the man’s gender identity, his sense of belonging to the male sex....The male child’s ability to dis-identify will determine the success or failure of his later identification with his father.”

Ralph R. Greenson, “Dis-Identifying From Mother: Its Special Importance for the Boy,” *International Journal of Psychoanalysis*, 49 (1968): 370.

## *Parental influence*

Another focus of researchers has been how the personality traits of the parents may contribute to same-sex attraction, Theories about how a child’s relationship with his or her parent can effect homosexual feelings can be traced in the psychiatric literature going back nearly a century:

- According to the *Archives of Sexual Behavior* “Freud (1916) described the mothers of homosexuals as excessively loving and their fathers as retiring or absent. Stekel (1930) noted strong, dominant mothers and weak fathers. In 1936, Terman and Miles found the mothers of homosexuals to be especially demonstrative, affectionate, and emotional, while the fathers were typically unsympathetic, autocratic, or frequently away from home.”

Marvin Siegelman, “Parental Background of Male Homosexuals and Heterosexuals,” *Archives of Sexual Behavior*, 3 (1974): 3-4.

- Similarly, the *Bulletin of the Menninger Clinic* noted the following from a review of the literature back in 1963: “Bender and Paster in a study of 19 actively homosexual children, found either a grossly defi-

cient or very negative relationship with the same-sex parent, coupled with an overly intimate attachment to the opposite-sex parent.”

- “And, in a recent publication by West, a number of contemporary investigators are cited who independently have reached the same conclusion concerning the mother-son factor in male homosexuality. In this same publication, West presents his own study in England of 50 homosexual males and 50 matched control (nonhomosexual) males. His findings clearly show that male homosexuals are much more likely to come from a family constellation involving an overintense mother and unsatisfactory father relationship.”

Daniel G. Brown, “Homosexuality and Family Dynamics,” *Bulletin of the Menninger Clinic* 27 (5): 229–30 (Sept. 1963).

## The Work of Irving Bieber

A study conducted by a team of researchers headed by Irving Bieber, published as a book in 1962, is still considered a landmark in the field. Bieber, an influential researcher in the field of the etiology of homosexuality in the 1960s, summarized the team’s findings this way:

- “The ‘classical’ homosexual triangular pattern is one where the mother is CBI [close-binding-intimate] with the son and is dominant and minimizing toward a husband who is a detached father, particularly a hostile-detached one. From our statistical analysis, the chances appear to be high that any son exposed to this parental combination will become homosexual or develop severe homosexual problems.”

Irving Bieber et al., *Homosexuality: A Psychoanalytical Study* (New York: Vintage Books, 1962), 172.

- Subsequent studies confirmed Bieber’s findings. In 1964 a British psychiatrist compared his patients who were either homosexuals or neurotic heterosexuals, and reported that “approximately 70 percent of the homosexuals (62 percent plus one-third of 28 percent) were either over-attached to their mother or did not get on well with their father.”

P. J. O’Connor, “Aetiological Factors in Homosexuality as Seen in Royal Air Force Psychiatric Practice,” *British Journal of Psychiatry*, 110 (May 1964): 384–85.

- A 1965 study of homosexual college students in *Genetic Psychology Monographs* also generally confirmed Bieber's work: "Our findings are similar. Whereas the percent of close-binding-intimate mothers was 55 for the Homosexuals, the corresponding percent for the Controls was only 20." In addition, "Bieber *et al.* found a little less than twice as many detached-hostile and detached-indifferent fathers among their Homosexuals as compared with their control group. We had 42 percent detached fathers for the Homosexuals and 24 percent for the Controls. Whereas in Bieber's study most of the fathers were detached and *hostile*, our trend was that the fathers were rather detached and *indifferent*. The present authors feel that the function of the *detached* father in the psychogenesis of male homosexuality deserves a more important place than hitherto it has been given."

Leif J. Braaten and C. Douglas Darling, "Overt and Covert Homosexual Problems Among Male College Students," *Genetic Psychology Monographs*, 71 (1965): 302-03.

- Daniel G. Brown reported that when he served as a psychiatrist in the U.S. Air Force, "there was the opportunity to interview and test approximately 40 male airmen in whom predominant or exclusive homosexuality was the major problem. In more than 30 of these cases, the mother-son and father-son relationship conformed to the family pattern described above. Not one of these airmen had a close, warm, affectionate attachment to his father or a father-substitute in childhood."

Daniel G. Brown, "Homosexuality and Family Dynamics," *Bulletin of the Menninger Clinic* 27 (5): 229 (Sept. 1963).

### *Evelyn Hooker questions Bieber's findings*

UCLA professor Evelyn Hooker was the author of numerous studies that purported to show that homosexuality does not involve pathology. Hooker criticized Bieber's work because it involved subjects who were undergoing psychiatric treatment and thus the results are presumably not applicable to the broader population of homosexuals. Hooker, who has been accused of introducing methodological errors and bias into her own work, claimed:

- "The etiological role of parental relationships in producing homosexuality is an *inference* which cannot be justified from psychiatric samples alone, in part because of the contamination of homosexuality with psychopathology."

## *Reply to Hooker's criticism*

What Hooker failed to acknowledge was that the study of research subjects under medical evaluation and treatment is a widely-used and valid research method. In addition, Hooker herself has been criticized for recruiting research subjects from radical homosexual groups and others with an obvious agenda to promote (see Thomas Landess, "The Evelyn Hooker Study and the Normalization of Homosexuality," [Family Research Council, 1995]).

Meanwhile, other research drawn from subjects in the general population confirmed the work of Bieber and others.

## *The contribution of Ray B. Evans*

In 1969 a study by Ray B. Evans of the Loma Linda University School of Medicine was published in the *Journal of Consulting and Clinical Psychology*. It addressed Hooker's criticism and supported Bieber's findings of greater family dysfunction in the childhood of homosexuals.

- Whereas Bieber's data was "based on psychoanalysts' reconstructions of patients' early life circumstances, derived from impressions during psychotherapy ... in the present study, the data were based on retrospective self-reports of how they now view their childhood, by (study subjects) who had never been in psychotherapy." Study author Ray B. Evans of the Loma Linda University School of Medicine concluded: "The present results were remarkably similar to those of Bieber et al. in revealing more 'negative' features in the backgrounds of homosexuals."

Ray B. Evans, "Childhood Parental Relationships of Homosexual Men," *Journal of Counseling and Clinical Psychology*, 33 (April 1969): 129, 133.

- Describing his findings in more detail, Evans reports that the mothers of homosexuals "more often were considered puritanical, cold toward men, insisted on being the center of the son's attention, made him her confidant, were 'seductive' toward him, allied with him against the father, openly preferred him to the father, interfered with his heterosexual activities during adolescence, discouraged masculine attitudes and encouraged feminine ones. The fathers of the homosexuals were retrospectively considered as less likely to encourage masculine attitudes and activities, and (the study subjects) spent little time with their fathers, were more often aware of hating him and afraid he might physically harm them, less often were the

father's favorite, felt less accepted by him, and in turn less frequently accepted or respected the father....The results strongly suggested poor parental relationships during childhood for the homosexual men, at least as seen in retrospect."

Evans, "Childhood Parental Relationships of Homosexual Men," 133.

### *Was Evans too cautious regarding his conclusions?*

- Evans concluded his article with a note of caution as to whether his findings show that poor parental relationships cause homosexuality: "The results of the present study agreed closely with those obtained by Bieber et al.. but they neither supported nor refuted the Bieber conclusions as to causal relationships."

Evans, "Childhood Parental Relationships of Homosexual Men," 135.

However, two other scholars, asked to comment on Evans' article in the same issue of *Journal of Consulting and Clinical Psychology*, said that his caution was unwarranted:

- The first, Ralph H. Gundlach, flatly stated that "Evans' argument, that an association between questionnaire items regarding parent-to-child behaviors and later homosexuality is not causal, is rejected as not applicable."

Gundlach, "Childhood Parental Relationships and the Establishment of Gender Roles of Homosexuals," 136.

- The second response came from none other than Evelyn Hooker: "Evans' study is therefore of special importance as a partial confirmation of the Bieber assumption about causal relation between parental relations in early childhood and adult homosexuality. In my view, Evans is overly cautious in his assertion that his findings neither confirm nor refute the etiological role of parent-child relations as one set of many variables influencing or causing homosexuality in adult life. Indeed, his study necessitates this generalization since it is a replication."

Hooker, "Parental Relations and Male Homosexuality in Patient and Nonpatient Samples," 140-41.

## *Hooker v. Hooker*

We find in Hooker's writings other admissions that seem at odds with her overall viewpoint.

- While admitting that while studies such as Evans were a “partial confirmation” of Bieber, she insisted they were “not conclusive.” Nonetheless, she acknowledged the validity of studies that do not utilize patients who are undergoing therapy: “Similar results with nonpatient samples tend to confirm ... the etiological role of parental relationships in producing homosexuality.”
- In fact, Hooker herself mentions several studies that control for the presence of unrelated psychopathology: “In the Schofield (1965) study, homosexual men in a nonpatient sample reported a higher incidence of poor relations with the father and of overprotective or overpossessive mothers than did a similar sample of heterosexual men.”

Hooker, “Parental Relations and Male Homosexuality in Patient and Nonpatient Samples,” 140.

## *Other confirmations of Bieber's findings*

- A 1969 study was published in *Psychological Reports* that was designed to address criticisms such as raised by Hooker. Authors John R. Snortum, et al., incorporated a non-patient control group: “In this study, then, the investigators, the methods, and (subjects) were drawn from outside of the circle of psychoanalytic study.” The authors concluded that “the present findings lend strong support to the earlier results obtained by Bieber, *et al.* (1962),” including the formative influence of “the pathological interplay between a close-binding, controlling mother and a rejecting and detached father.”

John R. Snortum, James F. Gillespie, John E. Marshall, John P. McLaughlin, and Ludwig Mosberg, “Family Dynamics and Homosexuality,” *Psychological Reports*, 24 (1969): 763.

- A 1974 study comparing 307 homosexuals with a control group of 138 heterosexuals, both from nonclinical samples, confirmed Bieber, et al.: “The homosexuals, in contrast to the heterosexuals, reported their fathers to be more rejecting and less loving. The homosexuals also described their mothers as more rejecting and less loving...the

homosexuals indicated less closeness to their fathers than the heterosexuals.”

Marvin Siegelman, “Parental Background of Male Homosexuals and Heterosexuals,” *Archives of Sexual Behavior*, 3 (1974): 10 .

- Others studies could be cited, as they were by Siegelman: “Support for the ‘triangular system’ hypothesis has been presented by Benda (1963), Braatan and Darling (1965), Brown (1963), Edwards (1963), Evans (1969), Jonas (1944), O’Connor (1964), Snortum et al. (1969), and Whitener and Nikelly (1964).”

Ibid., 3 (1): 3-4 (1974).

- Even three decades of research later, the *Archives of General Psychiatry* found that “the literature suggests that many, perhaps a majority, of homosexual men report family constellations similar to those suggested by Bieber et al. to be causally associated with the development of homosexuality (e.g., overly involved, anxiously overcontrolling mothers, poor father-son relationships). This association has been observed in nonclinical as well as clinical samples.”

Byne and Parsons, “Human Sexual Orientation: The Biologic Theories Reappraised,” 236.

### *Summary of findings regarding the developmental causes of homosexuality*

Daniel G. Brown put it well forty years ago—but his ringing call to action has gone unheeded for the past thirty years:

- “In summary, then, it would seem that the family pattern involving a combination of a dominating, overly intimate mother *plus* a detached, hostile or weak father is beyond doubt related to the development of male homosexuality. Beginning with the penetrating clinical insights of Freud 50 years ago, the systematic investigation by Terman and Miles some 30 years ago, the independent findings of a number of clinical and research workers, and the recent noteworthy contributions of West and Bieber, there is now strong evidence and considerable agreement as to family dynamics in the development of male homosexuality. It is surprising there has not been greater recognition of this relationship among the various disciplines that are concerned with children. A problem that arises in this connection is

how to inform and educate teachers and parents relative to the decisive influence of the family in determining the course and outcome of the child's psychosexual development. There would seem to be no justification for waiting another 25 or 50 years to bring this information to the attention of those who deal with children. And there is no excuse for professional workers in the behavioral sciences to continue avoiding their responsibility to disseminate this knowledge and understanding as widely as possible."

Daniel G. Brown, "Homosexuality and Family Dynamics," *Bulletin of the Menninger Clinic* 27 (5): 232 (Sept. 1963).

### *The "exotic becomes erotic" theory*

Another more recent theory regarding the development of same-sex attraction is offered by Daryl Bem of Cornell University.

- Bem's "Exotic Becomes Erotic" theory of erotic/romantic attraction held that "biological variables, such as genes, prenatal hormones, and brain neuroanatomy, do not code for sexual orientation per se but for childhood temperaments that influence a child's preferences for sex-typical or sex-atypical activities and peers. These preferences lead children to feel different from opposite or same-sex peers—to perceive them as dissimilar, unfamiliar, and exotic. This, in turn, produces heightened nonspecific autonomic arousal that subsequently gets eroticized to that same class of dissimilar peers: Exotic becomes erotic... The theory claims to accommodate both the empirical evidence of the biological essentialists and the cultural relativism of the social constructionists."
- Bem's theory suggests that a child's experience of being "different" from peers of the same gender leads to a reaction of physical arousal, which then later in life becomes translated into sexual arousal—a process he calls 'sexual imprinting.' Bem mentions the illustration of a "gender-nonconforming boy who is taunted by other boys. At first this produces strong negative arousal, but with repeated encounters over time, the fear and anger habituate and the opponent process becomes the conditioned, dominant affect. He thus emerges into late childhood or adolescence experiencing positive affective arousal to males, an arousal ready to be eroticized."

Bem, "Exotic Becomes Erotic: A Developmental Theory of Sexual Orientation," *Psychological Review*, 103 (1966): 327.

- In a similar vein, psychiatrists Byne and Parsons propose an “interactional model,” in which genes or hormones do not specify sexual orientation per se, but instead bias particular personality traits and thereby influence the manner in which an individual and his or her environment interacts as sexual orientation and other personality characteristics unfold developmentally. Such a mechanism would allow for multiple developmental pathways leading to homosexuality and would account for the high concordance rate for homosexuality among identical twins reared together, as well as for the failures of various psychosocial theories that have focused exclusively either on personality traits of individuals or on various environmental factors, but not on the interaction of the two.”

Byne and Parsons, “Human Sexual Orientation: The Biologic Theories Reappraised,” 236–37.

## Child Sexual Abuse and the Development of Homosexuality

A study in the *Journal of Sex & Marital Therapy* examined the past sexual experiences, sexual thoughts, and fantasies regarding the sexual contacts of 35 adult men who were sexually abused during their childhoods. The study found that among men, a history of homosexual child abuse was linked both to an adult homosexual orientation and to sexual attraction to children:

- “According to existing literature, gender identity confusion and gender preference are often cited as being affected by childhood sexual abuse. In this study, 46 percent of the abused men, as opposed to 12 percent of the nonabused men, defined their sexual orientation as either bisexual or homosexual. Therefore, these findings further validate previous research regarding the sexual orientation of children who have been sexually abused.”
- The study concludes: “Given these findings, it appears that being sexually abused as a child may affect the propensity of adult men to fantasize about young men.”

James R. Bramblett, Jr., and Carol Anderson Darling, “Sexual Contacts: Experiences, Thoughts, and Fantasies of Adult Male Survivors of Child Sexual Abuse,” *Journal of Sex & Marital Therapy*, 23 (4): 313 (Winter 1997).

- The *Bulletin of the Menninger Clinic* mentions “seduction” among a list of other possible childhood experiences that could contribute to

same-sex attraction: “There are a number of factors that occur in childhood which appear to be related to the development of homosexuality in adults. Such conditions as prolonged segregation of the sexes; specific, intensely exciting, and gratifying homosexual experiences in childhood; seduction by adult homosexuals; threatening and painful experiences in connection with sex play or relationships with the opposite sex; these and related factors in childhood and adolescence are correlated with the occurrence of homosexuality in adulthood.”

Brown, “Homosexuality and Family Dynamics,” 228.

## Cultural Factors in the Development of Homosexuality

If homosexuality were a result of biological or genetic factors, one might expect that it would be fairly evenly distributed both geographically and sociologically among all types of people. However, the research into Americans’ sexual behavior and self-identification by sexual orientation indicates that this is not the case. Two factors, in particular, stand out as having a strong correlation with a greater likelihood of engaging in homosexual acts or self-identifying as homosexual: urbanization and education.

### *Urbanization*

Homosexuals tend to be concentrated in urban areas:

- The National Health and Social Life Survey found that homosexuals and lesbians are not evenly distributed across the country. Rather, “more than 9 percent of the men in the nation’s twelve largest cities identify themselves as gay. But just 3 or 4 percent of men living in the suburbs of these cities or in most of the larger cities of the nation say they are gay, and about 1 percent of men in rural areas identify themselves as gay. Lesbians, too, cluster in cities, but the tendency is not so pronounced as for gay men.”

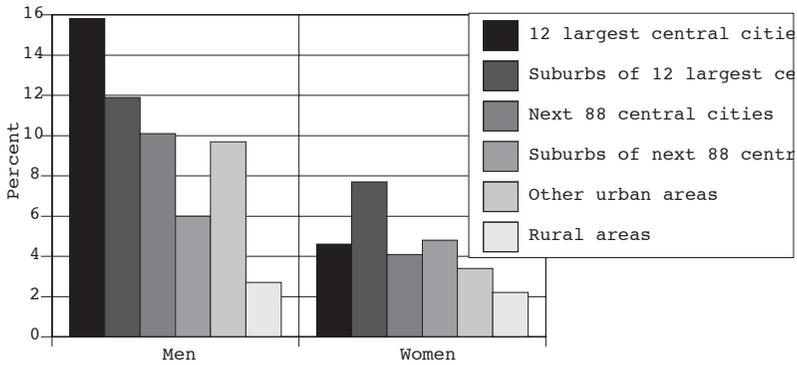
Robert T. Michael, John H. Gagnon, Edward O. Laumann, and Gina Kolata, *Sex in America: A Definitive Survey* (Boston: Little, Brown and Co., 1994), 177–79.

- *The Social Organization of Sexuality* gives additional details on the “striking” relationship between “the level of urbanization of the cur-

rent residence of respondents and the various measures of same-gender sexuality. Men living in the central cities of the twelve largest metropolitan areas reports rates of same-gender sexuality of between 9.2 and 16.7 percent (...referring to *identity* and *desire*, respectively), a compared to rates for all men on these measures of 2.8 and 7.7 percent, respectively. And the rates generally decline monotonically with decline in urbanization. While the rates of reported same-gender sexuality for women generally follow a similar pattern to those for men, that is, they are positively correlated with degree of urbanization, this pattern is not nearly so marked as with the men.”

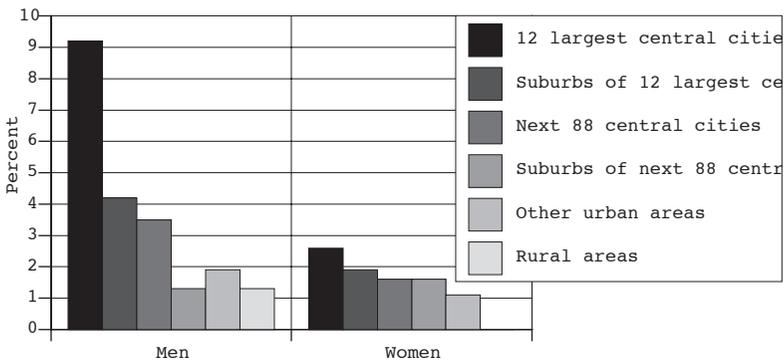
Laumann, et al., *The Social Organization of Sexuality*, 306.

Chart 1: Percentage with any same-gender



sexual contact since puberty, by place of residence

Chart 2: Percentage self-identifying as



homosexual or bisexual, by place of residence

Source: Edward O. Laumann, John H. Gagnon, Robert T. Michael, and Stuart Michaels, *The Social Organization of Sexuality: Sexual Practices in the United States* (Chicago: The University of Chicago Press, 1994), Table 8.2, 305.

- The *Demography* study elaborates on the high levels urbanization among homosexuals: “The 20 cities with large numbers of gay couples, which are home to less than 26 percent of the U.S. population, contain nearly 60 percent of our sample of gay men. Clearly gay men are concentrated in a selected number of urban areas. Lesbian women are somewhat less geographically concentrated....some cities have atypically high concentrations of gays and lesbians. For example, a randomly selected gay man in our sample is about 12 times more likely to live in San Francisco than are other individuals in the U.S. population. Other cities with especially high concentrations of gays include Los Angeles, Washington, DC, and Atlanta. High concentrations of lesbian women are found in San Francisco, Seattle, and Minneapolis. When we look at concentrations of gay and lesbian couples in smaller cities (e.g., 200,000 to 700,000), we find a disproportionate number of ‘college towns’ such as Ann Arbor and Madison. (For both gays and lesbians, seven of the 10 smaller cities with high concentrations contain a major university.)”

Black, et al., “Demographics of the Gay and Lesbian Population in the United States,” 148–49.

- Of course, one possible explanation for such a finding could be that homosexuals are more evenly spread across the country at birth, but tend to gravitate in adulthood toward larger cities where they can find greater acceptance and a substantial community of other homosexuals. However, Michael et al. question this interpretation, pointing out that “it is not just that homosexuals tend to move to large cities from the small towns and rural areas where they grew up.” Instead, they cite findings that “people who were raised in large cities were more likely to be homosexual than people who were raised in suburbs, towns, or the countryside. This relationship also showed up in the General Social Survey, an independent national sample.”

Robert T. Michael, John H. Gagnon, Edward O. Laumann, and Gina Kolata, *Sex in America: A Definitive Survey* (Boston: Little, Brown and Co., 1994), 182.

- Michael et al. directly contradict the notion that people are “born gay” with their hypothesis on why an urban upbringing is correlated with homosexuality: “It might be that it is easier for a person to be gay to learn to be gay or to explore a gay lifestyle growing up in a larger community that has other gays.”

Michael, et al., *Sex in America*, 182.

- A more detailed explanation of how a social environment that affirms a homosexual identity and behavior can increase the prevalence of homosexuality was described decades ago in the *British Journal of Psychiatry*: “If he frequents certain inns and other haunts where homosexuals foregather, he is encouraged to practice homosexuality; frequent indulgence may remove the anxiety about failing to mature and at the same time satisfy the sexual drive. The mutual support given by the rest of the coterie encourages the homosexual to believe he is one of a race apart without hope of cure and therefore entitled to indulge in his now firmly established homosexual habits.... If the outlook is hopeless and cure is impossible, the subject argues that he is entitled to indulge his sexual drive in the only way he can and that society must accept his homosexuality.”

P. J. O'Connor, “Aetiological Factors in Homosexuality as Seen in Royal Air Force Psychiatric Practice,” *British Journal of Psychiatry*, 466: 386 (May 1964).

## Education

As with urbanization, higher levels of education are directly correlated with higher levels of homosexual behavior and self-identification.

- A study in the journal *Science* found, “Men with four or more years of college are estimated to have a higher proportion” with same-gender sexual experience, “particularly compared to those with no college education.”

Robert E. Fay, Charles F. Turner, Albert D. Klassen, John H. Gagnon, “Prevalence and Patterns of Same-Gender Sexual Contact Among Men,” *Science* 243, Issue 4889 (20 January 1989): 342.

- Another study of men, published in *Family Planning Perspectives*, found that education was “positively associated with having had a same-gender sexual experience within the last ten years....”

John O. G. Billy, Koray Tanfer, William R. Grady and Daniel H. Klepinger, “The Sexual Behavior of Men In the United States,” *Family Planning Perspectives* 25, no. 2 (March/April 1993): 59.

- The comprehensive National Health and Social Life Survey (NHSL) reached the same conclusion: “Our study shows that twice as many college-educated men identify themselves as homosexual as men with high-school educations, 3 percent of college-educated men said they were gay compared to 1.5 percent of men with high-school educations.”

- However, the authors of *Sex in America* reported, “For women the trend is even more striking. Women with college educations are eight times more likely to identify themselves as lesbians as are women with a high-school education. Four percent of female college graduates identify themselves as lesbians as compared to less than half a percent of female high-school graduates.”

Michael et al., *Sex in America: A Definitive Survey*, 182.

Chart 1: Percentage with any same-gender sexual contact since puberty, by level of education

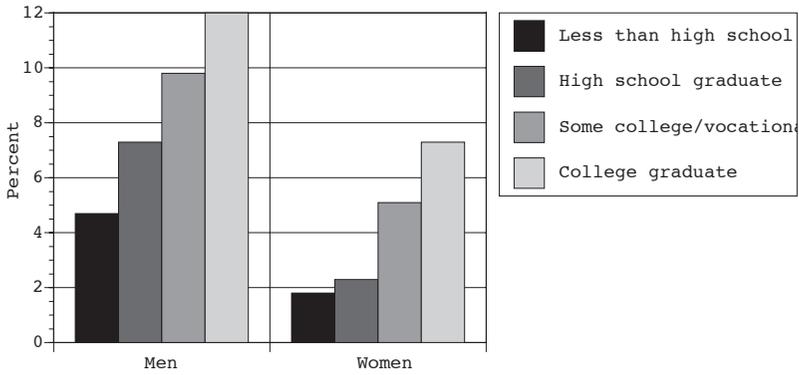
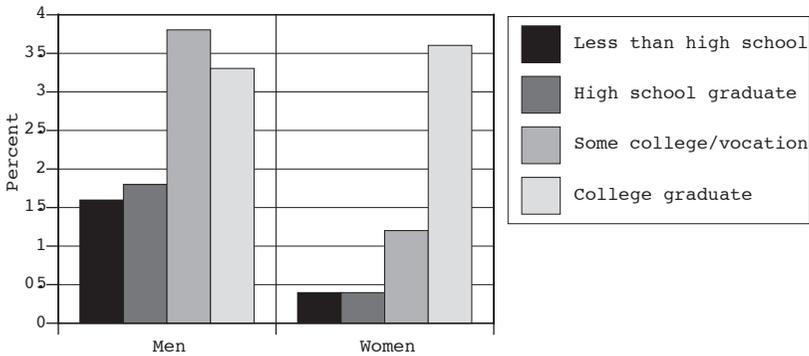


Chart 2: Percentage self-identifying as homosexual or bisexual, by level of education



Source: Edward O. Laumann, John H. Gagnon, Robert T. Michael, and Stuart Michaels, *The Social Organization of Sexuality: Sexual Practices in the United States* (Chicago: The University of Chicago Press, 1994), Table 8.2, 305

- Again, the interpretation offered in the most scholarly treatment of the NHSLs data, *The Social Organization of Sexuality*,<sup>1</sup> supports the notion that the ideological environment rather than any innate characteristic accounts for this finding: “Higher levels of education are associated with greater social and sexual liberalism” and with “greater sexual experimentation.” Laumann et al. conclude, “Acceptance of nontraditional sexual behavior is likely to be higher among the more educated.”

Edward O. Laumann, John H. Gagnon, Robert T. Michael, and Stuart Michaels, *The Social Organization of Sexuality: Sexual Practices in the United States* (Chicago: The University of Chicago Press, 1994), 309.

## The Role of Personal Choice in Becoming Homosexual

Debates over homosexuality are often presented in terms of a false dichotomy—either a person is “born gay,” or a person “chooses to be gay.” The truth lies between these two extremes. For the most part, people do not choose what sexual feelings or attractions they experience. Each of us does, however, choose the sexual behaviors in which we engage:

- Writing with reference to lesbians, Bem notes that “some women who would otherwise be predicted by the EBE model to have a heterosexual orientation might choose for social or political reasons to center their lives around other women. This could lead them to avoid seeking out men for sexual or romantic relationships, to develop affectional and erotic ties to other women, and to self-identify as lesbians or bisexuals.”

Bem, “Exotic Becomes Erotic: A Developmental Theory of Sexual Orientation,” 331.

- Writing in *Archives of General Psychiatry* Byne and Parsons note the role that “choice” plays in the development of one’s “sexual orientation”: “Conspicuously absent from most theorizing on the origins of sexual orientation is an active role of the individual in constructing his or her identity.” The authors explain: “This is not meant to imply that one consciously decides one’s sexual orientation. Instead, sexual orientation is assumed to be shaped and reshaped by a cascade of choices made in the context of changing circumstances in one’s life and enormous social and cultural pressures.”

Byne and Parsons, “Human Sexual Orientation: The Biologic Theories Reappraised,” 236–237.