Biology and sexual orientation

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Biology and sexual orientation is the proposition that there may be, at least in part, a biological basis for sexual orientation. An increasing number of studies have investigated this link, but no scientific consensus exists as to the specific biological factors that may play a role, nor to the precise nature of their influence on sexual orientation. Causal areas these studies have examined include morphological brain structure, prenatal environment, chromosomes, and viral genetic influence. Methodologically, some studies have used twins as controls.

The main biological determinants of sexual orientation are generally thought to be genetic and hormonal, with some writers suggesting either one or the other are the key factor, and many suggesting that both play a role. Most scientists agree that it is unlikely that there is a single "gay gene" that determines something as complex as sexual orientation, and that it is more likely to be the result of a number of biological factors. Many also agree that social and environmental factors intersect with biology to produce sexual orientation, while many strongly believe that sexual orientation is purely biological and inborn. The view that post-natal environmental influence is the sole determinant of sexual orientation and gender is increasingly rare among researchers, as the Putlizer prize-winning science author Matt Ridley recently summed it up "Nobody in science now believes that sexual orientation is caused by events in adolescence".[1] Evidence suggests that this view is also increasingly common among the educated general public.[2] On the other hand, others suggest that these views are the product of particular scientific foundations promoting social agendas. The American Psychiatric Association has only recognized homosexuality as anything other than an emotional dysfunction since 1973.

Sexual orientation and evolution

A central problem of the genetics of sexual orientation is the question of adaptiveness. In evolutionary terms, an "adaptive" trait is one that increases the chances for reproduction of the genes that code for that trait in the organism. Non-heterosexual orientations significantly decrease the chances of successful reproduction (they are "anti-adaptive") and so should be selected out of a population over long periods of time. Many anti-adaptive genetic traits exist in the current human population in significant numbers, including a variety of genetic diseases (some fatal.) So settling the question of whether or not non-heterosexual orientations are adaptive, anti-adaptive, or neutral, does not necessarily settle the question of whether or not they have genetic determinants, and vice versa.[[]

Evidence is increasing that homosexuality is a result of a complex interaction of many genes which are spread throughout human populations.[3] It has been suggested that male homosexuality represents an excess of genes coding for female-like traits which tip gay males over a 'liability threshold' into homosexuality. Females selecting 'gay-enabled' men may have increased the viability of their offspring.[4]

Some have suggested that homosexuality is adaptive in a non-obvious way. By way of analogy, the allele (a particular version of a gene) which causes sickle-cell anemia when two copies are present may also confer resistance to malaria with no anemia when one copy is present.

The so-called "gay uncle" theory posits that people who do not themselves have children may nonetheless increase the prevalence of their genes in future generations by providing resources (food, supervision, defense, shelter, etc.) to the offspring of close relatives. The primary criticism of this theory has to do with the fact that children share on average 25% of their genes with their uncles and aunts, but on average 50% with their parents. This means that to be adaptive, a "gay uncle" would need to save from death (or other lineage-terminating events) on average two nieces or nephews for every one of their own offspring they give up. Critics of the theory find this trade-off to be unlikely to produce a net reproductive gain.

Another theory in support of positive adaptiveness of homosexuality and bisexuality is that these sexual orientations provide some benefit to the local community in general. Communities that are so

supported would be stronger and more likely to survive in the future. [[]*citation needed*[]] The primary criticism of this theory is that genetic evolution works on the level of individuals, not communities. If heterosexuals have an individual reproductive advantage over homosexuals and bisexuals in a given community, after a very long period of time, the community should be composed solely of heterosexuals, regardless of how this affects the community as a whole.

Finally, some posit that homosexuality would indeed be purely anti-adaptive, but like poor eyesight, still exists in the population because it has not, in the past, greatly affected an individual's chances of reproducing. Homosexual individuals in societies with no understanding of homosexuality and without a stigma placed on homosexuality have been observed to marry someone of the opposite sex and have children just like heterosexuals. The phenotype of the alleged "homosexual" genes is different and may actually be adaptive in such an environment.¹

Politics

The issue of genetic or other physiological determinants as the basis of sexual orientation is a highly politicised issue. *The Advocate*, a U.S. gay and lesbian newsmagazine, reported in 1996 that 61% of its readers believed that "it would mostly help gay and lesbian rights if homosexuality were found to be biologically determined".[5] A cross-national study in the United States, the Philippines, and Sweden found that those who believed that "homosexuals are born that way" held significantly more positive attitudes toward homosexuality than those who believed that "homosexuals choose to be that way" and/or "learn to be that way".[6] If sexual orientation is seen to be innate, it is a potential argument against attempts to "cure" homosexuality, as well efforts to censor positive representations of lesbians, gays and bisexuals in order to "protect" children or young people from adopting a homosexual orientation. In addition, it supports a minority rights conception of gays, lesbians and bisexuals in law and government policy.

Many objections to the idea of an innate cause of homosexuality come from religious groups and conservatives who focus on a moral rather than medical interpretation of sexual orientation, and seeing an individual's sexuality as a matter of personal choice or upbringing. However, seeing homosexuality as a "medical" rather than a "moral" issue does not guarantee better treatment for homosexuals. Indeed, many medical explanations of the innateness of sexual orientation have pathologised homosexuality and bisexuality as a kind of disability, and if a "gay gene" was discovered, parents might choose to screen and terminate embryos that carry the gene.

Empirical studies

Twin Studies

One common type of twin study compares the monozygotic (or *identical*) twins of people possessing a particular trait to the dizygotic (non-identical, or *fraternal*) twins of people possessing the trait. Since monozygotic twins have the same genotype (genetic makeup) while dizygotic twins share only, on average, 50% of their genotype, a difference in the prevalence of the trait in question between these types of twins provides evidence of a genetic component.

A few such studies began to examine homosexuality in the early 20th century, using small, nonrandom samples. The first relatively large-scale twin study on sexual orientation was reported by Kallman in 1952. Examining only male twin pairs, he found a 100% concordance rate for homosexuality among 37 monozygotic (MZ) twin pairs, compared to a 12%-42% concordance rate among 26 dizygotic (DZ) twin pairs, depending on definition. In other words, every identical twin of a homosexual subject was also homosexual, while this was not the case for non-identical twins. This study was criticised for its vaguely-described method of recruiting twins and for a high rate of psychiatric disorders among its subjects.

While Kallman's was the largest relevant study until the 1990s, other studies did provide examples of MZ twin pairs of both sexes who were discordant for sexual orientation; that is, they found that both male and female homosexuals did sometimes have twins that were not homosexual themselves. The

existence of such twins demonstrates that genes are not the *only* factor involved in determining sexual orientation, at least not for everybody. Environmental factors, in the womb or during life, must play a role.

However, studies such as Kallman's did suggest the existence of a significant genetic component to sexual orientation, and later researchers have attempted to use twin studies to quantify the size of such an effect. The most highly publicized was reported by J. Michael Bailey and Richard Pillard in "A Genetic Study of Male Sexual Orientation" in the *Archives of General Psychiatry*, December 1991. These researchers recruited gay male subjects through advertisements in the gay media and sent questionnaires, which included questions on sexual orientation, to their male siblings. They reported that:

52% (29/56) of MZ twins,

22% (12/54) of DZ twins,

9.2% (13/142) of non-twin brothers, and

11% (6/57) of adoptive brothers of gay men

were also gay themselves. The researchers estimated that the heritability of male homosexuality was between 31% and 74%. A similar study carried out by the same researchers on the siblings of lesbian women reported concordance rates of:

48% for MZ twins,16% for DZ twins,14% for non-twin sisters, and6% for adoptive sisters.

Heritability was estimated as between 27% and 76%.

Being based on samples of people who volunteered for studies specifically targeting gay men and lesbians, these results could only be suggestive. However, a number of more recent studies have examined sexual orientation in large "twin registries" recruited without reference to sexual orientation. Estimates of heritability for male and female homosexuality derived from these are shown below.

Study	Male	Female
Hershberger, 1997	0%	48%
Bailey <i>et al.</i> , 2000	40%	0%
Kendler e <i>t al.</i> , 2000	28–65%	
Kirk e <i>t al.</i> , 2000	30%	50–60%

Estimates of heritability of homosexuality

Conclusions

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One criticism often levelled at comparisons of MZ/DZ twins in general is that they depend upon an assumption that MZ and DZ twins share a similar amount of their environments with their twins. This assumption has been guestioned.

To consider specifically the studies under discussion here, the results of the Bailey and Pillard volunteer studies pose some problems for interpretation. Consider the male study. The higher concordance rate among MZ twins compared to DZ twins is consistent with substantial heritability. However, the lower rate among non-twin brothers compared to DZ twins (both share on average 50% of genes), and the high rate of homosexuality among adoptive brothers of gay men compared to the general population (when adoptive brothers are no more genetically similar than men from different families) provide evidence for environmental factors. It is also worth stressing that the MZ concordance was well short of 100%, which also points to a role for environmental factors.

It is considered likely by some authors, including Bailey, that the heritability of homosexuality has been overstated by volunteer studies. For example, a gay man with a gay brother may be more likely to volunteer for a study of gay men and their brothers than will a gay man with a heterosexual brother, perhaps because he feels that his brother will be more willing to cooperate. The prevalence of

stigmatization and discord within families occasioned by the "coming out" of a gay member might also reduce the likelihood of a heterosexual brother being willing to participate in the study. The lower heritability estimates from the more recent, probably more representative, studies seem to confirm this.

However, there is still considerable variation even between these studies. It is interesting to note that the Kirk *et al.* study (see table above) was a reanalysis of the same data used by Bailey *et al.* (same table), using different definitions of homosexuality. The striking difference in results, particularly for women, underlines the lack of definitive results at this point. One problem is that, for most definitions, the prevalence of homosexuality in the general population is low, which means that registries will contain relatively few twin pairs of which one is gay or lesbian. The lack of statistical power resulting from this may explain some of this lack of consistency. Meta-analysis might be of use in resolving this difficulty.

Overall, data appear to indicate that genetic factors play a significant part in the development of sexual orientation, accounting for about half of the variability. However, further work is needed to more precisely quantify the genetic contribution.

Any genetic component must be rooted in evolution by natural selection, and many non-scientists assume that a homosexual orientation would necessarily result in decreased reproduction. Gene prevalence and therefore selection, however, can be influenced by increasing the reproductive success of individuals with whom we share genes in common. While it may be unclear to some how homosexuality could offer a selective advantage to individuals, many hypotheses exist that explain why an inherited tendency toward this orientation might offer a selective advantage to the genes they carry. Most hypotheses speculate that the presence of homosexual members may also promote intragroup harmony. These are hypotheses: hard empirical data is lacking. Apparent homosexual behavior provides a stealth mechanism for slipping past alpha males in some species; like most putative explanations, that does not explain inheritance of female homosexuality. Some twin effects could be the result of their shared environment from conception to birth.

Studies of brain structure

A number of sections of the brain have been reported to be sexually dimorphic; that is, they vary between men and women. There have also been reports of variations in brain structure corresponding to sexual orientation. In 1990, Swaab and Hofman reported a difference in the size of the suprachiasmatic nucleus between homosexual and heterosexual men. In 1992, Allen and Gorski reported a difference related to sexual orientation in the size of the anterior commissure.

However, the best-known work of this type is that of Simon LeVay, reported in "A Difference in Hypothalamic Structure Between Hetero-sexual and Homosexual Men" in the journal *Science*, August 1991. LeVay studied four groups of neurons in the hypothalamus, called INAH1, INAH2, INAH3 and INAH4. This was a relevant area of the brain to study, because of evidence that this part of the brain played a role in the regulation of sexual behaviour in animals, and because INAH2 and INAH3 had previously been reported to differ in size between men and women.

He obtained brains from 41 deceased hospital patients. The subjects were classified as follows: 19 gay men who had died of AIDS, 16 presumed heterosexual men (6 of whom had died of AIDS), and 6 presumed heterosexual women (1 of whom had died of AIDS).

The AIDS patients in the heterosexual groups were all identified from medical records as intravenous drug abusers or recipients of blood transfusions, though only 2 of the men in this category had specifically denied homosexual activity. The records of the remaining heterosexual subjects contained no information about their sexual orientation; they were assumed to have been mostly or all heterosexual "on the basis of the numerical preponderance of heterosexual men in the population".

LeVay found no evidence for a difference between the groups in the size of INAH1, INAH2 or INAH4. However, the INAH3 group appeared to be twice as big in the heterosexual male group as in the gay male group; the difference was highly significant, and remained significant when only the 6 AIDS patients were included in the heterosexual group. The size of the INAH3 in the homosexual male

brains was similar to that in the heterosexual female brains. However, he also found some contrary results:

- Three of the 19 homosexual subjects had a larger group of neurons in the hypothalamus than the average control-group subject.
- Three of the 16 control-group subjects had a smaller group of neurons in the hypothalamus than the average homosexual subject.

William Byne and colleagues attempted to replicate the differences reported in INAH 1-4 size using a different sample of brains from 14 HIV-positive homosexual males, 34 presumed heterosexual males (10 HIV-positive), and 34 presumed heterosexual females (9 HIV-positive). They found a significant difference in INAH3 size between heterosexual men and women. The INAH3 size of the homosexual men was apparently smaller than that of the heterosexual men and larger than that of the heterosexual women, though neither difference quite reached statistical significance.

Byne and colleagues also weighed and counted numbers of neurons in INAH3, tests not carried out by LeVay. The results for INAH3 weight were similar to those for INAH3 size; that is, the INAH3 weight for the heterosexual male brains was significantly larger than for the heterosexual female brains, while the results for the gay male group were between those of the other two groups but not quite significantly different from either. The neuron count also found a male-female difference in INAH3, but found no trend related to sexual orientation.

Conclusions

LeVay concluded in his 1991 paper that "The discovery that the nucleus differs in size between heterosexual and homosexual men illustrates that sexual orientation in humans is amenable to study at the biological level, and this discovery opens the door to studies of neurotransmitters or receptors that might be involved in regulating this aspect of personality. Further interpretation of the results of this study must be considered speculative. In particular, the 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 covary under the influence of some third, unidentified variable." S. LeVay, "A Difference in Hypothalmic Structure Between Heterosexual and Homosexual Men" 253 Science 1034, 1036 (1991) [1] He later added, "It's important to stress what I didn't find. I did not prove that homosexuality is genetic, or find a genetic cause for being gay. I didn't show that gay men are born that way, the most common mistake people make in interpreting my work. Nor did I locate a gay center in the brain. ... Since I look at adult brains, we don't know if the differences I found were there at birth or if they appeared later." [2] In addition to this, of the men LeVay used in his studies, the sexual histories of the "heterosexual" men were unknown.

It is not clear from the research how HIV/AIDS may affect brain structure, a possible confounding factor. So rather than showing that differences in the neuron indicate homosexuality, LeVay's study may be showing that HIV/AIDS causes differences in neurons. It should be noted, however, that neither LeVay nor Byne found an HIV-related difference in INAH3 size.

The man considered the "dean of American sexologists", Johns Hopkins University psychologist John Money, concerning LeVay's studies, says, "Of course it [sexual orientation] is in the brain. The real question is, when did it get there? Was it prenatal, neonatal, during childhood, puberty? That we do not know." [[]*citation needed*[]]

Simon LeVay's finding points out a correlation between physiology and sexual orientation, but does not necessarily establish *by itself* a genetic basis for sexual orientation.

Chromosome linkage studies

In 1993, Dean Hamer published findings from a linkage analysis of a sample of 76 gay brothers and their families. Hamer *et al* (1993)[7] found that the gay men had more gay male uncles and cousins on the maternal side of the family than on the paternal side. Further analysis by Hu et al[8] revealed that 67% of gay brothers in a new saturated sample shared a marker on the X chromosome at Xq28.

Sanders et al (1998 cited in Wilson and Rahman 2005, p52)[9] replicated the study, finding 66% Xq28 marker sharing in 54 pairs of gay brothers. However, three teams (Bailey et al 1999, McKnight and Malcolm 2000, and Rice et al, 1999 cited in Vilain, 2000)[10] failed to replicate the finding. More recently, Mustanski (2005)[11] failed to find the Xq28 marker in a complete genome scan of gay men's DNA. Mustanski did however find autosomal markers at 7q36, 8p12 and 10q26. The evidence for the Xq28 marker has therefore become muddled by failures of replication.

A recent study supports X-linkage from a different perspective. Women have two X chromosomes, one of which is "switched off". In some cases, it appears that this switching off can occur in a non-random fashion. Bocklandt et al (2006)[12] reported that the number of women with extreme skewing of X chromosome inactivation is significantly higher in mothers of homosexual men than in age-matched controls without gay sons. 4% of controls showed extreme skewing compared to 13% of the mothers with gays sons and 23% of mothers with two or more gay sons.

Male homosexuality appears likely to be influenced by a complex genetic interaction which may be mediated by H-Y antigens in the mother's immune system (see below). Whichever genes are implicated they almost certainly cause male brains to differentiate in a female typical direction. As for female homosexuality, there remains little evidence from replicated genetic linkage studies.

Correlations with matriarchal lineage, birth order, and female fertility

A 1993 U.S. study found a correlation between male homosexuality and maternal lineage, suggesting that mothers have some special role in determining the sexual orientation of their male offspring. Studies since the 1960s (eg Slater, 1962) have noted that homosexual men tend to be the later-born among a group of siblings. Many recent studies (see Fraternal birth order) have indicated that homosexual men are more likely to have older brothers than the general male population. This difference is not observed among women.

A chemical called the histocompatibility Y-antigen (the "HY antigen") is found on the surface of the cells of male mammals. It is hypothesized (Wachtel, 1983; Blanchard & Bogaert, 1996) that the "fraternal birth order effect" may be related to increasing levels of antibodies produced by the mother in response to the presence of this chemical during pregnancy with the oldest son. These antibodies could then somehow trigger different brain development patterns in later male children, either in the uterus or in early childhood through breast milk. Later sons would then more likely to have a homosexual orientation as adults. At least one genetic study attempting to verify this theory claimed to find a correlation with a certain area of the X chromosome (of which all women carry two copies, and all men carry one), but these findings could not be replicated by other researchers. [[]*citation needed*[]]

An alternate theory was proposed by Italian researchers in 2004 (Camperio-Ciani et al. 2004), supported by a study of about 4,600 people who were the relatives of 98 homosexual and 100 heterosexual men. Female relatives of the homosexual men tended to have more offspring than those of the heterosexual men. Female relatives of the homosexual men on their mother's side tended to have more offspring than those on the father's side. The researchers concluded that there was genetic material being passed down on the X chromosome which both promotes fertility in the mother and homosexuality in her male offspring. The connections discovered, however, would explain only 20% of the cases studied, indicating that this might not be the sole genetic factor determining sexual orientation.

Sources

- Blanchard, R. & Bogaert, A.F. (1996) Homosexuality in men and number of older brothers. *Am. J. Psychiat.* **153**: 27--31
- Camperio-Ciani A., Corna F. & Capiluppi C. (2004). Evidence for maternally inherited factors favouring male homosexuality and promoting female fecundity. *Proc. R. Soc. Lond. B.* 271:2217-21.

Slater, E. (1962) Birth order and maternal age of homosexuals. *Lancet* i, 69–71

Wachtel, S. S. (1983) H–Y Antigen and the Biology of Sex Determination. Grune & Stratton, New

York. How homosexuality is 'inherited'. BBC News, 13 Oct 2004. [3]

Testosterone correlation

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Homosexuality has also been correlated with elevated testosterone levels in adult males, which may indicate an indirect genetic influence.

Studies involving mice have shown differences in female sexual behaviour depending on distance from testosterone sources in the womb (Ryan & Vandenbergh, 2002). Female mice flooded with higher levels of testosterone in the womb are prone to more masculine sexual behaviour, such as mounting other females, whereas their sisters exposed to lower levels of foetal testosterone act in more traditionally feminine ways, and are typically courted more by male mice (vom Saal, 1989; vom Saal & Bronson, 1980; Rines & vom Saal, 1984). This suggests a pivotal role of hormones, and specifically testosterone, in the development of sexual orientation.

Pheromones correlation

Recent research conducted in Sweden [4] has suggested that gay and straight men respond differently to two odors that are believed to be involved in sexual arousal. The research showed that when both heterosexual women (lesbians were included in the study, but the results regarding them were "somewhat confused") and gay men are exposed to a testosterone derivative found in men's sweat, a region in the hypothalamus is activated. Heterosexual men, on the other hand, have a similar response to an estrogen-like compound found in women's urine. The study was taken as evidence in support of the theory that certain chemicals act as pheromones in humans. The conclusion, that sexual attraction, whether same-sex or opposite-sex oriented, operates similarly on a biological level, does not mean that there is necessarily a biological cause for homosexuality. Researchers have suggested that this possibility could be further explored by studying young subjects to see if similar responses in the hypothalamus are found and then correlating this data with adult sexual orientation.

Early fixation hypothesis

If sexual orientation is fixed early in life, when exactly does that happen, and how? The following "environmental" mechanisms (as opposed to (genetic mechanisms) have been proposed.

Fetal development

See also: Prenatal hormones and sexual orientation

Some have theorized that events in the womb may contribute to some subset of homosexual behavior (though certain individuals may be genetically predisposed to be vulnerable to such events, and the conditions inside the mother's reproductive system are of course influenced by her genetics).

Masculinization/feminization

A popular hypothesis in this vein is that the developing brains of homosexual men are less masculinized than heterosexual men (i.e. they are partially "feminized") and that homosexual females are "masculinized" in some way.

Supporting evidence for this hypothesis includes:

Observed differences in three areas of the brain in homosexual vs. heterosexual men (the anterior commisure, the supra-chiasmatic nucleus the interstitial nuclei of the anterior hypothalamus). [citation needed]

Observed differences in cognitive testing showing results for homosexual men typical of

heterosexual women and results for homosexual women typical of heterosexual men. [[]*citation needed*[]]

Observed differences in the preferences that homosexual men, heterosexual men, and heterosexual women have for the age of their sexual partners. ^[citation needed]

It is unclear whether the observed anatomical and cognitive differences are signs of a (possibly genetic) mechanism that determines sexual orientation, or symptoms of the formation of an atypical sexual orientation during childhood.

One possible mechanism is differential fetal hormone exposure, especially to testosterone (and a compound it is transformed into, estradiol) and luteinizing hormone (LH) is proposed as the mechanism. The concentrations of these chemicals is thought to be influenced by fetal and maternal immune systems, maternal consumption of certain drugs, maternal stress, and direct injection.

Hormone levels may of course vary over time. Given the semi-sequential nature of fetal development, and because multiple hormones are involved, it is possible for the hypothesized "masculinization" or "feminization" process to affect only some body or brain systems. (This is necessary to explain why someone might be say, born with a male body but with a "feminized" sexual attraction.)

Prenatal hormones have been indicated both in Simon LeVay's study of the anterior hypothalamus in cadavers with homosexual contraction of AIDS as cause of death and Marc Breedlove's study of birth order and ring-finger length ratios in living individuals. [[]*citation needed*[]] LeVay's study suggests that homosexual men are "feminized", Breedlove's study suggests that both heterosexual men and homosexual women are "masculinized". [[]*citation needed*[]] Breedlove's study also suggests that homosexual men are "super-males", as their ring-finger lengths were the longest among the four sexual orientations studied.

Pre-natal hormones in females

(This summary has not yet been checked against the original scientific journal article, which is cited below.)

Researchers at the University of Texas at Austin, lead by Dennis McFadden, found the response of the inner ear to soft sounds tended to be weaker in homosexual women than in heterosexual women. The response among men tended to be weaker than either female group. Fetal exposure to androgens is hypothesized to affect this attribute, suggesting that fetal exposure to the same chemicals may also predispose a daughter to a lesbian orientation. However, lifestyle differences between the two female groups may lead to different sound exposures, and this could also explain the correlation.

References

D. McFadden and E. G. Pasanen. Comparison of the auditory systems of heterosexuals and homosexuals: Click-evoked otoacoustic emissions. PNAS, March 3, 1998; 95(5): 2709 - 2713.
[7]

Pre-natal hormones in males

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Researchers in the Breedlove study found evidence correlating prenatal hormones to male homosexuality. Males exposed to high levels of androgens (sexual hormones) as fetuses are predominantly homosexual.

Correlations with matriarchal lineage, birth order, and female fertility

Certain observations involving "environmental" factors in the womb have been proposed to explain some instances of homosexual orientation; see Genetics and sexual orientation, prenatal hormones

and sexual orientation and fraternal birth order for explanations.

Pathogenic theory

Main article: Pathogenic theory of homosexuality

Based on the relatively high frequency of homosexuality in the population, and the assumption that it is evolutionarily very disadvantageous, some have proposed that the cause may be a bacterium or virus.

Imprinting/Critical period

This type of theory holds that the formation of gender identity occurs in the first few years of life after birth. It argues that individuals can be predisposed to homosexual orientation by biological factors but are triggered in some cases by upbringing. Part of adopting a gender identity involves establishing the gender(s) of sexual attraction. This process is analogous to the "imprinting" process observed in animals. A baby duckling may be genetically programmed to "imprint" on a mother, but what entity it actually imprints upon depends on what objects it sees immediately after hatching. Most importantly, once this process has occurred, it cannot be reversed, any more than the duckling can hatch twice.

A sort of reverse sexual imprinting has been observed in heterosexual humans; see the section on the "Westermarck effect" in Behavioral imprinting.

Several different triggers for imprinting upon a particular sexual orientation have been proposed.

A common hypothesis, especially among non-scientists, is that something about what young children see in the gender-roles behavior of adults, or some differences (possibly unconscious) in the way adults treat young children, somehow influence or determine a child's eventual sexual orientation.

This hypothesis, however, has not been supported by research findings that children of homosexuals are just as likely to be heterosexual as the general population and in reverse for children of heterosexuals in prevalence of homosexuality.

Exotic becomes erotic

Daryl Bem, a social psychologist at Cornell University, has theorized that the influence of biological factors on sexual orientation may be mediated by experiences in childhood. A child's temperament predisposes the child to prefer certain activities over others. Because of their temperament, which is influenced by biological variables such as genetic factors, some children will be attracted to activities that are commonly enjoyed by other children of the same gender. Others will prefer activities that are typical of the other gender. This will make a gender-conforming child feel different from opposite-gender children, while gender-nonconforming children will feel different from children of their own gender. According to Bem, this feeling of difference will evoke physiological arousal when the child is near members of the gender which it considers as being 'different'. Bem theorizes that this physiological arousal will later be transformed into sexual arousal: children will become sexually attracted to the gender which they see as different ("exotic"). This theory is known as *Exotic Becomes Erotic* (EBE) theory.

The theory is based in part on the frequent finding that a majority of gay men and lesbians report being gender-nonconforming during their childhood years. A meta-analysis of 48 studies showed childhood gender nonconformity to be the strongest predictor of a homosexual orientation for both men and women (Bailey and Zucker, 1995, Developmental Psychology). For example, in a study by the Kinsey Institute of approximately 1000 gay men and lesbians (and a control group of 500 heterosexual men and women), 63% of both gay men and lesbians reported that they were gender nonconforming in childhood (i.e., did not like activities typical of their sex), compared with only 10-15% of heterosexual men and women. There are also six "prospective" studies--that is longitudinal studies that begin with gender-nonconforming boys at about age 7 and follow them up into adolescence and adulthood. These also show that a majority (63%) of the gender nonconforming boys become gay or bisexual as adults (reported by Zucker, 1990). There are no prospective studies of gender nonconforming girls.

References and external links

- ^ Matt Ridley (2003) Nature Via Nurture: Genes, Experience, and What Makes us Human
- A Biological explanations of homosexuality have become more popular with the public over the past several years: In 1983, 16% of Americans believed that "homosexuality is something that people are born with"; by 1993, that figure had nearly doubled to 31% (Moore, D. W. (1993, April). Public polarized on gay issue. The Gallup Poll Monthly, 30-34).
- ^ Mustanski, B. S., Dupree, M. G., Nievergelt, C. M., Bocklandt, S., Schork, N. J., & Hamer, D. H. (2005). A genomewide scan of male sexual orientation. Human Genetics, 116, 272-278
- ^A Wilson, G. and Rahman, Q., (2005). Born Gay. London: Peter Owen Publishers. p64.
- [^] The Advocate (1996, February 6). Advocate Poll Results. p. 8.
- * Ernulf, K. E., Innala, S. M., & Whitam, F. L. (1989). Biological explanation, psychological explanation, and tolerance of homosexuals: A cross-national analysis of beliefs and attitudes. Psychological Reports, 65, 1003-1010. See also: Whitley, B. E., Jr. (1990). The relationship of heterosexuals' attributions for the causes of homosexuality to attitudes toward lesbians and gay men. Personality and Social Psychology Bulletin, 16, 369-377.
- A Hamer, Hu, Magnuson, Hu and Pattatucci (1993) A linkage between DNA markers on the X chromosome and male sexual orientation. Science 261(5119): 321-7
- ^A Hu, S., Pattatucci, A. M. L., Patterson, C., Li, L., Fulker, D. W., Cherny, S. S., Kruglyak, L., & Hamer, D. H. (1995). Linkage between sexual orientation and chromosome Xq28 in males but not in females. Nature Genetics, 11, 248–256.
- [^] Wilson, G. and Rahman, Q., (2005). Born Gay. London: Peter Owen Publishers
- [^] Vilain, E. (2000). Genetics of Sexual Development. Annual Review of Sex Research, 11.
- ^ Mustanski, B. S., Dupree, M. G., Nievergelt, C. M., Bocklandt, S., Schork, N. J., & Hamer, D. H. (2005). A genomewide scan of male sexual orientation. *Human Genetics*, 116, 272-278 PDF accessed 2 September 2006.
- A Bocklandt, S.B., Horvath, S., Vilain, E., Hamer, D.H. (2006). Extreme skewing of X chromosome inactivation in mothers of homosexual men. Human Genetics, 118:691-694
- BBC (April 23, 1999). Doubt cast on 'gay gene'. BBC News.
- William Byne (May 1994). The Biological Evidence Challenged. *Scientific American*, vol. 270, pp. 50-55.
- William Byne *et al.* (2001). The Interstitial Nuclei of the Human Anterior Hypothalamus: An Investigation of Variation with Sex, Sexual Orientation, and HIV Status. *Hormones and Behavior*, vol. 40, pp. 86-92.
- Simon LeVay (1991). A Difference in Hypothalmic Structure Between Heterosexual and Homosexual Men. *Science*, vol. 253, pp. 1034-1037.
- Simon LeVay & Dean H. Hamer (May 1994). Evidence for a Biological Influence in Male Homosexuality. *Scientific American*, vol. 270, pp. 44-49.
- Simon LeVay (updated at intervals) | The Biology of Sexual Orientation, a literature review web page.
- Trisha Macnair (undated). Genetics and human behaviour. BBC Health.
- Timothy F. Murphy (Fall 2000). Now What? The Latest Theory of Homosexuality. APA Newsletter on Philosophy and Lesbian, Gay, Bisexual and Transgender Issues.
- Nuffield Council on Bioethics (2002). *Genetics and human behaviour*. London: Author. Chapter 10 discusses sexual orientation.
- T. J. Taylor (1992).

Twin Studies of Homosexuality. Part II Experimental Psychology Dissertation (unpublished), University of Cambridge, UK.

Rosemary C. Veniegas & Terri D. Conley (2000). Biological Research on Women's Sexual Orientations: Evaluating the Scientific Evidence. *Journal of Social Issues*, vol. 56, pp. 267-282.

Nicholas Wade Gay Men Are Found to Have Different Scent of Attraction *The New York Times*, May 9, 2005.

Articles by Dr. Daryl Bem will be found at [8], including several on his EBE theory

Rahman Q. (2005). The neurodevelopment of human sexual orientation. *Neuroscience and Biobehavioral Reviews* **29** :1057–1066.

- Rines, J.P. & vom Saal, F.S. (1984). Fetal effects on sexual behavior and aggression in young and old female mice treated with estrogen and testosterone. *Horm. Behav.* **18**:117--12.
- Ryan, B.C. & Vandenbergh, J.G. (2002) Intrauterine position effects. *Neurosci. Biobehav. Rev.* **26**:665--678.
- vom Saal, F.S. (1989) Sexual differentiation in litter bearing animals: influence of sex of adjacent fetuses in utero. *J. Anim. Sci.* **67**:1824--1840.

Vom Saal, F.s. & Bronson, F. (1980). Sexual characteristics of adult female mice are correlated with their blood testosterone levels during prenatal development. *Science* **208**:597--599.

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