

Controversies in Sexual Medicine

Male Homosexuality: Nature or Culture?

Emmanuele A. Jannini, MD,* Ray Blanchard, PhD,[†] Andrea Camperio-Ciani, PhD,[‡] and John Bancroft, MD[§]

*Course of Endocrinology and Medical Sexology, Department of Experimental Medicine, University of L'Aquila, L'Aquila, Italy; [†]Law and Mental Health Program, Centre for Addiction and Mental Health, and Department of Psychiatry, University of Toronto, Toronto, ON, Canada; [‡]Department of General Psychology, University of Padua, Padua, Italy; [§]The Kinsey Institute for Research in Sex, Gender and Reproduction, Bloomington, IN, USA

DOI: 10.1111/j.1743-6109.2010.02024.x

ABSTRACT

Introduction. Debate continues on whether or not male homosexuality (MH) is a result of biological or cultural factors. The debate persists despite the fact that these two sides have different abilities to create a scientific environment to support their cause. Biological theorists produced evidence, however, that these are not always robust. On the other hand, social theorists, without direct evidence confirming their positions, criticize, with good argument, methods and results of the other side. The aim of this *Controversy* is to understand the reasons of both perspectives.

Methods. Two scientists (R.B. and A.C.C.) with expertise in the area of biology of MH were asked to contribute their opinions. The nurture position is discussed by a third expert in sexology (J.B.).

Main Outcome Measure. Expert opinion supported by the critical review of the currently available literature.

Result. The role of the *Controversy's* editor (E.A.J.) is to highlight the strengths and weaknesses of both sides. The two experts of the biological issue answer with their data to the questions: "Is male homosexuality partly explainable by immunology?" and "How is male homosexuality a Darwinian paradox?", respectively. Genetic and immunological factors, birth order, and fertility of relatives are largely discussed. Finally, the expert sustaining the idea that culture and experiences are important determining factors in sexual orientation used a psychosocial and holistic perspective to explain his position.

Conclusions. The *JSM's* readers should recognize that there are several biological factors in MH. However, these findings do not seem to be able to explain all cases of homosexuality. Some others may be due to particular environmental factors. The issue is complicated and multifactorial, suggesting that further research should be undertaken to produce the final answer to the question raised in this *Controversy* section. **Jannini EA, Blanchard R, Camperio-Ciani A, and Bancroft J. Male homosexuality: Nature or culture? J Sex Med 2010;7:3245–3253.**

Key Words. Homosexuality; Nature; Culture; Nurture; Genetics; Birth Order; Hormones; Testosterone; Reparative Therapy

Is there anything more controversial than homosexuality and its origin? Despite the evidence that bisexual or homosexual behavior is largely practiced in nature, in close to 1,500 species no less, ranging from primates to gut worms [1], the majority of religious authorities, as well as some political institutions, consider sex

with people of the same gender unnatural. Confusion between the level of biological evidence with the level of moral and metaphysic aspects has been and is, unfortunately, frequent. However, we can immediately answer the question in the title: yes, definitively, homosexuality is natural. Would this suggest that biology plays a major role in

homosexuality? Probably, but not necessarily. While some people believe that sexual orientation is innate and fixed, for others, sexual orientation may develop across a person's lifetime. The words of John Bancroft, which conclude this Controversy, clarify several aspects of these opposite interpretations.

This JSM Controversy is limited to male homosexuality (MH) for three reasons: (i) homosexuality in males (HM) is more common than homosexual females; (ii) MH is much more scientifically studied than female homosexuality; and (iii) moral and religious concerns seem much more concentrated on HM than on lesbian behavior.

An impressive amount of empirical data suggests that biology is an important regulator of both heterosexual and homosexual behaviors (Table 1). Evidence has been produced showing the importance of genetic, autoimmune, and neurohormonal factors in the development of sexual orientation. Criticizing methods and findings produced in the field of biology of sexual orientation,

Mustanski et al. admit that genetic research using family and twin methodologies has produced consistent evidence that genes influence sexual orientation, but molecular research has not yet produced compelling evidence for specific genes [24]. Although it has been well established that older brothers increase the odds of homosexuality in men, the route by which this occurs has not been fully resolved. Even the robust and elegant evidence solving the Darwinian paradox (how an anti-reproductive gene may survive?) produced by Camperio-Ciani need to be confirmed in larger samples. This author discusses here the fertility advantages of carrying the "gene" of HM, using arguments similar to that known for the thalassemia trait, which may confer a degree of protection against malaria, prevalent in the regions where the trait is common, thus conferring a selective survival advantage on carriers and perpetuating the mutation.

Although a number of excellent articles have been produced, the biology of HM is far from

Table 1 Biological evidences on male homosexuality

Site or mechanism	Finding	Author, Year (Ref.)
Anatomy (autopsies)	In the postmortem examination of HM brains, the suprachiasmatic nucleus of hypothalamus was found to be twice the size of its heterosexual counterpart.	Swaab, 1990 [2]
	The third interstitial notch of the anterior hypothalamus (INAH3) is two to three times smaller in HM than in heterosexual men. The women examined also exhibited this phenomenon.	LeVay, 1991 [3]
	The anterior commissure of the hypothalamus is significantly larger in the HM subjects than that of the heterosexuals.	Allen, 1992 [4]
	The functional response patterns of the brain to sexual stimuli contain sufficient information to predict individual sexual orientation with high accuracy.	Siebner, 2009 [5]
Genetics (twin studies)	100% concordance between homosexual monozygotic twins, and only a 12% concordance for dizygotic twins.	Kallman, 1952 [6]
	52% of monozygotic twins, 22% of dizygotic twins, and 11% of adoptive brothers were homosexual.	Bailey, 1991 [7]
Genetics (Xq28)	An estimated level of Xq28 allele sharing between gay brothers is 64% instead of the expected 50%.	Hamer, 1999 [8]
	The Xq28 linkage is not completely confirmed. Evidence for linkage at three other sites—on chromosomes 7, 8, and 10.	Hamer, 2005 [9]
	Linkage between the Xq28 markers and sexual orientation was detected for the HM families but not for the lesbian families.	Hamer, 2006 [10]
Genetics (fertility)	Genetic factors influencing homosexuality contribute to female fecundity of the maternal line.	Camperio-Ciani 2004–2009 [11–14]
Immunology	Following maternal immunization against male-specific molecules, maternal antimale antibodies may divert the sexual differentiation of the fetal brain from the male-typical pathway.	Blanchard, 1996–2008 [15–21]
Hormones	Higher circulating androgens in adult HM respect to heterosexual HM and heterosexual women respond similarly to male pheromones.	Brodie, 1974 [22]
	Both gay men and heterosexual women display a brain activation pattern distinct from that of heterosexual men.	Lindstrom, 2005 [23]

Note that the articles of two of the authors of this Controversy (Camperio-Ciani and Blanchard) are here pulled, being better explained in the corresponding sections.
HM = homosexuality in males.

complete. Morphofunctional studies suggest that HM is due to brain “feminization.” I invited Dr. Blanchard to summarize here the interesting findings of his team that hypothesize the presence of maternal antibodies against some structures of male brain. Some cases of HM can be considered consequences of a kind of autoimmune feminization during fetal life. This theory follows tradition: because HM shows sex-atypical partner preference, they should be incompletely masculinized. Apparently in keeping with this idea are the experiments of Per Lindstrom who found with his coworkers that in contrast to heterosexual men and in congruence with heterosexual women, HM displayed hypothalamic activation in response to male pheromones [23]. However, this pattern can be a consequence of the sexual interest for the same gender rather than a cause of sexual orientation. Furthermore, this theory works only for the HM with an older brother.

Other evidence exists that disputes the notion that HM are “half women.” If hormones play a role in HM behavior, this role is more on the hyperandrogenic rather than on the hypogonadal side. Hypersexuality (as measured in the number of partners and sexual acts per time unit) seems more present in HM than in heterosexuals. In fact, testosterone levels are higher in HM than in non-homosexuals [22,25], perhaps reflecting a sexual activity level that is particularly frequent [26]. Even average genital size (provoking the desperation of legions of heterosexual dysmorphophobics [27]) has been suggested as being larger in HM [28]. Another dimorphic parameter, under androgen control, such as auditory evoked potentials, seems more “hypermasculine” than feminine when 53 homosexual and bisexual males were compared with 50 heterosexual males [29]. Finally, the length of the second and fourth fingers is a sexually dimorphic trait, which reflects the prenatal influence of androgen on males. HM without older brothers have a 2D:4D ratio indistinguishable from heterosexual. HM with older brothers have a “hypermasculinized” ratio, suggesting an androgenic maternal “memory” [30].

The groups supporting “nature” (genetics, anatomy, immunology, hormones, etc.) have produced several articles, some with methodological weaknesses and others with problems surrounding the reproducibility of data. This is probably due to the obvious (but rarely admitted) evidence that the HM are definitively not a homogenous group. On

the contrary, in the “culture/nurture” group (more frequent in the psychological and religious environment) the same risk does not occur, being almost nothing empirically verified. Another important difference between the two groups is the fact that “naturalists” (with few exceptions [31]) do not exclude the role of nurture, while who is sure that HM is a choice, a sin, or a consequence of experience usually scotomizes the importance of inherited or biological factors.

The psychosexual neutrality theory proposes that gender and orientation is the result of environmental influences, particularly by parents, guardians, friends, and relatives, that humans are psychosexually neutral at birth, and that the gender and sexual orientation are a consequence of the nurture received as children. Although apodictic in nature, this theory is on the basis of a possible therapy of homosexuality, more or less overtly considered a disease if not a sin. However, position statements of the major mental health organizations in the United States state that there is no scientific evidence that a homosexual sexual orientation can be changed by psychotherapy [32], often referred to as “reparative” or “conversion” therapy. The hypothesis is that some individuals whose sexual orientation is predominantly homosexual can, with some form of reparative therapy, become predominantly heterosexual. The unique article showing effectiveness of reparative therapy deals with 143 male and 57 female homosexuals in a particular sample of people who reported at least some minimal change from homosexual to heterosexual orientation that lasted at least 5 years [33]. Interestingly, this article, which contains methodological problems, admits that complete shifts were uncommon and that female participants reported significantly more change than did HM. It is also interesting to note that while a very large number of books have been published on the successes of reparative therapy, crossing the three terms reparative + therapy + homosexuality in Medline, the only experimental article so far published is the Spitzer article [33].

Nurture theorists claimed to be able to identify typical familiar patterns (low paternal presence, high maternal cures) in HM. This idea follows the Freudian hypothesis that HM is the consequence of an unresolved Oedipus complex due to the male’s castration anxiety. However, the cause-effect relationship could be totally reverted, with the described familiar pattern being a consequence

of having a homosexual son rather than a cause of it.

After centuries of persecution, homosexuality has been removed from the list of diseases. In the index of the Diagnostic and Statistical Manual of Psychological Disorders (DSM-IV-TR) [24] the term “homosexuality” is simply absent. Being not a disease, homosexuality cannot be cured. However, homophobia, a phobic, psychopathologic trait, possibly should be present within the forthcoming edition of DSM.

Emmanuele A. Jannini, MD

Is MH partly explainable by immunology? Numerous studies have found that older brothers increase the odds of homosexuality in later-born human males. Older sisters, younger brothers, and younger sisters have no effect on the odds of homosexuality in males, and no class of siblings has consistently been shown to influence the odds of homosexuality in females. This phenomenon has therefore been called the *fraternal birth order effect*.

Research published since the last major review [21] has continued to demonstrate the wide variety of populations in which homosexuality is statistically associated with having a greater number of older brothers. These include males in the United States and Canada [34], Italy [13], Spain [35], and Independent Samoa [36]. They also include Canadian sex offenders [37]. Gómez-Gil et al. [35] confirmed previous findings that within the heterogeneous population of male-to-female transsexuals, it is that subgroup who is exclusively attracted sexually to other males who has the greater average number of older brothers. This suggests that homosexual orientations in males have the same origins, whether the individual thinks of himself as a gay man or as a “heterosexual woman” who happens to be “trapped in a man’s body.”

Blanchard and Bogaert [15] calculated that each additional older brother increases a male’s odds of homosexuality by 33%. Subsequent studies have produced both higher and lower estimates (see [21]). Cantor, Blanchard, Paterson, and Bogaert [18] showed that if one accepts an odds increase of 33% and assumes a prevalence of homosexuality of 2% for men with no older brothers, then the effect of fraternal birth order would exceed all other causes of homosexuality in groups of gay men with three or more older brothers and would precisely equal all other causes in a theoretical group with 2.5 older brothers.

Bogaert [38] found that biological brothers increase the odds of homosexuality in later-born males, even if they were reared in different households, whereas stepbrothers or adoptive brothers have no effect on sexual orientation. Thus, the available evidence indicates that the effect is prenatal.

To explain the fraternal birth order effect, Blanchard and Bogaert [15] formulated the *maternal immune hypothesis*, which was gradually elaborated in subsequent articles (see [21] for references). This hypothesis runs as follows: The fraternal birth order effect may be triggered when fetal cells (or cell fragments) enter the maternal circulation, an event especially common during childbirth. If these cells are from a male fetus, they may include substances that only occur in, or on the surfaces of, male cells. The mother’s immune system recognizes these male-specific molecules as foreign and starts producing antibodies to them. Following maternal immunization, maternal antimale antibodies are available to cross the placental barrier and enter the brain of a male fetus. These antibodies somehow divert the sexual differentiation of the fetal brain from the male-typical pathway, so that the individual will later be attracted to men rather than women. The probability—or strength—of maternal immunization increases with each male fetus; therefore, the probability of homosexuality increases with each older brother.

The maternal immune hypothesis was never intended to account for the sexual orientation of all homosexual men. The mere fact that half or more of all homosexual men have zero older brothers (see [18], Table 1) demonstrates that other etiological factors must account for at least half of existing homosexual men, and formal calculations of the population attributable fraction indicate that such factors cumulatively account for approximately 70–85% of them [18,19]. These other etiological factors probably include polymorphic genes and possibly include atypical hormone levels at critical stages of fetal development.

The fraternal birth order effect is the most broadly established causal factor in the whole research field of human homosexuality. In contrast, the maternal immune hypothesis advanced to explain it has no direct empirical support. It may be many years before anyone acquires funding to carry the question into the laboratory, partly for historical reasons. There is no research tradition of immunology and sexual behavior comparable with

the rich research tradition of hormones and sexual behavior, or the accepted tradition of studying genetics in relation to everything.

Ray Blanchard, PhD

There is a growing interest about the influence of both genetic and environmental components on the development of sexual orientation. This is a hot nature–nurture scientific conflict. There are a number of familial and population studies [11,14,36,39,40], as well as developmental and clinical studies [3,16,22], all suggesting a role of behavioral genetics in sexual orientation. Recent estimates show that at present around at least 180–200 million individuals of both sexes exhibit a homosexual orientation: is this due to their “nature” or to something that happened during their development? Recently there have been controversial “rehabilitative therapies” popularized in the media, which promise a “cure” for homosexuality. The promoters argue that “abnormal” sexual orientation is a consequence of early childhood traumas [41,42] and propose a reparative therapy based on psychoanalysis, group therapy, and spiritual help. These claims received a vast echo in society despite scientific associations (American Medical Association, American Psychiatric Association, etc.) having censured such rehabilitative “therapies” as non-evidence based and possibly harmful for clients. It is therefore important that such bio-social issues be investigated in with a sound scientific perspective.

We empirically demonstrated, in 2004, the “maternal fertile female hypothesis,” which is a genetic model for MH [11]. We offered a convincing solution to the possible “Darwinian Paradox” (genes for homosexuality could not survive or diffuse in a population as they promote nonreproductive behaviors). We showed, on the contrary, that families with homosexuals reproduce even more than the families with no homosexuals. Investigating a sample of homosexual males and their families (over 5,000 individuals), we found in the homosexual sample that the ascendant females in the maternal line, and not in the paternal line, were significantly more fecund than the ascendant females in the maternal line of heterosexual males. The ascendants of homosexuals generated, in fact, up to one-third more offspring than those of heterosexuals [11]. We also found that this fecundity model can be applied to bisexual males and their families [14]. This was also extended to other

European populations, and the results showed that the “maternal fertile female hypothesis” is not limited to the Italian population. Further confirmation was obtained in studies conducted in England and Canada [13]. We found that the same genetic factors that, when inherited by males, influence the likelihood of becoming homosexual promote female fecundity when inherited by females, balancing these particular factors’ fitness. We confirmed that the genetic factor Genetic factor of Maternal line Fecundity of Homosexuals is associated with the X chromosome. This is in keeping with the results of Hamer, who has long ago suggested the importance of the q28 region on the X chromosome [10,40]. Our research also confirmed a complementary role of a developmental phenomenon called “maternal immunization,” originally described by Blanchard [16], which influence birth order in homosexuals.

A number of hypotheses have attempted to give an evolutionary explanation for the long-standing persistence of this trait and for its asymmetric distribution in family lines; however, a satisfactory understanding of the population genetics of MH was lacking for a long time.

Kin selection was earlier invoked, later refuted, and lastly resurged [36]. The more recent debate is broadly focused on three arguments, not all based on genetic factors: the *overdominance* (i.e., male heterozygous advantage), the *maternal effects* on male offspring (such as maternal selection or maternal genomic imprinting), and the *sexually antagonistic selection* (SAS) (see [14] for references).

We recently performed a systematic mathematical analysis of the propagation and equilibrium of the putative genetic factors for MH in the population, based on the selection equation for one or two di-allelic loci and Bayesian statistics for pedigree investigation. We showed that only the two-locus genetic model with at least one locus on the X chromosome and in which gene expression is sexually antagonistic accounts for all previously described empirical data [12]. The SAS model for homosexuality works by disfavoring reproductive success on males while promoting it on the females. Our model clarifies the basic evolutionary dynamics of MH, confirming that this trait will never go extinct or to fixation in the population and will always be at low frequency, universally distributed, and with marked asymmetries both in its distribution and fecundity effects. The SAS is a well-described selective process in insects, in birds,

and in some mammals. Through different explorations of this model, we concluded that the GFMH excludes epigenetic maternal factors and includes more than one gene. The first one is a regulatory gene placed on the X chromosome, and the other(s) are on the autosomes, being coessential in influencing both female fecundity and male sexual orientation. Our model shows that all else being the same, a higher proportion of homosexuals in a population indicates a comparatively higher total fecundity increment. If, because of external conditions, the population's baseline fecundity is falling, the increment of the population's fecundity due to the presence of the GFMH becomes proportionally more pronounced, mimicking a "buffer effect" on any factors inducing the total fecundity decrease.

It should be noted that most studies focused exclusively on MH and that not all conclusions can be transferred to lesbians. Female homosexuals are somehow more complex to study, and we are currently developing a new lesbian behavior model.

In conclusion, these studies suggest that the dynamics of genetic factors influencing homosexuality and contributing to maternal line female fecundity point at homosexuality are useful in promoting fecundity in our populations and are a natural aspect of human sexual variability. These findings further discredit the assumptions that homosexuality is pathological and that it should be cured rather than accepted and respected.

Andrea Camperio-Ciani, PhD

The explanation of homosexuality has had a chequered career within the medical profession. Reactions have included it being rejected as a sin to attempts to excuse it as a pathology, thereby converting sin to sickness. Attempts to give a scientific explanation continue, with varying degrees and types of observer bias.

From my perspective, I find it useful to start with a "three-strand model" of sexual development. The three strands are (i) gender identity (the sense of being male or female); (ii) capacity for dyadic relationships; and (iii) sexual responsiveness. During childhood these three strands are relatively independent of each other. However, as puberty is approached they start to interact in various ways, generating the need for the individual to sort out what sort of sexual person he is. This results in what can be called "sexual identity," and I prefer this term to the more usual "sexual

orientation" as it acknowledges this active process of identity formation and how it is markedly influenced by socio-cultural factors.

Gender identity is a key factor. A boy who develops gender nonconformity, i.e., feeling and/or behaving in ways that are more typical of girls, has a substantially increased likelihood of ending up with a homosexual identity [43]. What we do not know is whether the gender nonconformity leads to the homosexual identity, which it may do, for example, by its impact on the boy's peer group relationships, or whether there is some developmental factor that is relevant to both gender and sexual identity, although separately. Early in the 20th century, Hirschfeld proposed that homosexuality was a form of hormonal intersex. When it became possible to measure the relevant hormones, no support for this was found. Similarly, the gender differences in the ratio of 2D to 4D finger length, which are attributed to early androgen effects, have been looked for in homosexual men, with so far inconsistent and inconclusive results [24]. Given the differences in brain structure and function between men and women, there has been a search for evidence of female brain characteristics in homosexual men. There is very limited evidence that INAH 3, one of the nuclei in the preoptic anterior hypothalamic region, which is smaller in women than in men, is also smaller in homosexual than heterosexual men [44]. An interesting difference in the brain processing of olfactory cues has been identified [23]. Homosexual men are similar to heterosexual women in their brain reaction to a typical male pheromone. However, we cannot say whether this reflects an innate brain difference in homosexual men or is a learned pattern that follows the establishment of homosexual identity. This is an issue when considering brain differences in general, given the scope for the effects of learning, particularly during the important phase of brain development in the second decade of life.

The capacity for dyadic relationships, while of considerable relevance to sexual identity, has received little research attention so far. Of obvious importance is the extent to which an individual can integrate his same-sex attraction into a close, rewarding same-sex relationship.

When we consider sexual responsiveness, it is clear that children vary markedly in the age when they start to experience sexual arousal. Of more direct relevance is when they start to link sexual

arousal to a sexually attractive “other person.” There is evidence across a number of studies for sexual attraction to emerge slightly earlier in pre-homosexual males, around an average age of 10 (i.e., before puberty), although with considerable variability [45]. This raises the possibility that experiencing sexual attraction at an earlier age may increase the likelihood of developing same-sex attraction. This might result from differences in peer group contacts at the earlier age, but as yet we can only speculate.

An additional factor of potential importance is the conditionability of sexual response to specific stimuli. There is a striking gender difference in this respect; the more unusual forms of sexual preference, such as fetishism, are almost exclusively found in men. This is consistent with the idea that sexual attraction is stimulus specific in men, whereas in women it is more determined by relationship factors. But as yet we do not know to what extent the conditioning of specific sexual stimuli, presumably during a critical phase of conditionability, applies to men in general. It may only be relevant to a minority, whether the specific stimulus is normal female, male, or fetish. In such individuals there may be less bisexual potential.

All of these potential determinants considered so far may be genetically influenced. Genetic factors have been looked at in three ways; (i) does homosexuality run in families? The rate of homosexuality in brothers is around 9%, higher than prevalence estimates in population samples [46]. (ii) Monozygotic twins have, consistently across studies, shown higher concordance for homosexuality than dizygotic twins. These results point to both genetic and nonshared environmental influences [24]. (iii) Are there specific genetic markers of homosexuality? Hamer and his colleagues reported evidence of a relevant gene in the Xq28 region of the X chromosome, which in males is inherited from the mother. They went on to report one successful replication of this finding, whereas two attempts to replicate this by other researchers failed [24]. In a more recent genetic study involving a full genome scan of 456 individuals from 146 families with two or more homosexual brothers, the authors commented that given the complexity of sexual orientation, numerous genes are likely to be involved, and the modest levels of linkage found for the X chromosome can account for only a fraction of the overall heritability [9]. They went on to report suggestive findings for three addi-

tional regions of the genome. These findings require replication, but in any case they may relate to genotypic mechanisms of only indirect relevance to sexual identity development.

As mentioned earlier, the process of integrating these strands of sexual development into a “sexual identity” is influenced substantially by sociocultural factors. Herdt [47], a cultural anthropologist, divides patterns of development into continuous and discontinuous, which vary across cultures, and through history. The discontinuous pattern is probably of most relevance in demonstrating sociocultural determination. This involves a series of stages that, although they follow a particular sequence, differ in substantial ways from each other, with varying degrees of awareness or involvement by family or society. Of particular relevance is the pattern in which the individual passes through a phase of homosexual activity onto a later, final heterosexual phase. One of the best known examples is from the Sambia, a mountain people in Papua New Guinea, well described by Herdt. This is a strongly sex-segregated society, in which young boys around the age of 10 are taken from their mother’s care into an all-male dormitory in which they are taught to fellate older boys and to swallow their semen. The justification for this was the belief that ingesting the semen was necessary for normal masculine development. As each boy passed through puberty he would enjoy being fellated by prepubertal boys and eventually leave the dormitory and start his heterosexual life, leading to marriage. Here we have an institutionalized discontinuous pattern. In Western society, we see discontinuous patterns that have varied in the extent to which they have been recognized. In the first half of the 20th century, we saw a covert pattern in early adolescence. Kinsey’s finding that 37% of men reported some homosexual experience mainly involved early adolescent experience [48]. Schmidt et al. [49] showed, in Germany, that such early adolescent same-sex male interaction declined substantially between 1970 and 1990. This decline may have resulted from more opportunities for young adolescent boys to have sexual interaction with girls but may also have reflected a much greater awareness that such same-sex behavior is taken to indicate homosexual identity.

For understandable political reasons, the Gay Rights movement that emerged in the 1960s and 70s asserted a clear distinction between homo-

sexual and heterosexual identity, dismissing bisexuality in the process. However, the cross-cultural and historical evidence points to a bisexual potential that becomes shaped in various ways and at different times by sociocultural processes. It is also noteworthy that since the impact of the Gay Rights movement, we have been seeing the impact of the Internet with its facilitation of a much more variable and less stable range of "sexual identities." We should also keep in mind that whereas homosexual interactions are common across many species, exclusive homosexual involvement, with the rejection of opportunities for heterosexual activity, is exceedingly rare in nonhumans.

At the present time, therefore, we have no clear evidence of a specific determinant of homosexuality, but indications that a number of factors, varying in importance across individuals, can interact to make same-sex interaction and attraction more likely, followed by the impact of socio-cultural "constructionism" on sexual identity formation.

John Bancroft, MD

Corresponding Author: Emmanuele A. Jannini, MD, Course of Endocrinology and Medical Sexology, Department of Experimental Medicine, University of L'Aquila, L'Aquila 67100, Italy. Tel: +39 0862433530; Fax: +39 0862433523; E-mail: emmanuele.jannini@univaq.it

Conflict of Interest: None.

References

- 1 Bagemihl B. Biological exuberance: Animal homosexuality and natural diversity. New York: St. Martin's Press; 1999.
- 2 Swaab DF, Hofman MA. An enlarged suprachiasmatic nucleus in homosexual men. *Brain Res* 1990;537:141-8.
- 3 LeVay S. A difference in hypothalamic structure between heterosexual and homosexual men. *Science* 1991;253:1034-7.
- 4 Allen LS, Gorski RA. Sexual orientation and the size of the anterior commissure in the human brain. *Proc Natl Acad Sci U S A* 1992;89:7199-202.
- 5 Ponseti J, Granert O, Jansen O, Wolff S, Mehdorn H, Bosinski H, Siebner H. Assessment of sexual orientation using the hemodynamic brain response to visual sexual stimuli. *J Sex Med* 2009;6:1628-34.
- 6 Kallmann FJ. Twin and sibship study of overt male homosexuality. *Am J Hum Genet* 1952;4:136-46.
- 7 Bailey JM, Pillard RC. A genetic study of male sexual orientation. *Arch Gen Psychiatry* 1991;48:1089-96.
- 8 Hamer D. Genetics and male sexual orientation. *Science* 1999;285:803.
- 9 Mustanski BS, Dupree MG, Nievergelt CM, Bocklandt S, Schork NJ, Hamer DH. A genomewide scan of male sexual orientation. *Hum Genet* 2005;116:272-8.
- 10 Hu S, Pattatucci AM, Patterson C, Li L, Fulker DW, Cherny SS, Kruglyak L, Hamer DH. Linkage between sexual orientation and chromosome Xq28 in males but not in females. *Nat Genet* 1995;11:248-56.
- 11 Camperio-Ciani A, Corna F, Capiluppi C. Evidence for maternally inherited factors favouring male homosexuality and promoting female fecundity. *Proc Biol Sci* 2004;271:2217-21.
- 12 Camperio Ciani A, Cermelli P, Zanzotto G. Sexually antagonistic selection in human male homosexuality. *PLoS One* 2008;3:e2282.
- 13 Iemmola F, Camperio Ciani A. New evidence of genetic factors influencing sexual orientation in men: Female fecundity increase in the maternal line. *Arch Sex Behav* 2009;38:393-9.
- 14 Ciani AC, Iemmola F, Blecher SR. Genetic factors increase fecundity in female maternal relatives of bisexual men as in homosexuals. *J Sex Med* 2009;6:449-55.
- 15 Blanchard R, Bogaert AF. Homosexuality in men and number of older brothers. *Am J Psychiatry* 1996;153:27-31.
- 16 Blanchard R, Klassen P. H-Y antigen and homosexuality in men. *J Theor Biol* 1997;185:373-8.
- 17 Lalumiere ML, Blanchard R, Zucker KJ. Sexual orientation and handedness in men and women: A meta-analysis. *Psychol Bull* 2000;126:575-92.
- 18 Cantor JM, Blanchard R, Paterson AD, Bogaert AF. How many gay men owe their sexual orientation to fraternal birth order? *Arch Sex Behav* 2002;31:63-71.
- 19 Blanchard R, Bogaert AF. Proportion of homosexual men who owe their sexual orientation to fraternal birth order: An estimate based on two national probability samples. *Am J Hum Biol* 2004;16:151-7.
- 20 Blanchard R. Quantitative and theoretical analyses of the relation between older brothers and homosexuality in men. *J Theor Biol* 2004;230:173-87.
- 21 Blanchard R. Review and theory of handedness, birth order, and homosexuality in men. *Laterality* 2008;13:51-70.
- 22 Brodie HK, Gartrell N, Doering C, Rhue T. Plasma testosterone levels in heterosexual and homosexual men. *Am J Psychiatry* 1974;131:82-3.
- 23 Savic I, Berglund H, Lindstrom P. Brain response to putative pheromones in homosexual men. *Proc Natl Acad Sci U S A* 2005;102:7356-61.
- 24 Mustanski BS, Chivers ML, Bailey JM. A critical review of recent biological research on human sexual orientation. *Annu Rev Sex Res* 2002;13:89-140.
- 25 Meyer-Bahlburg HF. Psychoendocrine research on sexual orientation. Current status and future options. *Prog Brain Res* 1984;61:375-98.
- 26 Jannini EA, Fisher WA, Bitzer J, McMahon CG. Is sex just fun? How sexual activity improves health. *J Sex Med* 2009;6:2640-8.
- 27 Ghanem H, Shamloul R, Khodeir F, ElShafie H, Kaddah A, Ismail I. Structured management and counseling for patients with a complaint of a small penis. *J Sex Med* 2007;4:1322-7.
- 28 Bogaert AF, Hershberger S. The relation between sexual orientation and penile size. *Arch Sex Behav* 1999;28:213-21.
- 29 McFadden D, Champlin CA. Comparison of auditory evoked potentials in heterosexual, homosexual, and bisexual males and females. *J Assoc Res Otolaryngol* 2000;1:89-99.
- 30 Williams TJ, Pepitone ME, Christensen SE, Cooke BM, Huberman AD, Breedlove NJ, Breedlove TJ, Jordan CL, Breedlove SM. Finger-length ratios and sexual orientation. *Nature* 2000;404:455-6.
- 31 Swaab DF, Garcia-Falgueras A. Sexual differentiation of the human brain in relation to gender identity and sexual orientation. *Funct Neurol* 2009;24:17-28.

- 32 American Psychiatry Association (APA). Commission on Psychotherapy by Psychiatrists. Position statement on therapies focused on attempts to change sexual orientation (reparative or conversion therapies). *Am J Psychiatry* 2000;157:1719–21.
- 33 Spitzer RL. Can some gay men and lesbians change their sexual orientation? 200 participants reporting a change from homosexual to heterosexual orientation. *Arch Sex Behav* 2003;32:403–17. discussion 419–72.
- 34 Schwartz G, Kim RM, Kolundzija AB, Rieger G, Sanders AR. Biodemographic and physical correlates of sexual orientation in men. *Arch Sex Behav* 2010;39:93–109.
- 35 Gomez-Gil E, Esteva I, Carrasco R, Almaraz MC, Pasaro E, Salamero M, Guillamon A. Birth order and ratio of brothers to sisters in spanish transsexuals. *Arch Sex Behav* 2010 Mar 16 [Epub ahead of print] doi: 10.1007/s10508-010-9614-3.
- 36 Vanderlaan DP, Vasey PL. Male sexual orientation in Independent Samoa: Evidence for fraternal birth order and maternal fecundity effects. *Arch Sex Behav* 2009 Dec 29 [Epub ahead of print] doi: 10.1007/s10508-009-9576-5.
- 37 Rice ME, Harris GT, Lang C, Chaplin TC. Sexual preferences and recidivism of sex offenders with mental retardation. *Sex Abuse* 2008;20:409–25.
- 38 Bogaert AF. Biological versus nonbiological older brothers and men's sexual orientation. *Proc Natl Acad Sci U S A* 2006;103:10771–4.
- 39 Bailey JM, Pillard RC, Dawood K, Miller MB, Farrer LA, Trivedi S, Murphy RL. A family history study of male sexual orientation using three independent samples. *Behav Genet* 1999;29:79–86.
- 40 Hamer DH, Hu S, Magnuson VL, Hu N, Pattatucci AM. A linkage between DNA markers on the X chromosome and male sexual orientation. *Science* 1993;261:321–7.
- 41 Nicolosi J, Freeman L. *Healing homosexuality: Case stories of reparative therapy*. New York: J. Aronson; 1993.
- 42 Nicolosi J. *Shame and attachment loss: The practical work of reparative therapy*. Darthmouth Groove, IL: IterVersity Press; 2009.
- 43 Zucker KJ. *Gender identity disorders in children: Clinical descriptions and natural history*. In: Blanchard R, Steiner BW, eds. *Clinical management of gender identity disorders in children and adults*. Washington, DC: American Psychiatric Press; 1990:1–23.
- 44 Hines M. *Sexual differentiation of human brain and behavior*. In: Pfaff DW, Arnold AP, Etgen AM, Farhbach SE, Rubin RT, eds. *Hormones, brain and behavior*. San Diego, CA: Academic Press; 2002:425–62.
- 45 Bancroft J. *Human sexuality and its problems*. 3rd edition. Edinburgh: Churchill Livingstone/Elsevier; 2009.
- 46 Baron M. *Genetics and human sexual orientation*. *Biol Psychiatry* 1993;33:759–61.
- 47 Herdt G. *Developmental discontinuities and sexual orientation across culture*. In: McWhirter D, Sanders SA, Reinisch JM, eds. *Homosexuality/heterosexuality: Concepts of sexual orientation*. Oxford: Oxford University Press; 208–36.
- 48 Kinsey A, Pomeroy WB, Martin CE. *Sexual behavior in the human male*. Philadelphia: W.B. Saunders; 1948.
- 49 Schmidt G, Klusmann D, Zeitzschel U, Lange C. *Changes in adolescents' sexuality between 1970 and 1990 in West-Germany*. *Arch Sex Behav* 1994;23:489–513.