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Science and the Ecclesiastical Homosexuality Debates

Stanton L. Jones and Mark A. Yarhouse

Wheaton College

Contemporary American ecclesiastical debates about the moral status of homosexual acts and orientation have focused primarily on two matters: the blessing or sanctioning of homosexual relationships or the performing of marriages of same gender individuals, and the ordination of noncelibate homosexual persons. Casual citation of scientific research on homosexuality is common in the context of these debates, especially in the study and support documents of the mainline Christian denominations. After the stem "Science says. . .," sweeping generalizations often are made, after which follow ethical conclusions which are loosely tied to the putative scientific evidences cited. Because the linkage between scientific findings and moral argumentation is usually implicit rather than explicit, we recently examined Christian mainline denominational documents from these debates to try to make sense of the uses to which such supposed scientific findings are put.[1] We concluded that "science" typically is used in two ways in these documents: as a rhetorical device to undermine the supposed assumptions upon which traditionalist arguments rest, and as implicit support for consideration of homosexual acts as morally neutral in themselves.

Citations of the scientific findings on homosexuality in these debates continue to be poorly grounded in the primary literature. For that reason, we here review the contemporary status of this scientific research under the major headings which continue to best describe the types of evidences cited,[2] namely 1) prevalence, 2) etiology, 3) status as a mental disorder, and 4) efficacy of change methods. In offering this review, we hope to increase awareness that the actual scientific findings in this area are more complex and puzzling than is usually acknowledged, and to discourage future selective or simplistic reporting of such findings. In closing, we will briefly argue that the formal

relevance of the findings of science to the ecclesiastical debate is minimal, and that the moral status of homosexual behavior must be adjudicated on theological grounds alone.

We note at the outset that research on sexual orientation generally or homosexuality specifically is plagued by pernicious problems. One problem is the diversity of persons to whom the description "homosexual" is applied and the question of whether to categorize people by behavior (which behaviors and to what degree?), self-identification, or some other variable. Individual and subgroup differences may be sacrificed when the generic labels "homosexual" or "gay and lesbian" are used. The essentialist/social constructionist debate further clouds these definitional issues. Essentialist assumptions, basically that sexual orientation is a stable and fundamental aspect of human character which is accurately described by the taxonomy of our contemporary understanding, are common on the part of both researchers and research participants alike and considerably complicate how we understand the data generated.[3] Constructivists such as Haldeman, in contrast, argue that our contemporary taxonomies are social constructions, and thus that the "categories of homosexual, heterosexual, and bisexual, considered by many researchers as fixed and dichotomous, are in reality very fluid for many;"[4] if so, the dichotomous "gay and straight" or trichotomous "gay, straight and bi" may not be the firm foundation for asking questions which many imagine. A second problem has been the research focus upon male homosexuals (gays), with very little in comparison being done with lesbians. This may be an expression of rampant sexism, but at least in recent years seems mostly due to the targeting of recent research at understanding HIV-infection risk behaviors which men are more likely to display. A third problem is the difficulty or impossibility of obtaining a random and representative sample of homosexual individuals. While the studies in Table 1 below, for example, each utilized nationally representative samples, the subpopulation of homosexual individuals for each was small and the survey protocols were designed for a generic (largely heterosexual) sample rather than being able to ask the many specific questions regarding etiological variables, behavior patterns, and life-style of homosexuals which might be desirable. In contrast, many of the most famous studies of homosexual individuals[5] were performed on convenience samples of questionable representativeness. Fourth, failure to replicate findings has plagued research in this area.

Prevalence

The apocryphal prevalence figure that "10% of the adult population is homosexual" appears frequently in church documents. One denominational report[6] listed ten "myths" it aspired to dispel, the first being that, "Gays and

lesbians constitute only a small segment of the general population and are an urban phenomenon," to which the authors replied, "Research from several sectors indicates that at least 10 percent of the American population or approximately 22 million persons are predominantly gay or lesbian." This apocryphal 10% figure has been attributed to the Kinsey study of males,[7] where he reported that 4% of white males were exclusively homosexual throughout life after adolescence, and that a total of 10% of white males were mostly or exclusively homosexual during at least a three year period between the ages of 16 and 55. Kinsey's data appear to have over-represented male homosexuality due to sampling biases in his research, including the over-sampling of prison inmates and members of gay-affirming organizations.[8]

Numerous recent and more credible studies have produced remarkably consistent and much lower prevalence estimates than has been commonly assumed. We will summarize this data according to self-identification as homosexual and according to occurrence of same-gender sexual behavior. When prevalence is defined by self-identification of sexual orientation by the respondent, prevalence estimates range from 2% to 4%. Laumann et. al.[9], in the best survey study to date, found that 2.0% of men and 0.9% of women self-identified as homosexual, with an additional 0.8% and 0.5 % respectively self-identifying as bisexual and 0.3% and 0.1% as "other;" they also inquired of the degree to which respondents were attracted to members of the same sex (finding 6.2% of men and 4.4% of women reporting such attraction, independent of heterosexual attraction) and the degree to which they found the idea of sex with a same gender partner appealing (finding 4.5% of men and 5.6% of women reporting this appealing). Harry[10] similarly found that 2.4% of a national probability sample of men described themselves as homosexual, but added that if all respondents who described themselves to be bisexual and all subjects who refused to answer that particular survey question (the latter a dubious assumption) were also classified as homosexual, then up to 5.7% of the sample could be described as homosexual. In considerable contrast, one recent study[11] reported the highly provocative statistics that 20.8% of males and 17.8% of females in the U.S. (and similar incidences for the United Kingdom and France) report either sexual attraction toward or behavior with persons of the same gender. This remarkably higher statistic appears to be an artifact of a questionable survey design which forced respondents who had never engaged in homosexual behavior to choose between the items "I have absolutely never felt any sexual attraction towards someone of my own sex" and "I have felt attracted towards someone of my own sex, but never had any sexual contact with anyone." It would appear that this question forced anyone with the most vague attraction in

adolescence to respond to the latter stem, resulting in a considerable inflation of their reported incidences of same gender attraction. A reluctant rejection of the former stem hardly constitutes a declaration of stable same-gender attraction.

Studies of the prevalence of homosexuality grounded in same-gender sexual behavior yield the lowest estimates of prevalence. Table 1 contains a summarization of the major prevalence findings regarding same gender sexual behavior reported from studies which utilized national probability surveys, the best and most representative research method available to answer questions about prevalence. Most authors described their findings as "lower bound" estimates of the prevalence of homosexual behavior given likely reluctance on the part of survey respondents to admit to stigmatized behavior patterns.

Study[12]	Exclusive Same Gender Sex in Last ___ Years	Occurrence of Same Gender Sex in Last Year	Occurrence Last ___ Years	Occurrence Since Adulthood (Age __)	Occurrence Since Puberty (or Lifetime)
Billy et al. (1993) Males Only	1.1% in last 10 years		2.3% in last 10 years		
Fay et al. (1989) Males Only		1.6 - 2%		6.7% since age 19; (3.3% occasionally or fairly often)	20.3%
Laumann et al. (1994) Males		2.7%	4.1% in last five years	4.9% since age 18	9.1%
Laumann et al. (1994) Females		1.3%	2.2% in last five years	4.1% since age 18	4.3%
Rogers & Turner (1991) Males Only		Study 1- 1.9% Study 2- 1.2% Study 3- 2.4% Study 4- 2.0%		All since 18 Study 1- 4.8% Study 2- 4.9% Study 3- N/A Study 4- 6.7%	
Sell et al. (1995) Males	last 5 years: US- 0.82% UK- 1.15% Fr.- 0.72%		last 5 years: US- 5.42% UK- 3.51% Fr.- 9.94		
Sell et al. (1995) Females	last 5 years: US- 0.27% UK- 0.54% Fr.- 0.14%		last 5 years: US- 2.96% UK- 1.54% Fr.- 3.02%		
Spira et al. (1993) Males Only (France)		1.1%			4.1%
Stall et al. (1990) Males Only	0.8% in last five years	1.4% in last five years			
Wellings et al. (1994) Males (Britain)		1.1%			6.1%

Wellings et al. (1994) Females (Britain)		0.4%			3.4%

Table 1: Frequency Reports of Same Gender Sexual Behavior Over Various Time Periods From Major National Probability Survey Studies

Numerous findings are striking. One is the gap between the approximately 1% of males who engaged exclusively in same sex behavior in the past year and the 1.1% to 2.7% which experienced any same sex behavior in the past year. This gap may be partially explained by a separate and surprising finding[13] that 42% of the gay or bisexual men in a national probability sample were married to women. The low frequency of male lifetime homosexual experience in Table 1 compared to Kinsey's estimate of 37%, and the impossibility of his 10% estimate of 3-year exclusive male homosexual experience, are both remarkable.

The "myth" that "Gays and lesbians constitute only a small segment of the general population and are an urban phenomenon" may not be a myth after all. The population percentage of gays and lesbians is certainly smaller than previously estimated. Curiously, it is precisely because homosexuality is predominantly an urban phenomenon (whether by migration or subculture disinhibition[14]) that it may seem to some that 10% of the population is gay. A probability sample[15] of males in major urban centers (which was not representative of the country at large) was recently compared to a national probability sample, and found the percentages of men who had had sex only with other males in the last five years rose from 1.4% (national) to 3.7% (urban), with an additional 2.0% in the urban sample having had sexual relations with both men and women in the last five years. Similarly, Laumann et al. selectively examined respondents who lived in the 12 major urban centers of the U.S., and found that self-identification as either homosexual or bisexual increased to 9.2% of men and 2.6% of women, with reports of any same sex behavior since puberty increasing to 15.8% of men and 4.6% of women. There is some validity to the perception that homosexuality is more common in urban settings.

Conclusion. The rate of homosexuality as a stable life orientation in our culture is certainly not 10%. There is good evidence to suggest that less than 3%, and perhaps less than 2%, of males are homosexually active in a given year. The rate of males who engage in sustained homosexual practice over a significant period of adult life is probably less than 5% of the male population, and the rate of men who manifest a sustained and exclusive commitment to homosexual practice is certainly less than 3%. Female homosexuality continues to be estimated at

approximately half or less than the male rates; it appears to characterize less than 2% of the female population. So when the genders are combined, homosexuality almost certainly characterizes less than 3% of the population, and the correct percentage combining men and women might be lower even than 2%.

What causes homosexual orientation?

The etiology of homosexual behavior and orientation figures prominently in ecclesiastical debates. Much of this casual discussion springs from the caricature of the traditionalist position as asserting that homosexuality is always a "willful perversion;" hence, if homosexuality can be shown to be neither "willful" (by showing that development of the orientation is nonvoluntary) nor a "perversion" (by showing that the orientation is somehow "natural" to the person), the traditionalist case loses credibility. Regarding willfulness, one document leapt from the claim that "Expert opinion is largely agreed . . . that a sexual orientation is not, in the vast majority of cases, voluntary in the sense of a self-conscious choice" to the conclusion that "If it is granted that a homosexual orientation is involuntary . . . it is unjust to present celibacy as a calling."[16] Regarding naturalness, Sedgwick[17] argued that "Studies from the natural sciences suggest that homosexuality is an outcome of both biological and social factors. Homosexuality is not simply a matter of arrested development but a variable form of sexual identity in animals and human beings. Homosexual relations would then be moral as those relationships realize or embody the broader values of pleasure, mutuality, and generativity in their interrelationship." But what does the research on etiology show?

The past decade has witnessed a dramatic swing toward biological theories for the etiology of homosexuality. The major proposed causes for a homosexual orientation have included genetic, prenatal hormonal, adult (postnatal) hormonal, and psychological factors. The following is a brief summary of the current state of the evidence for each.[18]

Genetic factors. Judgments about the viability of a hypothesized genetic "cause" of homosexuality have been on a roller coaster in the last four decades. From a peak in the 1950s in response to Kallman's report of 100% concordance for homosexuality in identical twins,[19] to a subsequent valley produced by failures to replicate Kallman's findings lasting into the 1980s,[20] the genetic hypothesis has made a remarkable comeback.[21] Genetic studies look for concordance for homosexuality between siblings (or other family members), where concordance is

the rate at which two siblings both show the characteristic being studied; greater concordance between more closely genetically related siblings presumably indicates greater genetic causation. Recently, research into the actual chromosomal foundations for such correlations has begun. It appears that there is strong evidence of some genetic influence on the development of homosexual proclivity for some subpopulation of persons; the size and nature of this influence is unclear.

Bailey and Pillard published studies of male[22] and female[23] homosexuals which have forcefully advanced the genetic causation hypothesis. In terms of sample size and methodological sophistication, these are surely the most significant studies yet published in this area. Their study of male homosexuals reported a 52% concordance rate for homosexual preference among monozygotic twins, a 22% concordance for dizygotic twins, a 9.2% rate for nontwin brothers, and an 11% rate between adoptive brothers. Their study of female homosexuals reported a 48% concordance rate for homosexual preference among monozygotic twins, a 16% concordance for dizygotic twins, a 14% rate for nontwin sisters, and a 6% rate between adoptive sisters. Their statistical conclusion for both studies was that genetic factors, heritability, explain the majority of the variance in sexual orientation. One other study to date[24] has produced comparable results.

We would raise several concerns about these studies. First, there is the issue of replication. Other recent studies of the genetic hypothesis have not produced comparable results. A study[25] with a smaller sample size and simpler methodology than Bailey and Pillard's studies reported concordance rates for male and female homosexual identical twins mixed together of only 10% (if bisexuals are not counted as homosexual) or 25% (if bisexuals are counted as concordant with the homosexual twin); the higher estimate is still half or less of the rates reported by Bailey and Pillard.[26] Research from the Minnesota Twin Project[27] (which examines identical twins reared apart since birth) has also challenged Bailey and Pillard's findings by suggesting lower heritability rates; the researchers reported that all four female monozygotic twin pairs raised apart in which there was one homosexual twin were discordant for lesbianism, one male twin pair was concordant,[28] and one male twin pair was discordant.[29] The lesbian discordance in this study is utterly striking in comparison to Bailey and Pillard's remarkably high concordance rates for lesbians.

Second, and of greater concern, are the sampling methods of the Bailey and Pillard studies. With the biases of Kinsey's samples in mind, we may note that subjects for the studies were recruited through openly homophilic magazines and tabloids, and through general advertisements in the gay community. The ads stated that the

researchers were looking for gay men and lesbians with twin or same-sex adopted siblings. Given the political sophistication of the gay community, it is conceivable that some degree of volunteer bias could have affected the results of the study. If monozygotic twins who had homosexual twin brothers and sisters were more likely than discordant twin pairs to volunteer because they believed the study would produce benefits for the gay community, substantial bias could have swayed the findings.[30]

There were numerous other problems with the Bailey and Pillard studies. There should have been only insignificant differences in the concordance rates between dizygotic twins and nontwin brothers (these two sibling relationships share identical genetic overlap) but instead they found the dizygotic twin brothers to have twice the rate of concordance for homosexuality as nontwin brothers; their study of lesbians produced similar (and extraordinarily high) concordance rates for dizygotic twins and nontwin sisters. Further, the estimates of heritability generated in the study were speculative, as they had to incorporate estimates of the amount of error in the population and estimates of the base rate of homosexuality in the general population into their statistical model. How could their mathematical model produce estimates of heritability approaching 75% when fully half of the twin sets were discordant in sexual orientation, and when the base rates for homosexuality from these families far exceed the base rates reported in the best recent estimates of prevalence which we reviewed earlier? If the true population base rate of male homosexuality is 2%, then some process in these families increased the risk for male children in these families (even nonrelated adopted brothers which share no genes with their siblings) becoming homosexual five-fold over the base rate (from 2% to 10%). If the 50% estimate of monozygotic twin concordance holds, which seems unlikely, then genes increased the risk for these men by an additional five-fold; i.e., equivalent effects for nature/heredity and nurture/environment. Similarly for women, if the base rate of lesbianism is 1%, then nonrelated adopted women were six times more likely to become a lesbian by growing up in these families than in the general population, and fraternal siblings more than fourteen times more likely than the general population. Finally, if a genetic explanation is to be accepted as the most important causal influence in homosexuality, the 50% <u>non</u>concordance rate between monozygotic twins begs explanation. If two individuals share 100% of their genetic makeup (nature) and almost 100% of their formative developmental experiences (nurture), how can the high rate of discordance be explained? We would note for the reader that our core concern with the Bailey and Pillard studies is engendered by the large size of the genetic influence they claim to have obtained, not by the claim of genetic influence per se.

Only recently has the technology been developed to directly examine specific genetic sequences that may influence the development of homosexuality. In 1993, the media reported the discovery of a "sexual orientation gene" by Dean Hamer and his research team, though the authors of the research were much more circumspect in their report. There were two aspects to their project. First, a pedigree analysis of 76 men recruited out of an AIDS treatment program was performed, from which the researchers reported a strong pattern of homosexual orientation represented in maternal relatives but not in paternal relatives. This led to the second part of the study which focused on subjects only from families where there was pre-existing evidence of maternal transmission of homosexual orientation from the pedigree analysis. A "DNA linkage analysis" of 40 pairs of homosexual brothers from this subsample was conducted, which produced a finding that 33 of the 40 pairs shared a statistically significant concordance of the "Xq28 subtelomeric region of the long arm of the sex (X) chromosome." [31]

A replication and extension study by the same research team[32] reported three significant findings. First, this study of a new sample of homosexual males which again met the criterion of having a gay brother (a very select subsample of gay men) and being from a family which evidenced maternal genetic transmission produced nearly identical results to the previous study, with 22 out of 32 brothers sharing identical Xq28 markers, a statistically significant concordance. Second, the original Hamer study only examined genetic material shared by gay male siblings and did not check to see if discordant (non-gay) siblings shared the exact same material; if they had, it would have suggested that the chromosomal overlap was a related to a shared family trait that had little to do with sexual orientation; the second study found that heterosexual brothers were strikingly unlikely to share the same Xq28 markers, thus increasing the likelihood that the marker is indeed related to homosexual orientation. Third, a sample of lesbian sisters were also examined (though there is no evidence of maternal transmission of female homosexuality), with the result that no statistically significant effects were found; the authors conclude from this last finding that the Xq28 region is uninvolved with the occurrence of female homosexuality. The technical methodology of these studies appear to have been exceptional.

There are, naturally, problems and limitations with these studies as well. First, these findings have yet to be replicated by other research teams, and similar findings in the last decade reporting "genes for" manic-depression, violence, alcoholism and schizophrenia have often not been replicated or have been formally retracted.[33] A newspaper has reported,[34] in fact, that Hamer et al.'s studies have actually failed attempted direct replication in a

Canadian laboratory, though the Hamer team has warned that their findings depend critically on exact usage of their sample inclusion criteria, which may or may not explain any specific failure to replicate. Second, though the researchers described their samples as "randomly ascertained,"[35] they were in fact anything but random. The same types of volunteer bias noted in the Bailey and Pillard studies (or worse) could be operative here as well, though in this case such sample bias would challenge not the findings themselves, but the generalizability of the findings. In their second report, the authors[36] dealt with this limitation with commendable explicitness, noting that their findings really speak only to a subpopulation of homosexuals, i.e., predominant or exclusive male homosexuals with a homosexual brother where there is evidence of maternal genetic transmission. Third, and very seriously, allegations of selective reporting of data by Hamer and his collaborators to enhance statistical power have been made by a researcher on Hamer's team and are apparently in the process of being adjudicated.[37]

The major limitation (as opposed to flaw) of these studies is that they did not, contrary to the media reports about the first study, find a sexual orientation gene. They rather appear to have found a cluster of shared genetic segments which seem to relate to sexual orientation in this unusual and selective sample of male homosexuals. There are huge gaps between genotype and phenotype; i.e., there are many possible ways in which some shared genetic material (the genotype) might contribute to the highly complex and variable human outcome which we call sexual orientation. It is quite clear that these studies did not find a chromosome which causes homosexual orientation: "the Xq28 region was neither necessary nor sufficient for a homosexual orientation."[38] The findings may be of a chromosome set that has a somewhat direct effect on orientation, or may be a marker for or cause of temperamental or other variables that exert their influence indirectly and thus make homosexuality more likely to occur.[39] Hu et al. acknowledge this explicitly by not calling the Xq28 region a sexual orientation locus, but a "sexual orientation related locus."[40] The region appears to relate to the development of sexual orientation for a subpopulation of men, but how it does so is as of now a mystery.

Turner[41] presented evidence in favor of Hamer et al.'s hypothesis of causation by a gene in the Xq28 region of the X chromosome. He offered analyses of familial "pedigree charts" of a sample gathered by convenience which purported to show transmission of homosexuality only through the maternal side of each family. Turner suggested that there was a correlation between transmission of homosexuality and transmission of various chromosomally based disease processes (which also result in various fertility problems such as frequent miscarriage) which are

thought to be the result of genes in the Xq28 region. These results await replication. One recent finding[42] appears to challenge the Hamer and Turner findings of maternal-only transmission of homosexual orientation by reporting elevated levels of homosexuality among sons of homosexual fathers. Though the authors argued that their finding that 9% of the sons were "nonheterosexual" was within the normal range, if the general population incidence among men is approximately 3%, the presence of a gay father would appear to have tripled the incidence of homosexuality. The exact percentage of gay sons in this study is disputable; if analyses were limited only to cases where the son himself rated his own sexual orientation, then 6 out of 43, or 14%, were gay, a quadrupling over the base rate. Paternal influence, either genetic or environmental, is inconsistent with Turner's and Hamer et al.'s arguments; whether it contradicts maternal transmission or merely indicates a different subsample of male homosexuals with different modes of development (whether genetic or via modeling, identification or other processes) is unclear.

Brain differences. We will discuss possible brain differences between homosexuals and heterosexuals as a separate category of etiological possibilities, even though the putative brain differences could be conceptualized as a byproduct of developmental biological mechanisms such as genetic factors or prenatal hormone influences, or by post hoc biological factors such as disease processes, or by nonbiological (or rather secondarily and derivatively biological) influences such as adult behavior patterns, or by any combination of these in interaction.

Some studies infer indirectly that the brains and/or neurohormonal systems of homosexuals are different from their heterosexual peers based on neuropsychological performance studies. For instance, research suggests that male and female homosexuals are less right-handed than heterosexuals.[43] This apparent shift is statistically significant but surprisingly modest on an absolute basis, and the correlation with sexual orientation may be spurious. There is also some evidence of differing mental abilities compared to heterosexuals which may be based on different brain structures (specifically, in some studies and on some tasks, male homosexuals perform in a manner unlike male heterosexuals and in a manner not statistically different from females or intermediate between heterosexual males and females).[44] Finally there have been suggestions of cerebral lateralization differences.[45] These findings can only be termed suggestive at this time, and are only indirect indicators of brain differences.

Researchers recently have been exploring the possibility that the physical structures of the brains and/or neurohormonal systems of male homosexuals are different from their heterosexual peers, being significantly "feminized," or more accurately "not defeminized."[46] The first direct evidence of such possible differences emerged

from studies which examined hormonal responses to injection with estrogen. The initial finding of a gender differentiated response to estrogen injection occurred in studies of rodents. Initial human studies suggested the hormonal response of male homosexuals to be intermediate between those of normal women and heterosexual men, which was suggestive of less defeminized brain structure for male homosexuals. These results are in dispute, first, because the occurrence of this response, which is well-established for rodents, may be weak and/or secondary to other factors such as stress in humans, may be irrelevant to the development of sexual orientation for humans, and because there have been several major failures to replicate the original findings with humans.[47]

Most recently, attention has moved to direct dissection studies of brain differences between women and hetero- and homosexual men. Seven areas of the brain have been studied for gender differences (gender dimorphism) and/or sexual orientation differences (orientation dimorphism). Findings in this area often conflict or await replication, as summarized in Table 2.[48]

Study[49]	**Brain Region**[50]						
	INAH1	**INAH2**	**INAH3**	**INAH4**	**SCN**	**SDNH**	**MPAC**
Swaab & Fliers(1985)	HetM > HetF						
Allen, et al. (1989)	HetM = HetF	HetM > HetF	HetM > HetF	HetM = HetF			
LeVay (1991)	HetM = HetF	HetM = HetF	HetM > (HetF & HomM)	HetM = HetF			
Swaab & Hofman (1988)						HetM = HomM; (HetM & HomM) > HetF	
Swaab & Hofman (1990)					HomM >HetM; HetM = HetF; shape spherical in HetM; elongated in HomM and HetF		
Allen & Gorski (1991)							HetF > HetM
Allen & Gorski (1992)							(HetF = HomM) > HetM
Demeter et al. (1988)							HetF < HetM

Table 2: Brain Differences Between Heterosexual And Homosexual Men and Heterosexual Women

Findings on the midsagittal plane of the anterior commissure are conflicting. Two findings of orientation dimorphism await replication and have not as yet been contradicted by other findings. Swaab and Hofman (1990) found that the suprachiasmic nucleus (SCN) of homosexual men was larger in volume and number of neurons than that of heterosexual men, though this is an area of the brain which does not differ by gender in volume or cell count[51] and which appears to have no direct bearing on sexual behavior at all,[52] which obscures the meaning of the finding. The shape of this anatomical feature, as noted in the chart, did suggest parallels between females and homosexual males. The second and most widely publicized finding, that of LeVay, that the third interstitial nuclei of the anterior hypothalamus (INAH 3) of homosexual males is on average structurally more like that of heterosexual females than heterosexual males, is problematic for a number of reasons. "First, his [LeVay's] work has not been replicated, and human neuroanatomical studies of this kind have a very poor track record for reproducibility."[53] Second, the samples used by LeVay were of uncertain definition. Individuals were presumed heterosexual on the basis of no mention of sexual orientation in their medical charts; the death of a significant number of the heterosexual subjects from HIV-infection so early in the AIDS crisis makes their identification as heterosexuals suspect. Third, all of his homosexual population died of AIDS, and the disease processes may have produced the noted anatomical anomalies; there is research suggesting that end-stage AIDS suppresses testosterone levels which may influence the structure of INAH 3.[54]

Assuming the replication of either of these findings (or even of future unanticipated findings), the direction of causation may be difficult to establish, as behavior both effects and is effected by brain structure and function. For example, the gay men in LeVay's study may have had smaller INAH 3 areas because of years of action peculiar to a male homosexual lifestyle, rather than the structure of the INAH 3 causing them to be homosexual. Contrary to popularly shared myths, the brain is structurally plastic throughout life, and the size and nature of different brain structures are influenced by behavior.[55] The conclusion of an eminent research scientist, John Bancroft,[56] regarding the current state of brain research on sexual orientation seems reasonable: "The reader is entitled to be sceptical if not confused by these findings. There is either a lack of consistency or replication. There are methodological

problems. . . . It certainly seems unlikely that there is any direct relationship between structure of a specific area of the brain and sexual orientation per se."

Prenatal hormonal factors. Some researchers propose that human sexual orientation is largely determined between the second and fifth month of pregnancy by fetal exposure to the principal sex hormones. Again, hypothesized genetic and prenatal hormonal causal processes may be independent and mutually exclusive or interdependent and complementary. Several studies have administered abnormal sex hormone levels to animal fetuses in their mothers' wombs to study the effects this has on sexual differentiation and the development of sexual behavior patterns in the adult animals. It has been shown that the abnormal doses of sex hormones administered to an animal fetus at a critical developmental juncture can result in that adult animal showing inverted sexual behavior in conjunction with mating. These effects are complex and multifaceted, and have been taken by some as evidence suggesting that similar hormone variations must be causal factors in human homosexuality,[57] while others have argued that there are monumental problems in establishing the relevance of this animal research for human beings including the highly abnormal hormone levels used to create these inversions and the vast differences between animal and human sexual behavior.[58] For example, what is called "homosexual behavior" in rats -- such as lordosis in males (elevation of the rump to facilitate being mounted from behind as characterizes normal females) or mounting by females (which is characteristic behavior of the male) -- occurs as a reflex which the experimental rats emit upon sexual stimulation without reference to the gender origin of the sexual stimulus;[59] a behavioral reflex which can be "mechanically" elicited is a poor analog for human sexual orientation. Such rats do not have a "homosexual orientation" as understood for humans.

Four major types of evidence grounded in research for the prenatal hormone hypothesis in humans are cited. Some theorists argue on the basis of these evidences that homosexuality is biologically determined, while others disagree.[60] First, both the direct and indirect evidences of male homosexual brains being less defeminized than their heterosexual peers (as discussed in the previous section) are cited as evidence in favor of the prenatal hormonal hypothesis, as early hormonal exposure may be the prime mechanism producing gender-based and/or orientation-based brain differences. The lack of well-established findings in this area, as well as the speculative nature of connections to prenatal hormonal causation, are problematic.

A second type of evidence comes from "quasi-experiments" on prenatal hormonal levels. Although experiments directly manipulating hormones in the womb cannot be ethically performed with human fetuses, a number of naturally and accidentally occurring medical conditions have served as quasi-experiments of sorts. Studies of these unfortunate occurrences have shown that some human fetuses exposed to abnormal hormone levels during development can show altered physical development, brain functioning, gender orientation, and sexual behavior when mature.[61] For example, one recent study[62] reported that the female children of mothers who unwittingly exposed their fetuses to elevated intrauterine levels of estrogen by taking a synthetic estrogen drug during pregnancy were disproportionately (though modestly) more likely to become bisexuals (or much less likely, lesbians) as adults. Application of these studies in understanding homosexuality is questionable. First, the homosexual population shows no elevated rates of the expected physical abnormalities which often occur with prenatal hormonal aberrations. Second, the fact that certain outcomes can be created by one set of abnormal conditions (such as synthetic hormone administration) by no means leads to the conclusion that "naturally occurring" instances of the same outcome occurred because of the same abnormal conditions; the fact that delirium can be induced by striking another's head hard with a bible hardly implicates such as a standard cause of delirium. Finally, few of the unfortunate subjects in these "quasi-experiments" manifest "pure" homosexual identity in isolation from other broad disruptions of gender identity and behavior.

A third type of human evidence for prenatal causation is that which suggests that the most powerful predictor of adult male homosexuality is striking gender non-conformity or gender inappropriateness early in childhood. Boys who are strikingly effeminate as young children appear to be much more likely to become homosexual men than their more typically masculine peers, though some effeminate children do not grow up homosexual and many homosexuals do not report gender-inappropriate behavior as children.[63] This research has been criticized on the basis first that it "repathologizes" homosexuality by returning it to the status of a "deviation" from the normal path of development (as evidenced by some of the researchers in this area using such terms as "atypicality," "disorder," and "abnormality" in discussing homosexuality); second, that it restigmatizes male homosexuals as effeminate, not true men, or "sissies," an image gays have been trying to shed for years;[64] and finally, that this research may be founded upon outmoded and caricatured understandings of gender behavior.[65] There is no conclusive understanding of why early gender behavior distortion occurs. Although some regard it as

evidence for the prenatal hormone hypothesis, there is some evidence that the causes could be psychological. One study reported that "significantly fewer male role models were found in the family backgrounds of the severely gender-disturbed boys," and that there were more emotional problems in the families of the most disturbed boys.[66]

Finally, some researchers argue that maternal stress during pregnancy may predispose the child to homosexuality. The most frequently cited studies are those of Dorner showing that an unusual number of homosexuals were born to German women who were pregnant during World War II.[67] Similarly, a recent study of birth order found that homosexual men are likely to have an overabundance of brothers compared to sisters (by a ratio of approximately 1.5 to 1) and to have been later in the birth order of their mothers;[68] the authors suggest that the mothers of homosexuals are more likely to have been stressed, which tends to result in a higher incidence of male births and which is hypothesized to create androgen insufficiency which would produce incomplete masculinization of the male fetus, resulting in adulthood in homosexuality. Such indirect evidence, while compatible with the prenatal hormone hypothesis, could also be seen as compatible with other psychological theories of causation, as noted by the authors.

Adult (postnatal) hormonal factors. There is a long research tradition of investigating the possibility that male and female homosexuals have abnormal levels of certain sex hormones compared to normals. The consensus from research on males is that there are no substantial hormonal differences between homosexuals and their comparable heterosexual peers. Research which was once thought to show such differences in males has been shown to be plagued by inaccurate methods of measuring hormones and inaccurate ways of categorizing the sexual preferences of subjects in the studies.[69] The results of some studies of lesbians suggest that while most lesbians fall within the normal ranges of serum testosterone and estrogen, a subpopulation may be characterized by elevated testosterone levels. These findings could, however, be an artifact of sample selection, stress, occupational affiliation or physical exercise patterns.[70] In any case, the general consensus is that “it is unlikely that sex hormone levels have any direct bearing on sexual orientation in adults.”[71]

Psychological causation. Though much of the research on psychological causation is based upon the clinical impressions of practicing psychoanalysts (and hence dismissed by some as contaminated by the "heterosexist biases" of the therapists), there has been a considerable amount of prospective or retrospective research on families that produce homosexual sons, though that research has fallen into disfavor and has largely

disappeared from formal academic publication.[72] The bulk of the empirical research on the families of homosexuals documents patterns that would be predicted by psychoanalytic theory, such as patterns of distant relationships with the same gender parent or elevated incidences of same-sex play or abuse in childhood and adolescence. For example, one study reported a much higher loss of fathers and/or mothers to death or divorce in their homosexual sample than in the heterosexual sample.[73] Another reported that among those who had been sexually molested as children, 7.4% were gay men and 3.1% were lesbian women as adults, compared to those who had not been sexually molested as children, among whom 2.0% were gay men and 0.8% were lesbian women as adults; abuse appears to encourage homosexual adaptation.[74]

Many of the findings in this literature cannot be argued to support only a psychological theory. For example, proponents of the prenatal hormone hypothesis [75] would argue that all of the research documenting problematic relations between prehomosexual boys and their fathers, rather than proving that rejecting fathers cause homosexuality, instead reflects the tendency for fathers to reject their gender-inappropriate sons, with said gender-inappropriateness being the cause rather than effect of father bonding difficulties. But the reverse may be true as well; some of the indirect evidence for more biological causation could be taken as supportive of psychological hypotheses as well.

Several studies have produced evidence which has been taken as falsifying the notion of psychological causation. For example, the famous Bell, Weinberg, and Hammersmith study[76] is often cited as the definitive study refuting the psychoanalytic hypothesis. This study was the result of substantive interviews with approximately 1,500 homosexual persons in the San Francisco area in the late 1970s, but there are problems with using such a survey to refute the psychological causation hypothesis. All survey or interview research is subject to the phenomenon of adult reinterpretation of the past; as Risman and Schwartz[77] note, "Once an adult sexual orientation is adopted, gay men may reinterpret their childhood in light of current choices," and indeed not just in light of choices but in light of the most culturally prevalent theories of the origin of homosexuality. This is a problem for all retrospective studies of the families of homosexuals. Also, the psychoanalytic paradigm itself, with its emphasis upon repression and the other defense mechanisms, would not deem retrospective surveys about childrearing practices in the family of origin as a particularly useful method by which to test its hypotheses, any more than

addiction disease theorists would deem a survey about denial conducted with active alcoholics to be a good test of the denial phenomenon.

It seems that there is not enough evidence to prove the psychological causation hypothesis, but there is too much evidence to dismiss it at this time. At least some of the incentive to dismiss psychological causation at this time is based on renewed enthusiasm for biological explanations. Also, much of this literature is dismissed on the presumption that all of the "old" research was done on biased samples, such as those drawn from homosexuals under psychiatric treatment. The irony in this complaint, as we have already noted, is that there has never been a study of a representative sample of homosexual persons. Though the substantive body of psychological causation research is aging and not being regularly renewed, it has never been refuted and still holds promise for understanding part of the causal puzzle of homosexuality.

Conclusions regarding causation. The genetic, brain structure, and prenatal hormonal causation hypotheses are "hot" right now. Although there is an impressive amount of research cited in favor of the former three hypotheses, the direct research in support of each of them is not conclusive. On the other hand, there is a substantive legacy of research on psychological/familial factors which is being generally ignored today despite the statistically significant findings represented in that literature. It is worth noting that the recent movement toward biologic theories may be as much a product of the contemporary *zeitgeist* and of political forces as any real dissatisfaction with the psychosocial theories. In reality, the biologic theories at this point "seem to have no greater explanatory value"[78] than the psychosocial models they seek to displace.

It seems most reasonable to conclude that genetic, brain structure, prenatal hormonal, and psychological/familial factors may each be a facilitating or contributing cause of homosexual orientation in some individuals. We would argue with Bancroft[79] that if homosexual orientation "was determined solely by biological factors, it would be inconceivable that no counterpart could be found in other species" and thus that homosexual orientation must be "a consequence of a multifactorial developmental process in which biological factors play a part, but in which psychosocial factors remain crucially important." Similarly, some of the most respected proponents of biological causation argue that sexual orientation appears to be a "complex characteristic" of "multifactorial or heterogeneous" origin which probably involves "a complex interaction between genetic, biological, experiential and socio-cultural factors."[80] None of these influences can be presumed to be necessarily operative in all homosexuals,

and there is no evidence that any one factor can by itself "cause" homosexuality. The complex of factors which results in the orientation toward homosexuality probably differs from person to person. Thoughtful persons reflecting on the causation literature must strive to rid themselves of the simplistic thinking that demands a single cause for this complex phenomenon. Some of these influencing factors may be genetic in origin, but genetic influence may not mean a "sexual orientation gene;" rather, other higher-order traits may dispose some children to atypical social relationships, patterns of psychological identification, and so forth. Like Byne and Parsons (1993), we favor an interactional hypothesis for the formation of sexual orientation, one which suggests shifting ratios of influence from different sources for different persons, and with nature and nurture in constant interaction.

Does the presence of possibly powerful causative influences, be they biological or psychological/familial, render human choice utterly irrelevant to the development of sexual orientation? There appear to be a variety of factors which can provide a push in the direction of homosexuality for some persons, but there is no evidence that this "push" renders human choice utterly irrelevant. We again concur with Byne and Parsons, who argue that human choice can be construed to be one of the factors influencing the development of sexual orientation, but that 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,"[81] and, we would add, in the context of considerable predispositions toward certain types of preferences. And perhaps, as Baumrind[82] and others have argued, for some people erotic proclivities really are their chosen sexual preference, as adult converts to lesbianism seem to exemplify. Many are most troubled by the concept of "choice" when it is linked to "genetic causation." We are used to thinking of genes as causing us to have things like brown eyes or wavy hair, and choice has little to do with such phenomena. But behavior genetics has produced abundant evidence of genetic influences that clearly do not render human choice irrelevant. One study of the correlations between the television viewing of adopted children and their adoptive and biological parents produced evidence of "significant genetic influence on individual differences in children's television viewing."[83] Their findings had nearly the statistical power of the heritability studies of Bailey and his colleagues reviewed earlier. This finding helps to put the genetic evidence into perspective; all of us would reject the notion that our genes make us sit for a certain number of hours in front of a television screen, but we may have a

predisposition of some sort (sedentary tendencies?) which would make the choice to view television appealing to varying degrees.

Finally, we would note that a recent highly publicized study illustrates the complex inter-relationship between biological and environmental (nature and nurture) factors. Zhang and Odenwald[84] published a study which created genetic alterations in fruit flies which produced "homosexual behavior" in the altered fruit flies. Many tabloid headlines heralded the creation of a "homosexual gene." But curiously, when genetically normal or "straight" fruit flies were introduced into the habitat of the "gay" flies, they began engaging in the same type of "homosexual" behavior as the genetically altered flies. Thus, in a most biological experiment, evidence of environmental ("psychological") influence emerged once again. Clearly, Baumrind[85] is correct in arguing that "it is impossible to disentangle the biological and the psychological contributions to the behavioral differences that constitute sexual orientation."

Is homosexuality a psychopathology?

The view that homosexuality is not psychopathological, and in fact is judged to be a healthy lifestyle variant, is frequently cited in the mainline denominational literature, with implications drawn for the ethical argument. For example, one study stated, "If it could be shown that homosexuality is generally a symptom of unmet emotional needs or difficulties in social adjustment, then this might point to problems in relating to God and other persons. But if that cannot generally be shown, homosexuality may be compatible with life in grace." The report went on to conclude that "The scientific evidence is sufficient to support the contention that homosexuality is not pathological or otherwise an inversion, developmental failure, or deviant form of life as such, but is rather a human variant, one that can be healthy and whole."[86] What does the evidence actually show?

There is much less new research in this category than for the previous two sections. In our earlier analysis,[87] we argued that the decision by the American Psychiatric Association to remove homosexuality as a pathological psychiatric condition per se from its Diagnostic and Statistical Manual (DSM) in 1974 was as much a sociopolitical action as a scientific one. We suggested that the removal of homosexual orientation from the DSM does not answer definitively the thorny question of the psychopathological status of homosexual behavior, nor does it constitute an endorsement of homosexual orientation or lifestyle as necessarily healthy of wholesome. We summarized some of the available evidence relating to the four standard empirical criteria used to define behavior

patterns as abnormal: statistical infrequency, personal distress, maladaptiveness, and deviation from social norms. Regarding the first and last of these four criteria, since our earlier review, estimates of prevalence have more clearly suggested that homosexuality is less statistically frequent than would have been estimated in the 1980s,[88] and surveys of public opinion have continued to show for over two decades that almost 80% of the general public have continued to view all instances of homosexual behavior as immoral, even while support for equal civil rights for homosexuals has grown.[89] But we would like to more fully supplement our earlier analysis of the second and third factors, personal distress and maladaptiveness.

Personal distress. Personal distress is a common but not strictly necessary aspect of psychopathology. Most people with a diagnosable condition are subjectively distressed by their problems, but the notable examples of antisocial personality disorder and alcoholism/drug addiction, with their attendant patterns of denial and minimization of distress, serve as reminders that personal distress is not always present. Reflection on this topic is also complicated by the legitimate concern that distress in a homosexual population may be exogenous (the result of persecution, rejection, lack of acceptance, and so forth) rather than endogenous.

It is often asserted in the sexuality literature that "there is no evidence of higher rates of emotional instability or psychiatric illness among heterosexuals than among homosexuals."[90] While this has been repeated so often as to assume the status of a truth that "everybody knows," the factual basis for this assertion is questionable. The two most frequently cited studies in support of this platitude merit closer examination. The first major study to challenge the prevailing view of homosexuality as intrinsically pathological was the study by Hooker,[91] who conducted psychological tests on a group of "healthy" homosexuals and a comparison group of heterosexuals, and then (to the surprise of the mental health establishment) demonstrated that skilled psychological diagnosticians could not distinguish the heterosexuals from the homosexuals on the basis of their test results. By their test findings, the homosexual sample was judged to have no different and no worse problems than heterosexuals. Given that the prevailing wisdom was that to be homosexual was necessarily to be manifestly, obviously and deeply disturbed, Hooker's study was an apt refutation of that prevailing wisdom; she decisively refuted the absolute judgment that all homosexuals are manifestly disturbed. The study was the logical equivalent of refuting the judgment that "all women are intellectually inferior to men" by demonstrating that a select sample of intellectually gifted women outperformed a sample of men on an intelligence test.

But Hooker's study is often interpreted as having accomplished much more, not to have merely demonstrated that "it is not the case that all homosexuals are manifestly disturbed," but rather she is taken to have proven that "homosexuals are as emotionally healthy as heterosexuals" or to have proven that "homosexuality per se is not psychopathological." Logically and methodologically, her study accomplished neither of these ends. As a necessary but not sufficient condition for assessing either of these latter claims, Hooker would have had to study a representative sample of homosexual persons; but in Hooker's own words, "It should also be stated at the outset that no assumptions are made about the random selection of either group [homosexual or heterosexual]. No one knows what a random sample of the homosexual population would be like; and even if one knew, it would be extremely difficult, if not impossible, to obtain one."[92] Hooker worked explicitly with homophilic organizations in the mid-1950s (such as the Mattachine Society) to recruit a sample of well-adjusted homosexual persons. Further, she explicitly required of participants that they not be under psychiatric or psychological treatment (it is not clear from her report if her recruits were required to not be in therapy at the time of recruitment for the study or to never have been in therapy); this insistence on a non-therapy sample may have made Hooker's sample extraordinarily nonrepresentative of homosexuals.[93] It must be emphasized that a nonrepresentative sample is only a challenge if certain types of claims are being investigated; Hooker did empirically refute the claim that all homosexuals are manifestly disturbed in a similar way that one "conversion" of a homosexual to heterosexuality refutes the absolute claim that homosexuality is "immutable." We may conclude that the Hooker study proved that a select sample of homosexuals were no more distressed than and could not be psychometrically distinguished from a heterosexual sample, but because of the nonrepresentativeness of her sample, she did not in fact prove the conclusion which Masters et al. infer from her work.

Similarly, the sample in the famous Saghir and Robins[94] study was selected to minimize or exclude psychopathology. The authors noted that their subjects were recruited from "homophile organizations," and after volunteering, subjects were further screened and excluded on the basis of prior psychiatric hospitalization. Interestingly, 14% of the male homosexual sample and 7% of the female homosexual sample had to be excluded because of prior psychiatric hospitalization, while no subjects recruited in the heterosexual control sample had to be excluded on that basis.[95] While Saghir and Robins concluded that the homosexual population generally experiences no increased incidence of psychopathology, their study must be interpreted within the context of their having

screened out previously hospitalized individuals which, if included, would suggest a hospitalization rate for homosexuals much higher than the general population,[96] which in turn would suggest a conclusion which is the opposite of that stated. Like Hooker, these authors seem to have proved that a select sample of homosexual persons can be obtained which does not differ from a heterosexual sample in terms of distress.

Contemporary research continues to suggest higher levels of distress in the homosexual population even if that conclusion is usually not stated. For example, a recent study reported on the "National Lesbian Health Care Survey."[97] The authors minimized differences between homo sexual and heterosexual women, arguing that the two groups were comparable except for elevated use of alcohol and drugs[98] and elevated utilization of counseling (77.5% of the lesbian sample) for lesbians. But their empirical results suggest differently. Bradford et al. reported that 37% of the lesbians surveyed had experienced serious depression in their lifetime, 11% were experiencing depression at the time of the survey, and 11% were currently in treatment for that depression; in contrast, the best estimates for the general female population are 10.2% lifetime incidence of major depression, 3.1% current major depression, and probably less than 1% obtaining treatment for that depression in the year before the survey.[99] Bradford et al. further reported that 57% of the lesbians surveyed had experienced thoughts about suicide in their lifetime, and that 18% had attempted suicide at least once; the best estimates for the general population are that 33% of women report lifetime "death thoughts" (a category much milder than thoughts about suicide, as it included answering yes to having "thought a lot about death" at any point in life), while the frequency of suicide attempts was so infrequent that it was not reported.[100]

Maladaptiveness. In our prior analysis, we argued that maladaptiveness can only be judged against some implicit or explicit standard, some tacit model of wholeness and health, a vision of what constitutes a "good life." Any standard of "adaptiveness" can be challenged; neither vocational success nor income nor relational stability nor even the absence of self-injurious behavior is really an utterly reliable or indubitable standard of adaptiveness. Elevated rates of depression, substance abuse and suicide challenge the adaptiveness of homosexuality. Alternatively, the educational and vocational success, not to mention the recent political ascendancy of gay and lesbian concerns, might be seen as supporting the adaptiveness of this orientation.

Many Christian ethicists, including some who are gay-affirming, express a concern for monogamy (traditionalists for heterosexual monogamy, others for monogamy regardless of the genders of partners). This ethical

concern would lead us to examine adaptiveness in terms of relational stability, though this concern might be seen as peculiarly or parochially Christian. In our earlier report we cited evidence of relational instability and sexual promiscuity among homosexuals. As mentioned in the previous section, this reality may (or may not) be a byproduct of societal rejection and failure to provide support social structures (such as marriage) to gay peoples. Nevertheless, research continues to suggest contrasts between homosexuals and heterosexuals, though those contrasts are not as stark as a decade ago. We will just mention a sampling of studies which we either did not cite in our prior report or which have appeared since that time. In contrast to the huge lifetime sexual partner estimates of the famous Bell and Weinberg study, Laumann et al.[101] found that on average gay men reported 42.8 lifetime sexual partners compared to 16.5 for heterosexual men; lesbians reported almost exactly twice as many partners as the average heterosexual woman (9.4 and 4.6, respectively). Several studies have examined the issue of sexual exclusivity in homosexual partnerships. A study[102] of 156 stable, committed male homosexual couples found that none of the over 100 couples which had been together for more than five years had been sexually monogamous or exclusive. The authors argued that for male couples, sexual monogamy is a passing stage of internalized homophobia and that what matters for male couples is emotional, not physical, faithfulness. The second study,[103] also of 156 gay couples, reported that the majority of the partners in the couples (62%) had had sexual encounters outside of the relationship in the year before the survey, and that the average number of extrarelational sexual partners for each member of the gay couples in the year before the survey was 7.1. The largest study,[104] of 1000 gay and 800 lesbian couples, found a sexual "nonmonogamy" occurrence rate across the life of the couple relationship of 79% and 19% respectively, and reported that only 36% of gay men and 71% of lesbians value sexual monogamy. If one presupposes that the capacity to form and maintain exclusive monogamous erotic relationships is an essential adaptive capacity, then real difficulties for male homosexuals are suggested by this research. If the psychological community de-emphasizes relational stability among its criteria of adaptiveness or healthy emotional adjustment (or if Christians do, as some would urge[105]), then promiscuity in the male homosexual community does not constitute maladjustment.

Conclusion. The evidence cited above falls far short of a convincing case that homosexuality in itself constitutes a psychopathological condition. The evidence also suggests that one would be on shaky grounds in proclaiming that there is no evidence that homosexuality is anything other than a healthy, normal lifestyle variant.

Is change to heterosexuality impossible for the homosexual?

The issue of change surfaces often in denominational documents. One discussion guide, playing off the idea of homosexuality as a willful perversion, portrayed traditionalists as believing that "The orientation of all homosexual persons can be modified to conform to the heterosexual norm through conversion and healing."[106] It is supposed that if conversion to heterosexuality can be shown to be impossible, the traditionalist case is considerably weakened.

However the orientation toward homosexual preference develops, there is substantial agreement that it is not a preference that can be easily changed by a simple act of the will. A number of authors argue that homosexual orientation is "immutable" or unchangeable[107] despite the fact that every study ever conducted on change from homosexual to heterosexual orientation has claimed some successes, though the reported success rates have never been very high.[108] Interpretations of these findings vary widely. No new original research of real merit has emerged on the issue of change from homosexual orientation, with opinions varying as to whether this is because of the impossibility of obtaining positive results honestly, or the impossibility of funding, conducting and publishing a credible study in the current political environment. Haldeman[109] has furnished the most wide-ranging and negative critique of the "conversion" literature to date, and so we will evaluate his critique. First, Haldeman demeans the conversion literature in an ad hominem fashion in numerous places, describing various studies as "founded on heterosexist bias."[110] Such a criticism is vacuous and historically myopic. Reviewers should assess methods, findings and arguments, not the character or motivations of researchers; knowledge is not advanced when parties point and yell either "homophobe!" or "homophile!"

Second, he implies that the reported "conversions" are fraudulent; that those who are reported to have changed merely told the researcher therapists what they wanted to hear but never really changed at all. The clearest example of this was his dismissal of claims of religious healing of sexual orientation on the basis of anecdotes. There may indeed be substantial incentives for homosexual clients who are failing to experience change to dissimulate, feigning progress in order to disengage from therapy without disappointing or confronting the therapist. Haldeman's charge of fraud is possible. On the other hand, there is no evidence to support Haldeman's charge, and it verges on solipsism. Further, anecdotes lack any normative weight for the evaluation of therapeutic outcomes, negative or positive; if Haldeman finds it important to dismiss anecdotes of change, why should anecdotes of fraud or failure to change be given credence?

Third, he criticizes the conversion literature as naively founded upon essentialist assumptions about sexual orientation, with authors assuming naive and dichotomous views of "gays and straights." As quoted earlier, he states that, "The categories of homosexual, heterosexual, and bisexual, considered by many researchers as fixed and dichotomous, are in reality very fluid for many." This is a very strong and valid argument which deserves careful attention. Nevertheless, in rebuttal of conversion findings he quickly drops back into essentialist assumptions himself, describing homosexuals and bisexuals as "apples and oranges;" clearly, he contradicts himself. He also describes heterosexuals as coming out as "lesbian or gay later in life" but fails to describe similar shifts in the other direction (because of a commitment to immutability?), admitting instead only that homosexual persons might later "engage in heterosexual behavior and relationships for a variety of personal and social reasons."[111] The implication seems to be that "coming out" after a period of heterosexuality is a revelation of one's true sexual identity, while embracing heterosexual behavior after living in the gay lifestyle is a mere change of behavior.

Fourth, Haldeman attributes the putative successes of conversion efforts to the researchers having actually worked with a mixed sample of bisexuals and homosexuals, with resultant alterations in the behavior and perhaps inclinations of the bisexuals, but not the homosexuals. He appears to have a partially valid point here. Much of the conversion literature does indeed presume to diagnose an individual as homosexual on the basis of any homosexual desires or action, so that a bisexual married man with diminished heterosexual satisfaction who engaged in impulsive impersonal homosexual acts might well have been labeled a homosexual in these studies, and then declared a "success" in conversion therapy when extramarital behavior stopped and heterosexual satisfaction improved.[112] We would agree with Haldeman that most of the prior research in this area lacks diagnostic rigor and sophistication. But this criticism must be contextualized by recognizing that broadly accepted, rigorous and sophisticated definitions of what either homosexuality or bisexuality are simply do not exist. Most attempts at such definitions quickly fall into the essentialist trap which Haldeman claims to deplore. Further, Haldeman's criticism presumes that individuals who engage in sexual action with both genders are not "true homosexuals," but this conflicts with Harry's surprising finding that 42% of the gay or bisexual men in his national probability sample were married to women (and it was not just the bisexuals who were married).[113] Finally, it appears dangerously post hoc to examine past research and conclude, based on the a priori that homosexuality is immutable, that therefore any evidence of change must necessarily have occurred in a nonhomosexual (i.e., bisexual) subsample. At issue here is the question of whether

the weight of evidence would lead us to believe that change is possible for some, or that change is actually impossible. The absolute claim that homosexuality is immutable needs only one contrary case (i.e., one case of change) to lead us to conclude that homosexuality is not always immutable.

Fifth, Haldeman criticizes as trivial what might be described as the grafting of heterosexual action over a homosexual orientation without more basic change.[114] He critically quotes another author who described the change process as an "adaptation" which allows the client to function in heterosexual marriage while homosexual fantasies and attractions, and possibly behavior, never go away completely. He calls such change processes a "laboratory for heterosexual behavior, rather than a change of sexual orientation" and criticizes those who are "eager to equate heterosexual competence with orientation change."[115] In this view, any residual of homosexual attraction or action is taken as indication of treatment failure. Again, this criticism must be partially valid, in that mere behavior change (the lesbian wife who endures sexual intercourse with her husband by engaging in homosexual fantasy) falls short of a profound and pervasive change of orientation. But on the other hand, given how deeply rooted and pervasive we must understand sexual orientation to be, surely it is an overly stringent standard of success to demand that psychotherapeutic treatment of any sort must eradicate all vestiges of homosexual attraction and firmly establish heterosexual eroticism to be deemed successful. Such a stringent standard would never pass the test of generalization to other human conditions; we would not declare alcoholism treatment to be a failure on the basis of recurring attraction to or even occasional relapses into alcohol consumption, nor would we declare depression treatment unsuccessful on the basis of treated individuals needing to combat tendencies to re-experience depression. Haldeman's easy dismissal of change reports on the basis of some continuing homosexual attraction, fantasy or even action seems impossibly harsh.

What can be concluded from the change literature? We would not share the optimistic and seemingly universal generalization of some conservative authors that "healing is possible for homosexuals who are motivated to change,"[116] if change is taken to mean complete alteration of sexual orientation to replace homosexual with heterosexual erotic orientation. Even the most optimistic empirically-grounded spokespersons for change by psychological means say that change is most likely when motivation is strong, when there is a history of successful heterosexual functioning, when gender identity issues are not present, and when involvement in actual homosexual practice has been minimal. Change of homosexual orientation may well be impossible for some by any natural means.

Yet the obverse position that homosexuality is immutable seems questionable in light of reports of successful change. In informal ecclesiastical dialogue, reports of change by psychological or supernatural means are frequently dismissed by anecdotes such as "I tried that ministry (therapy) and it was a complete failure; I know lots of people who claimed to change by that means but they are now out in the gay lifestyle!" Yet it is standard in professional circles to recognize that such anecdotes have no power to either establish or discredit the efficacy of a change method. In that light, it is troubling that the many Christian ministries which attempt to provide opportunities for growth and healing for the homosexual person rarely if ever study and report their success rates.[117]

Conclusion

Citations of the scientific findings appear frequently in ecclesiastical debates about the morality of homosexual behavior. Such citations also occur in secular contexts; for example: "Five decades of psychiatric evidence demonstrates that homosexuality is immutable, and nonpathological, and a growing body of more recent evidence implicates biology in the development of sexual orientation. Some would ask: How can one justify discriminating against people on the basis of such a characteristic? And many would answer: One cannot"[118] or, "If being gay or lesbian is genetic, it ceases to be a sin. If sexual orientation, like gender, is immutable, then courts are obliged to protect nonheterosexuals from discrimination."[119]

We would argue based upon our review that the "findings of science" are not as clear as is commonly assumed, and that the logical implications of the findings of science are far less clear than is casually assumed in ecclesiastical study documents. To repeat the essence of an argument we have made before,[120] even if the scientific findings were clear and unequivocal, their relevance to the moral debate would still be less than determinative. The prevalence of a particular behavior pattern has no clear relevance to the moral evaluation of that pattern; patterns which are common or uncommon may be immoral or moral. With regard to etiology of dispositions toward different types of behavior, though it is common to assume that "sin," as a class, can only include those behavioral choices which the person embraces in an utterly voluntary and uncoerced manner (including having no predisposition toward any particular class of acts) while knowing that the action is a violation of the law of God, such logic would force the Church to throw out the possibility of moral evaluation of many conditions other than homosexuality which have also been shown to have contributing causes such as antisocial personality disorder[121] and alcoholism.[122] Further, the Church's moral concern is not fundamentally with homosexual orientation, but with what one does with

it. Only in the case of extreme biological determination at the level of individual acts would moral culpability be seen as obliterated, but homosexual persons are not sub-human robots whose acts are predetermined-- they are moral agents who inherit tendencies from biology and environment, and who share in shaping their character by the responses they make to their life situations. Like all persons, they must ask, "This is what I want to do, but is it what I should do?" The existence of proclivities or predispositions does not obviate the need for moral evaluation of those proclivities.

Ethical and psychological abnormality are not coterminous; there is no necessary overlap between sinfulness and status as a psychopathology. Many conditions which are "sins" are not pathologies (idolatry, pride, lust, fornication), and many conditions which are pathologies are not in themselves sins (anxiety, depression, psychosis). Finally, there is no direct and formal relevance of the efficacy of change methods to the moral or ecclesiastical argument. The fundamental teaching of the Christian tradition with regard to sexual ethics is that God wills chastity in marriage and celibacy outside of marriage; this standard does not require (or promise) change in orientation. It may be that the Church can no more guarantee healing to egodystontic homosexuals than it can guarantee healing to married but sexually dysfunctional heterosexuals or marriage to disconsolate single heterosexuals.

Science will not solve the ethical debate about homosexual behavior for the Church though good science competently understood should inform the ethical deliberations of the Church. Unfortunately, in this area science is often incompletely and inappropriately presented and then invoked for rhetorical rather than substantive purposes. Even if what some proponents of change regard as the most "optimistic" scientific scenario were realized-- that homosexuality was found to be common, utterly unassociated with psychological distress, the orientation clearly and determinately caused by genetic factors, and the orientation itself utterly immutable-- the traditionalist vision of sexual morality would still have to be engaged on ethical and theological grounds, informed but not decided by science.

Notes

[1]Jones, S. L., & Yarhouse, M. A., "A Critique of the Use of Scientific Evidence in the Contemporary Ecclesiastical Debates About Homosexuality," a paper presented to the consultation on "The Bible and Sexual Ethics," Brite Divinity School, Texas Christian University, Fort Worth, Texas, September, 1996. It was in this paper that the material in this present article was first presented, and our chapter in the edited book containing the proceedings of this consultation (D. L. Balch (Ed.), title and publisher to be determined) will contain a condensed version of the material presented here along with an expanded analysis of the uses to which "scientific findings" have been put in ecclesiastical documents.

[2]and which we used in our previous work-- Jones, S. L., & Workman, D., "Homosexuality: The behavioral sciences and the church," Journal of Psychology and Theology, 17 (1989); pp. 213-225.

[3]Laumann, E. O., Gagnon, J. H., Michael, R. T., & Michaels S., The social organization of sexuality (Chicago: University of Chicago Press, 1994).

[4]Haldeman, D. C., "The practice and ethics of sexual orientation conversion therapy," Journal of Consulting and Clinical Psychology, 62 (1994); pp. 221-227; quote p. 222. For more formal presentations of constructivism, see Blumstein, P., & Schwartz, P. "Intimate relationships and the creation of sexuality," in D. P. McWhirter et al. (Eds.), Homosexuality/heterosexuality: Concepts of sexual orientation, (New York: Oxford University Press 1990); Greenberg, D., The Construction of Homosexuality (Chicago: University of Chicago Press, 1988); and Laumann, et al., The social organization.

[5]E.g., Bell, A. P., & Weinberg, M. S., Homosexualities: A study of diversity among men and women (New York: Simon and Schuster, 1978); McWhirter, D. P., & Mattison, A. M., The male couple (New York: Prentice-Hall, 1984).

[6]Presbyterian Church in the United States of America, Keeping body and soul together: Sexuality, spirituality, and social justice, Reports to the 203rd General Assemble, Part I. Stated Clerk of the General Assembly (1991); p. 49.

[7]Kinsey, A., Pomeroy, W., & Martin, C., Sexual behavior in the human male (Philadelphia: W. B. Saunders, 1948).

[8]Reisman, J., & Eichel, E., Kinsey, sex, and fraud: The indoctrination of a people (Lafayette, LA: Huntington House, 1990).

[9]Laumann, et al., The social organization, pp. 283-320.

[10]Harry, J., "A probability sample of gay males," Journal of Homosexuality, 19 (1990); pp. 89-104.

[11]Sell, R. L., Wells, J. A., & Wypij, D. (1995). The prevalence of homosexual behavior and attraction in the United States, the United Kingdom, and France: Results of national population-based samples. Archives of Sexual Behavior, 24, 235-248.

[12]The citations for the specific studies are: Billy, J. O., Tanfer, K., Grady, W. R., & Klepinger, D. H., "The sexual behavior of men in the United States," Family Planning Perspectives, 25 (1993); pp. 52-61; Fay, R., Turner, C., Klassen, A., & Gagnon J., "Prevalence and patterns of same-gender sexual contact among men," Science, 243 (1989); pp. 338-348; Laumann, et al., The social organization; Sell, R. L., Wells, J. A., & Wypij, D., "The prevalence of homosexual behavior and attraction in the United States, the United Kingdom, and France: Results of national population-based samples," Archives of Sexual Behavior, 24 (1995); pp. 235-248; Rogers, S. M., & Turner, C. F., "Male-male sexual contact in the U.S.A.: Findings from five sample surveys, 1970-1990," Journal of Sex Research, 28 (1991); pp. 491-519; Spira et al. cited in Laumann, et al., The social organization; Stall, R., Gagnon, J., Coates, T., Catania, J., & Wiley, J., "Prevalence of men who have sex with men in the United States," in J. Catania (Chairperson), Results from the First National AIDS Behavioral Survey, Symposium presented at the convention of the American Psychological Association, San Francisco, CA (August, 1990); Wellings, K., Field, J., Johnson, A., & Wadsworth, J., Sexual behavior in Britain: The National Survey of Sexual Attitudes and Lifestyles, (New York: Penguin, 1994). Note that the Rogers and Turner study is a composite report of four national probability surveys, and that the Sell et al. study reports separately on U.S., United Kingdom, and French samples.

[13]Harry, "A probability sample," p. 97.

[14]Laumann, et al., The social organization; pp. 306-309

[15]Stall et al., "Prevalence."

[16]Protestant Episcopal Church, "Standing Commission on Human Affairs," Blue Book of the Episcopal Church General Convention (1991); pp. 196-204, quotes pp. 199, 202.

[17]Sedgwick, T., "Christian ethics and human sexuality: Mapping the conversation," In Continuing the dialogues: Sexuality: A divine gift (pp. 1-14). (Task Force on Human Sexuality, Education for Mission and Ministry Unit: The Episcopal Church, New York, NY, 1988); quote p. 11.

[18]For the best recent comprehensive summaries of the research on biological causation (with very different emphases and conclusions), see Byne, W., & Parsons B., "Human sexual orientation: The biologic theories reappraised," Archives of General Psychiatry, 50 (1993); pp. 228-239; Byne, W. "The biological evidence challenged," Scientific American, 270 (1994); pp. 50-55; Green, R., "The immutability of (homo)sexual orientation: Behavioral science implications for a constitutional (legal) analysis," The Journal of Psychiatry and Law, 16 (1988); pp. 537-575; LeVay, S., & Hamer, D., "Evidence for a biological influence in male homosexuality," Scientific American, 270 (1994); pp., 44-49; and Siegelman, M., "Kinsey and others: Empirical input," in L. Diamant (Ed.), Male and female homosexuality: Psychological approaches (pp. 33-80) (Washington: Hemisphere, 1987). For more detailed Christian reactions to this literature, see for example Cole, S. O., "The biological basis of homosexuality: A Christian assessment," Journal of Psychology and Theology, 23 (1995); pp. 89-100.

[19]Kallman, F., "Comparative twin study on the genetic aspects of male homosexuality," Journal of Nervous and Mental Disease, 115 (1952); pp. 137-159.

[20]For a period, it became the trend to deny that any genetic factors were active in the causation of homosexuality at all, leading such commentators as Masters and Johnson to comment throughout the 1980s, and as late as 1992, that "the genetic theory of homosexuality has been generally discarded today," Masters, W., Johnson, V., & Kolodny, R., Human sexuality (4th Ed.) (Glenview, IL: Scott, Foresman, and Co., 1992); quote p. 390.

[21]For example, Green, "Immutability," reviewed prior studies of the frequency of homosexuality in identical and fraternal twins and in near-relatives of homosexuals, and argued for some degree of genetic influence in the development of homosexual orientation in some persons.

[22]Bailey, J. M., & Pillard, R. C. "A genetic study of male sexual orientation," Archives of General Psychiatry, 48 (1991); pp. 1089-1096.

[23]Bailey, J. M., Pillard, R. C., Neale, M. C., & Agyei, Y. "Heritable factors influence sexual orientation in women," Archives of General Psychiatry, 50 (1993); pp. 217-223.

[24]Whitam, F. L., Diamond, M., & Martin, J., "Homosexual orientation in twins: A report on sixty-one pairs and three triplet sets," Archives of Sexual Behavior, 22 (1993); pp. 187-206.

[25]King, M., & McDonald, E. "Homosexuals who are twins: A study of 46 probands," British Journal of Psychiatry, 160 (1992); pp. 407-409.

[26]Parenthetically, King and McDonald, "Homosexuals," also reported a high likelihood of sexual relations occurring between identical twins, which the authors suggest as a possible nongenetic variable which could elevate concordance rates between monozygotic twins.

[27]Eckert, E., Bouchard, T., Bohlen, J., & Heston, L. "Homosexuality in monozygotic twins reared apart," British Journal of Psychiatry, 148 (1986); pp. 421-425.

[28]and in fact became homosexual partners to each other immediately upon rediscovering each other.

[29]with one twin homosexually identified and the other exclusively and happily heterosexual in marriage though he had a homosexual affair from ages 15 to 18.

[30]Interestingly, though, the King and McDonald, "Homosexuals," study used an identical recruitment method and came up with markedly lower concordance rates in their sample.

[31]Hamer, D. H., Hu, S., Magnuson, V. L., Hu, N., & Pattatucci, A. M., "A linkage between DNA markers on the X chromosome and male sexual orientation," Science, 261 (1993); pp. 321-327; quote p. 321.

[32]Hu, S., Pattatucci, A. M., Patterson, C., Li, L., Fulker, D. W., Cherny, S. S., Kruglyak, L., & Hamer, D. H. "Linkage between sexual orientation and chromosome Xq28 in males but not in females," Nature Genetics, 11 (1995); pp. 248-256.

[33]Byne & Parsons, "Human sexual orientation."

[34]Crewdson, J. "Study on 'gay gene' challenged," Chicago Tribune, June 25, 1995, pp. 1, 10, 11.

[35]Hamer et al., "Linkage," p. 322.

[36]Hu et al., "Linkage."

[37]Crewdson, "Study," p. 10.

[38]Hu et al., "Linkage," p. 253

[39]see Byne & Parsons, "Human sexual orientation," for a fine discussion of such an "interactionist" theory.

[40]Hu et al., "Linkage," p. 250; emphasis added.

[41]Turner, W. J., "Homosexuality, type 1: An Xq28 phenomenon," Archives of Sexual Behavior, 24 (1995); pp. 109-134.

[42]Bailey, J. M., Bobrow, D., Wolfe, M., & Mikach, S., "Sexual orientation of adult sons of gay fathers," Developmental Psychology, 31 (1995); pp. 124-129.

[43]Lindesay, J., "Laterality shift in homosexual men." Neuropsychologia, 25 (1987); pp. 965-969; Annett, M., "Comments on Lindesay: Laterality shift in homosexual men," Neuropsychologia, 26 (1988); pp. 341-34; McCormick, C. M., Wittleson, S. F., & Kingstone, E. "Sinistrality in male and female homosexuals: neurobiological implications." Society of Neuroscience Abstracts, 13 (1987); p. 851; McCormick, C. M., Wittleson, S. F., & Kingstone, E. "Left-handedness in homosexual men and women: neuro-endocrine implications." Psychoneuroendocrinology, 15 (1990), pp. 69-76.

[44]see Green, "Immutability," pp. 553-554, for a review of the pre-1987 data which dealt exclusively with males, and more recently Gladue, B. A., Beatty, W. W., Larson, J., & Staton, R. D., "Sexual orientation and spatial ability in men and women," Psychobiology, 18 (1990); pp. 101-108; McCormick, C. M., & Witelson, S. F., "A cognitive profile of homosexual men compared to heterosexual men and women," Psychoneuroendocrinology, 16 (1991); pp. 459-473; and McCormick, C. M., & Witelson, S. F., "Functional cerebral asymmetry and sexual orientation in men and women," Behavioral Neuroscience, 108 (1994); pp. 525-531.

[45]Allen, L., & Gorski, R. A., "Sexual orientation and the size of the anterior commissure in the human brain," Proceedings of the National Academy of Science USA, 89 (1992); pp. 7199-7202; McCormick & Witelson, "A cognitive profile;" McCormick & Witelson, "Functional cerebral asymmetry."

[46]Byne & Parsons, "Human sexual orientation;" p. 230.

[47]see reviews by Green, "Immutability," pp. 549-552; Gladue, B. A., "Hormones in relationship to homosexual/bisexual/heterosexual gender orientation," in J. M. A. Sitsen (Ed.), Handbook of Sexology, Volume VI: The pharmacology and endocrinology of sexual function (New York: Elsevier, 1988, pp. 388-409); Gooren, L. J. G., "An appraisal of endocrine theories of homosexuality and gender dysphoria," in J. M. A. Sitsen (Ed.), Handbook of Sexology, Volume VI: The pharmacology and endocrinology of sexual function (New York: Elsevier, 1988, pp. 410-424). See also the replication failure of Hendricks, S., Graber, B., & Rodriguez-Sierra, J., "Neuroendocrine responses to exogenous estrogen: No difference between heterosexual and homosexual men," Psychoneuroendocrinology, 14 (1989); pp. 177-185; see Byne & Parsons, "Human sexual orientation," for a general discussion of the significance of these findings.

[48] For the best reviews of this area, see Swaab, D. F., Gooren, L. J. G., & Hofman, M. A., "The human hypothalamus in relation to gender and sexual orientation," in D. F. Swaab, M. A. Hofman, M. Mirmiram, R. Ravid, & F. W. van Leeuwen (Eds.), Progress in brain research, Volume 93: The human hypothalamus in health and disease (New York: Elsevier, 1992); or Byne & Parsons, "Human sexual orientation."

[49]Swaab, D., & Fliers, E., "A sexually dimorphic nucleus in the human brain," Science, 228 (1985); pp. 1112--1114; LeVay, S., "A difference in the hypothalamic structure between heterosexual and homosexual men," Science, 253 (1991); pp. 1034-1037; Allen, L., Hines, M., Shryne, J. E., & Gorski, R. A., "Two sexually dimorphic cell groups in the human brain," Journal of Neuroscience, 9 (1989); pp. 497-506; Swaab, D., & Hofman, M., "Sexual differentiation of the human hypothalamus: Ontogeny of the sexually dimorphic nucleus of the preoptic area," Developmental Brain Research, 44 (1988); pp. 314-318; Swaab, D., & Hofman, M.; "An enlarged suprachiasmic nucleus in homosexual men," Brain Research, 537 (1990); pp. 141-148; Allen, L., & Gorski, R. A., "Sexual dimorphism of the anterior commissure and massa intermedia of the human brain," Journal of Comparative Neurology, 312 (1991); pp. 97-104; Allen, L., & Gorski, R. A., "Sexual orientation and the size of the anterior commissure in the human brain," Proceedings of the National Academy of Science USA, 89 (1992); pp. 7199-7202; Demeter, S., Ringo, J. L., & Doty, R. W., "Morphometric analysis of the human corpus callosum and the anterior commissure," Human Neurobiology, 6 (1988); pp. 219-226.

[50]Note: INAH means Interstitial Nucleus of the Anterior Hypothalamus (four different sections of the interstitial nucleus of the anterior hypothalamus have been examined and are here designated by the numbers 1-4); SCN means suprachiasmic nucleus, SDNH means Sexually Dimorphic Nucleus of the Hypothalamus, and MPAC means

midsagittal plane of the anterior commissure. In the body of the Table, < symbolizes "was significantly smaller than" and > symbolizes "was significantly larger than," HetF = Heterosexual Female; HomM = Homosexual Male; HetM = Heterosexual Male. A blank cell means that area of the brain was not examined in the study reported on that row.

[51]as shown by Swaab & Hofman, "An enlarged;" see also Byne, "The biological evidence."

[52]LeVay, "Difference."

[53]Byne, "The biological evidence;" p. 53

[54]Byne & Parsons, "Human sexual orientation," p. 235.

[55] See Breedlove, S. M., "Sexual dimorphism in the vertebrate nervous system," Journal of Neuroscience, 12 (1992), 4133-4142.

[56]Bancroft, J., "Homosexual orientation: The search for a biological basis," British Journal of Psychiatry, 164 (1994); pp. 437-440; quote p. 438.

[57]E.g., Ellis, L., & Ames A., "Neurohormonal functioning and sexual orientation: A theory of homosexuality-heterosexuality," Psychological Bulletin, 101 (1987); pp. 233-258.

[58]Adkins-Regan, E., "Sex hormones and sexual orientation in animals," Psychobiology, 16 (1988); pp. 335-347; Byne & Parsons, "Human sexual orientation;" Feder, H., "Hormones and sexual behavior," Annual Review of Psychology, 35 (1984), pp. 165-200; Ricketts, W., "Biological research on homosexuality: Ansell's cow or Occam's razor?," Journal of Homosexuality, 9 (1984); pp. 65-93.

[59]For example, the male rat exposed to prenatal feminizing hormones will exhibit lordosis in response to the experimenter's hand as easily as to another (normal) male rat.

[60]Ellis & Ames, "Neurohormonal," and Green, "Immutability," argue this most forcefully, while strong critics of this position include DeCecco, J., "Homosexuality's brief recovery: From sickness to health and back again," The Journal of Sex Research, 23 (1987); pp. 106-129; Hoult, T., "Human sexuality in biological perspective: Theoretical and methodological considerations," Journal of Homosexuality, 9 (1984); pp. 137-156; and Ricketts, "Biological research."

[61]see reviews by Byne & Parsons, "Human sexual orientation;" Green, "Immutability;" Gladue, "Hormones in relationship;" and Money, J., "Genetic and chromosomal aspects of homosexual etiology," in J. Marmor (Ed.), Homosexual behavior: A modern reappraisal, (pp. 59-74) (New York: Basic Books, 1980).

[62]Meyer-Bahlburg, H. F. L., Ehrhardt, A. A., Rosen, L. R., Gruuen, R. S., Veridiano, N. P., Vann, F. H., & Neuwalder N. F., "Prenatal estrogens and the development of homosexual orientation," Developmental Psychology, 31 (1995); pp. 12-21.

[63]Bailey, J. M., & Zucker, K. J., "Childhood sex-typed behavior and sexual orientation: A conceptual analysis and quantitative review," Developmental Psychology, 31 (1995); pp. 43-55; Green, R., The "sissy boy" syndrome and the development of homosexuality (New Haven: Yale University Press, 1987); Harry, J., Gay children grown up: Gender culture and gender deviance (New York: Praeger, 1982); Harry, J., "Sexual orientation as destiny," Journal of Homosexuality, 10 (1985); pp. 111-124

[64]DeCecco, "Homosexuality's brief recovery," p. 109

[65]Risman, B., & Schwartz P., "Sociological research on male and female homosexuality," Annual Review of Sociology, 14 (1988); pp. 125-147.

[66]Rekers, G., Mead, S., Rosen, A., & Brigham, S., "Family correlates of male childhood gender disturbance," The Journal of Genetic Psychology, 142 (1983); pp. 31-42; quote p. 31.

[67]discussed in Ellis & Ames, "Neurohormonal," and Green, "Immutability."

[68]Blanchard, R., Zucker, K. J., Bradley, S. J., & Hume, C. S., "Birth order and sibling sex ratio in homosexual male adolescents and probably prehomosexual feminine boys," Developmental Psychology, 31 (1995); pp. 22-30.

[69]Gooren, "An appraisal of endocrine theories;" Byne & Parsons, "Human sexual orientation;" p. 230; Green, "Immutability," pp. 543-545; Ricketts, "Biological research," pp. 71-76.

[70] Gladue, "Hormones in relationship."

[71] Gladue, "Hormones in relationship," p. 393.

[72]see Siegelman, "Kinsey and others," for a comprehensive review of the substantial amount of research in this area.

[73]Saghir, M. T., & Robins, E., Male and female homosexuality: A comprehensive investigation (Baltimore, MD: Williams and Wilkins, 1973); p. 139.

[74]Laumann, et al., The social organization; p. 345; see also Doll, L. S., et al., "Self-reported childhood and adolescent sexual abuse among adult homosexual and bisexual men," Child Abuse and Neglect, 16 (1992); pp. 855-864.

[75]E.g., Bailey & Zucker, "Childhood sex-typed behavior."

[76]Bell, A. P., Weinberg, M. S., & Hammersmith, S., Sexual preference: Its development in men and women (Bloomington, IN: Indiana University Press, 1981).

[77]Risman & Schwartz, "Sociological research," p. 129.

[78]Byne & Parsons, "Human sexual orientation;" p. 236

[79]Bancroft, "Homosexual orientation," p. 439

[80]Hu et al., "Linkage," pp. 252, 252, 248.

[81]Byne & Parsons, "Human sexual orientation;" p. 237.

[82]Baumrind, D., "Commentary on sexual orientation: Research and social policy implications," Developmental Psychology, 31 (1995); pp. 130-136.

[83]Plomin, R., Corley, R., DeFries, J. C., & Fulker, D. W., "Individual differences in television viewing in early childhood: Nature as well as nurture," Psychological Science, 1 (1990); pp. 371-377; quote p. 371

[84]Zhang, Shang-Ding, & Odenwald, W. F., "Misexpression of the white (w) gene triggers male-male courtship in Drosophila," Proceedings of the National Academy of Sciences USA, 92 (1995); pp. 5525-5529.

[85]Baumrind, D., "Commentary," p. 132.

[86]United Methodist Church, Report of the committee to study homosexuality to the general council on ministries of the United Methodist Church (General Council on Ministries, 1991); quotes pp. 13, 27-28.

[87]Jones & Workman, "Homosexuality."

[88]Further, the lifetime incidence estimates of the major psychopathological disorders range from 14.3% for phobias and 13.8% for alcohol abuse and dependence, to 1.6% for panic and 1.5% for schizophrenia, to 0.1% for somatization disorder; see Robins, L. N., Locke, B. Z., & Regier, D. A., "An overview of psychiatric disorders in America," in L. N. Robins & D. A. Regier, (Eds.), Psychiatric disorders in America: The epidemiological catchment area study (pp. 328-366) (New York: Free Press, 1991); p. 343. Thus, homosexuality is not so common as to be eliminated as a possible pathology on frequency bases alone.

[89]Greeley, A., Michael, R., & Smith, T., "Americans and their sexual partners," Society, 27 (1990); pp. 36-42; Laumann, et al., The social organization.

[90]Masters et al., Human Sexuality, p. 394; see also Ross, M., Paulsen, J., & Stalstrom, O., "Homosexuality and mental health," Journal of Homosexuality, 15 (1988); pp. 131-152.

[91]Hooker, E., "The adjustment of the male overt homosexual," Journal of Projective Techniques, 21 (1957); pp. 18-31; also reported in Hooker, E., "The adjustment of the male overt homosexual," in H. M. Ruitenbeek (Ed.), The problem of homosexu ality in modern society (pp. 141-161) (New York: E. P. Dutton, 1963).

[92]Hooker, "Adjustment," 1963, p. 142

[93]We have no idea of the rate of therapy utilization in the late 1950s, but a recent study found that 77.5% of lesbians (vs. 28.9% of heterosexual women) had been in therapy; Bradford, J., Ryan, C., & Rothblum, E. D., "National Lesbian Health Care Survey: Implications for mental health care," Journal of Consulting and Clinical Psychology, 62 (1994); pp. 228-242.

[94]Saghir, M. T., & Robins, E., Male and female homosexuality: A comprehensive investigation, (Baltimore, MD: Williams and Wilkins, 1973).

[95]It could certainly be credibly argued that these hospitalization rates were affected by heterosexist bias in the mental health establishment of that day, or that they were a secondary artifact of the stress of living in a homophobic social context.

[96]The best estimate we can obtain of lifetime psychiatric hospitalization comes from Robins et al., who report a lifetime prevalence of diagnosable mental dis order for women of 30% (p. 333), and report that on an annual basis only 2.4% of those with a diagnosable disorder are psychiatrically hospitalized (p. 341). If we double this estimate of hospitalization to be conservative in our estimate and to compensate for the higher psychiatric hospitalization rates for women, these findings would suggest that no more than 1.5% of the American female population is psychiatrically hospitalized in their lifetime (30% X 5%). This is probably an overestimate because many of the

psychopathologies included in the Robins et al. study (e.g., phobias, generalized anxiety, dysthymia) infrequently result in hospitalization. The hospitalization rate for men is unquestionably lower than that for women, which puts the high rate among male recruits for this study in even more stark contrast. Robins, L. N., Locke, B. Z., & Regier, D. A., "An overview of psychiatric disorders in America," in L. N. Robins & D. A. Regier, (Eds.), Psychiatric disorders in America: The epidemiological catchment area study (pp. 328-366) (New York: Free Press, 1991).

[97]Bradford et al., "National Lesbian."

[98]The elevations were quite serious; Bradford et al., "National Lesbian," reported that 30% of the lesbians surveyed currently abused alcohol more than once a month, 8% abused marijuana more than once a month, and 2% each abused cocaine, tranquilizers or stimulants more than once a month. In contrast, Robins and Regier (1991, Tables 5-1 and 6-4) estimated for the general population that 4.6% of women had abused alcohol in their lifetime and 1% in the last month, while 4.4% reported lifetime abuse of marijuana and less than 1% reported current abuse and abuse of other substances was very infrequent. Robins, L. N., & Regier, D. A. (Eds.), Psychiatric disorders in America: The epidemiological catchment area study. (New York: Free Press, 1991).

[99]Robins & Regier, Psychiatric disorders, Tables 4-3 and 13-5.

[100]ibid., Table 4-7. On suicidality among homosexual persons, see also Erwin, K., "Interpreting the evidence: Competing paradigms and the emergence of lesbian and gay suicide as a "social fact," International Journal of Health Services, 23 (1993); pp. 437-453.

[101]Laumann, et al., The social organization, p. 315

[102]McWhirter, D. P., & Mattison, A. M., The male couple (New York: Prentice-Hall, 1984).

[103]Deenen, A. A., Gijs, L., & van Naerssen, A. X., "Intimacy and sexuality in gay male couples," Archives of Sexual Behavior, 23 (1994); pp. 421-431.

[104]Blumstein & Schwartz, "Intimate relationships;" findings cited on p. 317 and p. 319 (footnote 9).

[105]Williams, R., Just as I am: A practical guide to being out, proud and Christian (New York: HarperPerennial, 1992).

[106]Protestant Episcopal Church, Human sexuality: A Christian perspective: A study course and leader's guide prepared for Province VII, (Province VII's Committee on the Study of Human Sexuality, 1992); p. 63

[107]E.g., Green, "Immutability;" also Burr, C., "Homosexuality and biology," The Atlantic Monthly, (March, 1993); pp. 47-65; Haldeman, "Practice;" Harry, "Sexual orientation as destiny."

[108]For a reasonably complete review of existing "conversion therapy" studies, see Nicolosi, J., Reparative therapy of male homosexuality (New York: Jason Aronson, 1991). Critics are right to note that many of these studies lack methodological rigor and are basically compilations of independent clinical interventions. Reported success rates have hovered in the 33% to 50% range.

[109]Haldeman, "Practice."

[110]Haldeman, "Practice," p. 223.

[111]Haldeman, "Practice," p. 222.

[112] Blumstein & Schwartz, "Intimate relationships," p. 309, make the same point about "diagnosis as homosexual" on the basis of isolated attractions or behaviors, but with an emphasis on such categorizations in the gay community, where homosexual attraction or action must mark one's "true" orientation.

[113]Harry, "A probability sample," p. 97.

[114]see Green, "Immutability," p. 569, for similar criticisms

[115]Haldeman, "Practice," p. 223

[116]Davies, B., & Rentzel, L., Coming out of homosexuality: New freedom for men and women (Downers Grove, IL: InterVarsity, 1994); quote p. 25.

[117]For further discussion of the ethical concerns raised in the treatment of homosexual clients, see Yarhouse, M. A., & Jones, S. L., "The homosexual client," in R. K. Sanders (Ed.)., Ethics and the Christian mental health care professional (Downers' Grove, IL: InterVarsity Press, in press).

[118]Burr, "Homosexuality and biology," p. 65.

[119]Baumrind, "Commentary," p. 135.

[120]Jones & Yarhouse, "A critique;" Jones & Workman, "Homosexuality;" Jones, S., "1993 Addendum," in J. S. Siker (Ed.), Homosexuality in the church: Both sides of the debate (pp. 107-115) (Philadelphia: Westminster/John Knox Press, 1994).

[121]Sutker, P. B., Bugg, F., & West, J. A., "Antisocial personality disorder," in H. Adams & P. Sutker (Eds.), Comprehensive handbook of psychopathology (2nd Edition; pp. 337-369) (New York: Plenum Press, 1993).

[122]Kendler, K., Heath, A., Neale, M., Kessler, R., & Evans L., "A population-based twin study of alcoholism in women," Journal of the American Medical Association, 268 (1992); pp. 1877-1882.