
Interview: The Biological Evidence for Homosexuality Reappraised

William Byne, MD, PhD*

Abstract

Because of recent media reports and public interest in the role of biological and genetic factors in the development of homosexuality, the editor decided some information regarding this topic might be useful for AMCAP members. Dr. William Byne has recently published several professional articles in which he has criticized the biological and genetic evidence relevant to the etiology of homosexuality. Dr. Byne graciously consented to be interviewed for the *AMCAP Journal*.

Editor: Dr. Byne, recently you and Dr. Bruce Parsons published an article in the *Archives of General Psychiatry* entitled, "Human Sexual Orientation: The Biologic Theories Reappraised." In this article, you were critical of the research which has been advanced to date as "proof" that homosexuality is caused by biological or genetic factors. Could you briefly summarize for us the major conclusions of your article?

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Dr. Byne: The current appeal of biological explanations for sexual orientation results more from dissatisfaction with the present status of psychosocial explanations than from the strength of the biological evidence itself. In our review we subject to critical appraisal the recent genetic, hormonal and neuroanatomical evidence pertaining to sexual orientation and conclude that it is far from compelling. For example, until Dean Hamer's recent linkage analysis studies, the genetic evidence consisted only of reports that homosexuality tends to run in families and that identical twins are more likely to share the same sexual orientation than are fraternal twins. Such studies are absolutely useless in distinguishing between biological and environmental influences because related individuals share environmental variables as well as genes. Protestantism runs in families too, but no one would suggest it is genetic. In the case of the twin studies, it is plausible that identical twins, by virtue of their identical appearance, are treated more similarly and are more similar in their early developmental experiences than are fraternal twins. If so, that alone could account for the increased concordance for homosexuality in the identical twins. In any case, the fact that about 50% of the identical twins in the recent studies were discordant for homosexuality underscores our ignorance of the factors that influence sexual orientation. Those unknown factors could be biological or psychosocial, or both.

The other recent studies—those looking at hormonal responses or brain structure—are, with one exception, premised on the assumption that the brains of homosexuals should exhibit features typical of the opposite biological sex. The problem here is that most of the relevant sex differences have been demonstrated in laboratory rats, not humans. In fact, some of the sex differences alleged to be relevant to sexual orientation in humans are not found in any primate—including humans. If a particular feature of the brain does not differ between men and women, then it is illogical to suggest that the feature should be typical of the opposite sex in homosexuals. It is perplexing that even some of the high profile studies published in prestigious journals, most notably *Science*, failed to cite any studies pertaining to sex differences in the primate brain, but implicitly assumed that rats and humans display

the same sex differences. While some of these studies did report differences between homosexual and heterosexual men, more carefully executed studies have been unable to reproduce their results.

Unfortunately, these negative replication studies tend to go unheeded. For example, 25 studies were required to dispel the notion that homosexuality in men results from insufficient testosterone levels: While 3 studies did find homosexuals to have lower levels than heterosexuals, 2 found homosexuals to have higher levels and 20 found no differences. Similarly, 21 failures of replication have not laid to rest a single study that reported the splenic portion of the brain to be larger in women than in men. And at least 2 groups of highly esteemed researchers have recently predicted that the size of the splenium will be “sex-reversed” in homosexuals. The tenacity with which these researchers hold to their hypothesis in the face of overwhelming evidence against it suggests that it is something more than science that is operating here.

Editor: Dr. Byne, you have been critical of the highly publicized study published by S. LeVay in the prestigious journal, *Science*. Please briefly summarize what you believe are the major flaws in LeVay’s study?

Dr. Byne: My major criticism is actually directed more toward the sensationalistic editorial policies of *Science* than toward LeVay. A major shortcoming of his study is that he did everything single-handedly from collecting the brains and making the measurements to statistically analyzing the results. In this area of research, the traditional standard has been that all measurements be made by more than one investigator prior to publication. Surely, *Science* should have required that a co-investigator verify LeVay’s findings before publishing such a provocative and politically charged study. While LeVay has argued that no one was available to verify his measurements prior to publication, there is no shortage of qualified anatomists who would have been more than willing to have done so. More troubling, however, is that since the publication of his paper, LeVay has refused to allow me or a panel of anatomists to examine his material so that a consensus opinion could be reached

regarding the replicability of his findings. This is in violation of the stated editorial policy of *Science*. "When a paper is accepted for publication in *Science*, it is understood that any materials and methods necessary to verify the conclusion of the experiments reported will be made available to other investigators under appropriate conditions." On November 12, 1992, I wrote to *Science* asking what those "appropriate conditions" would be. To date there has been no response.

Before discussing my major technical criticism of LeVay's study, it is important to know that in some mammals the size of the brain structure comparable to INAH3 in humans (i.e., the structure examined by LeVay) varies with the amount of testosterone in the animal's blood. If a male is castrated, the structure shrinks but if testosterone is given after castration, the shrinkage does not occur. This is crucial to the interpretation of LeVay's study which relied heavily on the brains of men who had died with AIDS. Testosterone levels decrease dramatically as a direct consequence of AIDS itself, and as a consequence of some medications used to treat particular opportunistic infections. Furthermore, there are systematic differences between gay men and intravenous drug users in certain manifestations of AIDS and in their access to and compliance with medical care. The differences in the size of the INAH3 that LeVay attributed to sexual orientation, therefore, may have actually been the result of changes in testosterone levels as a result of AIDS or its treatment. Thus, my major technical criticism of LeVay's study is that his medical histories were not adequate to address this possibility.

LeVay also uses sleight of pen to exaggerate the significance of his findings. For example, he claims that the difference he found is in the region of the brain known to regulate male sex behavior. While INAH3 occupies a tiny portion of the brain region known as the medial preoptic area, the more exact portion of the medial preoptic area involved in male sex behavior is far removed from the INAH3. Thus, LeVay's claim would be analogous to the claim that the Statue of Liberty is in Boston because both the statue and Boston are in northeast region of the U.S.A.

Others have faulted LeVay's study for the small number of brains he studied and for the inadequate sexual histories he had on his subjects. These problems don't particularly trouble me. First of all, the differences he reported were large enough to have been detected with even smaller numbers of brains than he employed. Moreover, inadequate sexual histories would have decreased, rather than increased, the likelihood of detecting statistically significant differences.

Editor: Dr. Byne, in your *Archives of General Psychiatry* paper, you and Dr. Parsons briefly propose an "interactional model" of homosexuality. Could you briefly explain what you mean by an interactional model and why it is more scientifically plausible than exclusively biological or psychosocial models of homosexuality?

Dr. Byne: An interactional model is one in which the effect of one factor is dependent upon other factors in the model. Vocal learning in bullfinches serves as an illustrative example. These birds can only learn their native call during a restricted period of brain development. If they are allowed to hear only the call of another species during that period, they will learn it instead. While the bird's call seems to become hard wired into its brain, it is clearly learned by experience and is not innate. That is, the bird's song is determined by experience (i.e., nurture), whereas biology (i.e., nature) defines the crucial period during which that experience must occur.

I do not mean to imply that sexual orientation in humans is learned by simple mimicry. Instead, it seems reasonable to suggest that the stage for future sexual orientation may be set by experiences during early development, perhaps the first four years of life. This is not only the period during which gender identity is established largely in response to social cues, but also a period of tremendous brain development. In fact, the human brain quadruples in size after birth and the major expansion of its synaptic network occurs during the first two years following birth. Thus, a tremendous amount of brain development occurs at a time when the individual is in constant interaction with the outside world. This maturation is highly relevant to interactional models in light

of studies in laboratory animals showing that learning and environment influence the chemistry and structure of the brain itself.

In our review, we offer a hypothetical interactional model in which biological factors influence temperament rather than sexual orientation *per se*. We then offer some examples of how one's temperament could then bias the emergence of his sexual orientation in a context-dependent manner. This model is interactional because biology influences temperament which, in turn, influences how an individual shapes and is shaped by his environment. Such an interactional model allows for multiple developmental pathways leading to homosexuality and it is consistent with the replicable research suggesting an influence of biological factors on sexual orientation. Moreover, it could explain the failures of various psychosocial theories that have focused on either the personality of the individual or on his familial milieu but not on the interaction of the two.

Editor: In a soon-to-be-published anthology regarding psycho-biological research on homosexuality, your essay discusses some of your experiences with the peer review process and the press as you have attempted to publish your work on homosexuality in scientific journals. You characterized several recent scientific reviews of research on homosexuality as lacking in objectivity and fairness and attributed this to political and social influences. Have I understood your perceptions about this, and if so, could you briefly share with us the experiences you have had with the review process and press that have led you to feel this way?

Dr. Byne: Your perceptions are correct. Even when we strive for scientific objectivity, human nature dictates that we will be more skeptical of studies that fail to conform to our own belief systems. Thus, we will, perhaps unwittingly but nevertheless surely, hold to a higher standard of review studies that contradict our personal views. The field of sexology is small and appears to me to be dominated by a relatively few individuals who share the same biologically deterministic ideology. Because it is nearly impossible to publish in this area without having your paper reviewed by one or more of these individuals, the unfortunate result is that the

biologically deterministic ideology is sometimes protected at the expense of scientific rigor.

Perhaps my worst experience with the peer review process was a three-year delay in publishing a study with Ruth Bleier that failed to confirm an earlier report (published in *Science*) that the splenium is larger in women than in men. One can only wonder why *Science* even published that report since the finding was not even statistically significant. At the time Ruth and I submitted our paper the original report had already become entrenched in the medical literature including authoritative textbooks where it was referenced as “a clear cut sex difference in the anatomy of the human brain” and interpreted as the biological basis for a variety of presumed sex differences in abilities and social roles. The remarks of one of the reviewers of our manuscript are particularly informative: “The present paper uses magnetic resonance imaging to show that there is no significant [sex] difference in the splenium of the corpus callosum. We can assume that the earlier paper is wrong and misleading, and therefore correcting this error has some value to the scientific community. On the other hand, it is hard to argue that a negative finding contradicting a poor paper constitutes an advance in science. . . . My conclusion is that this paper is not appropriate for publication in the *Journal*.” In other words, published studies making unsubstantiated claims—even claims of potential social import—need not be challenged because of the very fact that they were “poorly conceived and poorly executed.” Such an attitude impedes the self-correcting process of the scientific method and thus undermines science at its foundation.

Editor: Since the publication of the *Archives of General Psychiatry* article, some people have accused you of having an “anti-gay” motive or agenda? Would you care to respond to this accusation?

Dr. Byne: Some gay activists believe that society will be more tolerant of homosexuality if sexual orientation can be shown to be innate. Thus, they view any criticism of the recent biological work as anti-gay. In other words, they feel that we should subjugate scientific rigor to political expediency. To support their belief these

activists cite the results of various surveys such as the *New York Times*/CBS News Poll suggesting that people who believe that homosexuality is a chosen lifestyle are less tolerant of homosexuality than are people who believe that homosexuality is immutable. But such polls don't show that belief in a biological etiology causes tolerance. Perhaps, intolerance is what leads to the belief that homosexuality is chosen. Very few who have spoken in depth with homosexuals regarding their orientation would conclude that one simply chooses to have homosexual attractions. Furthermore, it would be naive to merely assume that everything in life that is not chosen is biologically determined. We do not choose our native language. Nor do we simply choose our beliefs. Beliefs are based on our experiences, our character structure, and our cognitive style. For example, we could not simply choose to believe that the earth is flat or that the sun revolves around the earth.

For the record I support gay rights. I simply believe that we as a society must learn to be tolerant of individual differences and not make social tolerance contingent on biological immutability. Furthermore, biologically deterministic theories have been used historically to rationalize discrimination and social intolerance—not to end them. This applies to gays as well as to women and racial minorities. On the basis of presumed biological etiology, gays during this century have been subjected to forced hormone injections, castration, and brain surgery. Of course, gays have also suffered in the hands of psychoanalysts and social theorists. In the absence of social tolerance, any etiological theory is capable of being put into the service of social prejudice.

Editor: Some people seem to believe that if solid evidence is obtained showing that homosexuality is biologically or genetically determined that this would provide support for the notions that (1) human sexual preference cannot be changed, and, (2) homosexuality is a normal variation of human sexual functioning. Do you believe that if evidence were found that homosexuality is biologically or genetically determined that this finding would provide support for the notions above?

Dr. Byne: Your question seems to imply that sexual orientation could be changed if it is not biologically determined. But in the

example I gave of the bullfinch, his song is not biologically determined but once it is learned it is immutable. If sexual orientation were shown to be biologically determined, perhaps that would imply that only a biological intervention could change it. History suggests that unless society becomes tolerant of homosexuality, belief in biological causation is likely to lead to biological interventions aimed at changing it. If homosexuality were proven innate that would suggest that it is a *naturally occurring* variation, but not necessarily *normal*. *Normalcy*, has two connotations. The first simply refers to what is statistically average. The second connotation refers to the range of behaviors or states that a particular society views as desirable or acceptable. Schizophrenia, mental retardation, diabetes, and cancer are biological phenomena. While naturally occurring, they are not statistically average states, nor does society perceive them as desirable. The undisputed biological origin and immutability of skin color have not had a mitigating influence on racism. I see no reason to believe the case would be different for homosexuality.

Editor: We understand that your recent publications have generated considerable public and professional controversy and attention. Would you care to share any of your experiences in this regard?

Dr. Byne: Since I began working in this field as a neurobiologist 15 years ago, I have been periodically accused of searching for the cause of homosexuality so a “cure” could be found. So I was initially quite surprised when the very groups that had accused me of homophobia because of my biological research on animals began to accuse me of homophobia for my criticisms of attempts to apply that animal research to humans. More troubling, however, is that some of the most senior and influential figures in sex research have openly suggested that it is politically incorrect for anyone to criticize the biologically deterministic data pertaining to homosexuality.

I was also surprised that my opinion has been sought by governmental agencies regarding the issue of gays in the military and Colorado’s Amendment 2. The etiology of homosexuality is a totally separate issue from the issue of whether or not homosexual

men and women have the ability to honorably serve their country. They have been doing so for centuries. Suddenly, understanding more (or suddenly realizing that we know very little) about the origins of sexual orientation won't change history.

Editor: What are your plans for future research in this area and why do you plan to pursue these directions?

Dr. Byne: My primary research interest is in brain development and I am currently focusing on how maternal drug abuse disrupts fetal brain development. With regard to sexual orientation research, I am involved in two projects. First, I am trying to replicate the report that INAH3 is larger in the brains of men than in those of women. If I am successful in that regard, I will focus on the development of the sex difference and also attempt to replicate Simon LeVay's report that INAH3 is feminized (i.e. small) in gay men.

Editor: We greatly appreciate your time and willingness to discuss your research, thoughts, and experiences with us. Is there anything else you would like to say before we conclude.

Dr. Byne: Since the publication of our review, Dean Hamer's group at the National Cancer Institute has published their study suggesting a genetic linkage for homosexuality. Of the recent biological studies, that study is conceptually the most complicated and probably the most misunderstood.

I would like to address one of the most common misconceptions regarding its findings. That misconception is illustrated by the following from the August 1993 issue of *Clinical Psychiatry News*: "Science last month published a study that shows a particular genetic sequence on the tip of the long arm of the X chromosome. That sequence is the same in 33 of 40 pairs of gay brothers." That simply is not the case. Hamer's study did not show that 33 of the 40 pairs had anything in common other than sexual orientation. The concordance that he reported was within pairs, not across pairs. Specifically, both members of each concordant pair had received a copy of the same Xq28 region of his mother's X chromosomes. Each of the 33 mothers of the concordant pairs would have had unique genetic sequences in her Xq28 regions.

Because women have two of these regions but can pass a copy of only one on to their sons, one can calculate that the probability of two sons receiving the same Xq28 region from their mother is 50%. Hamer's study merely showed that for his pairs of gay siblings the probability that they had received the copy of the same maternal Xq28 region was significantly higher than the expected value. Thus, the study *suggests* that a particular genetic sequence predisposing to homosexuality might be located in the Xq28 region of the X chromosome—but no such sequence was actually detected in the study.

A problem that some have argued makes Hamer's study uninterpretable is that he did not analyze the Xq28 region of the heterosexual brothers of the gay siblings of the study. This is a problem because if one of a mother's two Xq28 regions contained genes that impaired fetal viability, then there would be an increased probability of all of her living sons, heterosexual and homosexual sharing the same Xq28. We should not merely assume that that is not the case in Hamer's highly selected family pedigrees. We should subject our assumptions to empirical test. Moreover, there was no pressing reason for rushing Hamer's study into print. It's not as if he were reporting a cure for cancer or AIDS. In my opinion, the editorial board at *Science* should have required Hamer to provide empirical evidence to support his assumptions prior to publishing the study.

One good thing about Hamer's study is that it is essentially atheoretical regarding the etiology of homosexuality. Even if he succeeds in finding genes associated with homosexuality, a tremendous amount of work will be required to demonstrate how those genes act. One possibility would be that they do not act on sexual orientation per se but, instead, influence temperament as in the interactional model proposed in my review with Dr. Parsons. The bottom line remains that we still know very little about the factors that influence sexual orientation.