Scientific Research and Moral Argumentation Regarding Homosexuality

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The pop music sensation Lady Gaga (Stefani Angelina Germanotta) released a song in 2011 which has become an anthem for many people in the homosexual community. Titled *Born This Way,* the lyrics articulate a crude sort of biological/genetic justification for various forms of behavior. In many ways the song reflects the spirit of a culture infatuated with a sexually libertine ethic:

> No matter gay, straight or bi
> Lesbian, transgendered life
> I'm on the right track, baby
> I was born to survive
>
> No matter black, white, or beige
> Chola or orient made
> I'm on the right track baby
> I was born to be brave
>
> I'm beautiful in my way
> 'Cause God makes no mistakes
> I'm on the right track, baby
> I was born this way[1]

Germanotta's lyrics reflect a moral reasoning common among many young people including young soldiers. She moves casually between the issues of sexual morality and ethnic heritage, asserting that the two are morally equivalent because each person is "born this way." To add greater strength to the argument, God Himself is credited with being the author of both

[1] Stefani Germanotta, *Born This Way*. http://www.azlyrics.com/lyrics/ladygaga/bornthisway.html. (Accessed January 16, 2013). The term "chola" is apparently used by Germanotta as a reference to people of Mexican or Mexican-American ethnic heritage.

racial distinctions and sexual preferences. The obvious inference is that sexual preference, like race, is hard-wired into who we are from birth. Since these sexual preferences are innate, they must be from God. Therefore, no moral criticism should be leveled at people involved in the homosexual lifestyle. After all, homosexuals are "born this way."

The moral argumentation of *Born This Way* reflects the way scientific research into human sexuality is communicated on a street level. In the last forty years, an intense effort has been underway to establish in the public mind that science confirms a biological/genetic connection to homosexual behavior and thus remove moral stigma associated with homosexual acts. When the popular press reports on scientific research regarding homosexuality, it often does so with headlines trumpeting a genetic or biological cause for homosexual behavior. Sadly, in our day of sound-bite moral reflection, most people have not thought deeply or seriously about the relationship between biology, genetics, and moral accountability. Instead of rigorously examining the topic, it is easier to engage in crude moral argumentation and say, "If there's a genetic component, then people must be born that way. It's not fair to judge people for the way they are born." The assumption is that if any biological or genetic aspect of homosexuality is discovered, then it is prejudicial to form a negative moral opinion about homosexual behavior.

How should Christians respond to the "born this way" moral argument? It is not enough for us to mourn the societal slide towards Gomorrah. We must attempt to interact with, evaluate, and respond to the research associated with homosexuality. As we do so, several questions emerge: To what degree, if any, does biology or genetics contribute to homosexuality? If there is a biological/genetic aspect of homosexual behavior at any level, then should people be held morally accountable for homosexual acts? If a biological/genetic component exists, then how do we reconcile this with the clear denunciations of homosexual behavior in Scripture? In what follows, I hope to offer a brief answer to these and other

questions associated with the relationship between scientific research and the moral status of homosexuality.

A review of the research will show that, while there are some genetic or biological factors that correlate with a higher incidence of same-sex attraction and homosexual behavior, there is no proof of genetic or biological causation for homosexuality. We study biology or chemistry or genetics in order to find out what the world is like independently of what we have said about it, or wish were the case about it, or think might be true about it. We study the natural sciences and research regarding human behavior with a view to discovering the truth.[2] A robust review of research to date indicates the truth about LGBTQ identities and claims they are predetermined and immutable traits like hair or skin color is far different than the popular consensus. Beginning with the work of Alfred Kinsey, I will address issues chronologically, focusing on some of the most significant research produced in relation to homosexuality and brain structure, twin studies, and genetics. I will conclude with several remarks concerning scientific research regarding homosexuality and Christian moral reflection with focus on the Christian doctrines of sin and humanity. Also, since male homosexuality has consistently been found to occur more frequently than female homosexuality, male homosexuality has been researched more extensively. In fact, claims about male homosexuality made by Alfred Kinsey serve as a good starting point for our survey.

I. Alfred Kinsey

During the Twentieth Century, no one individual did more to bring homosexuality into the public forum than Alfred Charles Kinsey (1894 – 1956). A professor at Indiana University, Kinsey was a zoologist by training and spent the early years of his career studying gall wasps, collecting thousands of specimens of the insects. Kinsey then transferred his obsessive and taxonomic approach of research to the study of human sexuality. Much like the gall wasps he collected, Kinsey and his colleagues

[2] Robert P. George and Christopher Tollefsen, *Embryo: A Defense of Human Life* (New York: Doubleday, 2008), 126.

gathered thousands of "interviews" in which he or his researchers asked detailed questions about the sexual backgrounds of research participants. Kinsey compiled the findings from these interviews into two books that were the opening salvos of the sexual revolution that soon swept the United States: *Sexual Behavior in the Human Male* (1948) and *Sexual Behavior in the Human Female* (1953). Both works contain many sweeping assertions and often move quickly from tables full of data to moral speculation about the repressed sexual ethics of America.

Kinsey officially began sexual research in 1941 with the help of funds from the Rockefeller Foundation and the assistance of the National Research Council. In 1947 Kinsey founded the Institute for Sex Research at Indiana University, now simply known as The Kinsey Institute. What has become clearer in the years since the publication of the Kinsey reports is that Kinsey was not merely gathering information about other people's sexual experiences, but he was also engaging in assorted sexual practices with various members of the research team. Instead of the staid atmosphere most people associate with academia, the Institute for Sex Research became a kind of sexual utopia for the gratification of the appetites of Kinsey and his team. According to one biographer, "Kinsey decreed that within the inner circle men could have sex with each other; wives would be swapped freely, and wives too, would be free to embrace whichever sexual partners they liked."[3] Kinsey himself engaged in various forms of heterosexual and homosexual intercourse with members of the institute staff, including filming various sexual acts in the attic of his home. My purpose here is not to engage in *ad hominem* attacks on Kinsey, but to emphasize that Kinsey was not a dispassionate scientist seeking truth; he was an agenda-driven reformer bent on changing the sexual ethics of a nation.

As Kinsey and his colleagues tabulated the data, they used a novel approach to defining human sexuality and employed a graded scale to

[3] James Jones, *Alfred Kinsey: A Public / Private Life* (New York: W. W. Norton, 1997), 83. I've borrowed the phrase "sexual utopia" from Jones.

define a person's sexuality. Prior to Kinsey, people were generally considered to be either heterosexual or homosexual. Instead of this binary approach, Kinsey saw sexual behavior on a continuum which rarely described individuals as either strictly homosexual or heterosexual. The Kinsey Scale is as follows:

0- Exclusively heterosexual with no homosexual

1- Predominantly heterosexual, only incidentally homosexual

2- Predominantly heterosexual, but more than incidentally homosexual

3- Equally heterosexual and homosexual

4- Predominantly homosexual, but more than incidentally heterosexual

5- Predominantly homosexual, but incidentally heterosexual

6- Exclusively homosexual[4]

On the Kinsey scale, six out of the possible seven scores could be interpreted as indicating some level of homosexual attraction. In this way, the Kinsey scale normalizes homosexuality and helped contribute to inflated percentages in some findings. The Kinsey scale has since been widely used in numerous research projects related to sexuality.

When *Sexual Behavior in the Human Male* was released in 1948, it sold thousands of copies. The report asserted that nearly 69% of white males in the United States had sex with prostitutes[5] and also said "it is probably safe to suggest that about half of all married males have intercourse with women other than their wives, at some time while they are married."[6] Most surprising were the claims about the incidence of homosexuality among American men. Kinsey claimed 37% of males had homosexual physical contact to the point of orgasm at least once.[7] Furthermore, he claimed 10% of all males are exclusively homosexual for at least three years between the ages of 16 – 55, and 4% of males are exclusively

[4] The Kinsey scale is found at Kinsey, Wardell Pomeroy, and Clyde Martin, *Sexual Behavior in the Human Male* (Philadelphia: W.B. Saunders Company, 1948), 638. Notice that while a score of "0" is defined as "heterosexual with no homosexual," a score of 6 simply says "homosexual," without a corresponding "with no heterosexual."
[5] Kinsey, *Sexual Behavior in the Human Male*, 597.
[6] Ibid., 585.
[7] Alfred Kinsey, *Sexual Behavior in the Human Male*, 650.

homosexual throughout the entirety of their lives.[8] In *Sexual Behavior in the Human Female,* Kinsey further asserted between 2 – 6% of unmarried females are exclusively homosexual between the ages of twenty and thirty-five.[9] While there was significant criticism of Kinsey's claims, Ronald Bayer notes that for "homosexuals who were just beginning their efforts at organization and the struggle for social acceptance and legal rights, the findings were emboldening."[10]

Reflecting on the public morality of the day, Kinsey suggested American society's moral revulsion to many of the sexual acts he described originated in "ignorance and superstition" and not in "scientific examinations of objectively gathered data."[11] After dismissing traditional morality as superstition, Kinsey then argued, "While this problem [ethics based on superstition] will be met again in other places, the present discussion of frequencies of total sexual outlet provides a good opportunity for understanding the futility of classifying individuals as normal or abnormal, or well-adjusted or poorly adjusted, when in reality they may be nothing more than frequent or rare, or conformists or non-conformists with the socially pretended custom."[12] In this way, Kinsey argues much like other sexually libertine propagandists of the second half of the Twentieth Century: We should no longer look at sexual behavior in the categories of right versus wrong, but instead in the categories of more common versus less common.

A closer look at Kinsey's research reveals many problems with his findings. The most glaring problem with his data is the source of his sample. While the sample for *Sexual Behavior in the Human Male* numbered over 5,000, a disproportionate number came from prison inmates, many of whom were sex offenders.[13] The Kinsey team interviewed some African Americans, but their data was not included in the tabulations. Furthermore, Kinsey over-sampled people recruited via homosexual-friendly organizations or magazines. College students also represented a disproportionate number of his sample. Jones and Yarhouse

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[8] Ibid., 651. Kinsey's data is the source for the widely repeated claim that 10% of all people are homosexual. Even the very well-respected New Testament scholar Richard Hays repeats the claim in *The Moral Vision of the New Testament: A Contemporary Introduction to New Testament Ethics* (New York: HarperSanFranciso, 1996), 397.

[9] Alfred Kinsey, Wardell Pomeroy, Clyde E. Martin, and Paul H. Gebhard, *Sexual Behavior in the Human Female* (Philadelphia: W. B. Saunders Company, 1953), 473 – 474. See also the statistical chart on page 488.

[10] Ronald Bayer, *Homosexuality and American Psychiatry: The Politics of Diagnosis* (Princeton, NJ: Princeton University Press, 1987), 44.

[11] Kinsey, *Sexual Behavior in the Human Male*, 203.

[12] Ibid., 203. For Kinsey, the term "total sexual outlet" meant the number of orgasms an individual had during a particular period regardless of the way the orgasm was achieved.

[13] It is difficult to determine exactly what percentage of Kinsey's sample came from prisoners. He does reference "many hundreds of histories which we have from men who have been confined to penal institutions." Kinsey, *Sexual Behavior in the Human Male*, 210.

rightly critique these problems with Kinsey's sample and say: "This is obviously not the type of methodology a person would implement if he or she were trying to get a representative outlook on the sexual behavior of the general population."[14] In many ways, Kinsey's sample assured he found what he was hoping to find: statistical confirmation of sexually adventurous behavior.

The manner in which Kinsey presents his data is also quite problematic. *Sexual Behavior in the Human Male* in particular often blurs the distinction between the statistical data gathered by the Kinsey researchers about the sexual behavior of white males with supplementary data. By supplementary data, Kinsey meant correspondences in which participants shared day-to-day records of their activities along with their thinking on the various aspects of sex. Apparently, this supplementary data became the source of most of Kinsey's conclusions concerning the sexual ethics and public policy in the mid-Twentieth Century. Kinsey said the supplementary data served as the source for "the psychologic and social concomitants of sexual behavior, particularly in relation to the factors which motivate and control the activities."[15] Writing in 1949, W. Allen Wallis of the University of Chicago criticized Kinsey's failure clearly to distinguish between hard statistical data and the more broad category of supplementary data and said, "Conclusions based on the sociological interpretations or the supplementary data are frequently stated along with those based on the statistical data, and it is frequently difficult to judge what the basis is for a given conclusion."[16]

Much of what Kinsey called "data" was actually vulgar, pornographic material with no morally redeeming value. He went so far as to include graffiti from bathroom walls in his research. Attempting to dignify the unwholesome filth often scrawled in public bathrooms, Kinsey noted, "From the days of ancient Greece and Rome, it has been realized that uninhibited expressions of sexual desires may be found in the anonymous inscriptions scratched in out-of-the-way places by authors who may freely express themselves because they never expect to be identified."[17] According to Kinsey, we should not think of such filth as inappropriate defacing of property; it is actually a venue for the sexually repressed. Furthermore, Kinsey says "such material epitomizes some of the most basic differences between male and female sexual psychology. . . . Since males are more prone to produce such graffiti, we particularly need additional collections of

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[14] Stanton Jones and Mark Yarhouse, *Homosexuality: The Use of Scientific Research in the Church's Moral Debate* (Downers Grove, IL: Intervarsity Press, 2000), 37.
[15] Kinsey, *Sexual Behavior in the Human Male,* 74.
[16] W. Allen Wallis, "Statistics of the Kinsey Report," *Journal of the American Statistical Association* 44 (December 1949): 466.
[17] *Sexual Behavior in the Human Female*, 87.

material originating from females."[18] So, some of Kinsey's conclusions about differences between male and female sexuality were influenced by bathroom graffiti and he was frustrated that he did not have more to add to his research.

Another glaring problem in Kinsey's report is the phenomenon of volunteer bias: Survey participants who volunteered to be questioned about their sexual experience were also more likely to be sexually adventurous and out of the mainstream.[19] Volunteer bias may have been especially prominent considering that most of Kinsey's research was done prior to 1950, an era of much more conservative ethics. Many people simply would not have discussed the intimate details of their sexual life, and those who were willing to do so were more prone to have a sexually libertine ethic. Writing in 1952, Abraham Maslow and James M. Sakoda noted the problem of volunteer bias in Kinsey's research and invited Kinsey and Pomeroy to interview Brooklyn College students. Maslow and Sakoda then compared self-esteem scores for students from Brooklyn College who agreed to volunteer for Kinsey's research versus those who chose not to volunteer for research and found that students who volunteered had a higher mean self-esteem score. Maslow and Sakoda concluded that "bias introduced into a sex study by the use of volunteers is, in general, in the direction of inflating the percentage reporting unconventional or disapproved sexual behavior."[20] Because of his work with Maslow and Sakoda, we know Kinsey was aware of the volunteer-bias problem. He even acknowledged that the people who answered his questions may have been "less inhibited sexually."[21] Just as was seen in the problems with his sample, the problem of "volunteer bias" skewed Kinsey's data toward the conclusions he wanted.

The most disturbing and hotly debated part of Kinsey's research is chapter 5 of *Sexual Behavior in the Human Male* titled, "Early Sexual Growth and Activity." Kinsey gathered data from people who can only rightly be called *child molesters*. Describing the source of some of his data on small children he said, "Better data on pre-adolescent climax come from the histories of adult males who have had sexual contacts with younger boys and who, with their adult backgrounds, are able to recognize and interpret the boys' experiences."[22] Kinsey then goes on to say that

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[18] Ibid.

[19] Bruce Westfall, "Kinsey Report," in *Encyclopedia of Biblical and Christian Ethics*, R.K. Harrison, ed., rev. ed. (Nashville: Thomas Nelson, 1992), 221.

[20] Abraham H. Maslow and James M. Sakoda, "Volunteer-Error In the Kinsey Study," *Journal of Abnormal Psychology* 47.2 (April 1952): 261. Maslow and Sakoda approved of Kinsey's basic procedures, but wanted to refine the techniques used.

[21] Alfred Kinsey, *Sexual Behavior in the Human Male,* 99.

[22] Kinsey, *Sexual Behavior in the Human Male*, 176- 177.

"9 of our adult male subjects have observed such [pre-adolescent] orgasm. Some of these adults are technically trained persons who have kept diaries or other records which have been put at our disposal; and from them we have secured information on 317 pre-adolescents who were either observed in self masturbation, or who were observed in contacts with other boys or older adults."[23] This disturbing description of child molestation is accompanied by a statistical chart that documents the observation of pre-adolescent experiences in orgasm for children between the ages of *5 months and "the age of adolescence*." Later on in the book, Kinsey discusses masturbation and says, "Of course, there are cases of infants under a year of age who have learned the advantage of specific manipulation, sometimes as a result of being so manipulated by older persons; and there are some boys who masturbate quite specifically and with some frequency from the age of two or three."[24] Another chart in the male report titled "Speed of Adolescent Orgasm" records the length of time it took for children to reach climax and includes the notation, "Duration of stimulation before climax; observations timed with a second hand or stop watch. Ages range from five months of age to adolescence."[25] Perhaps the most painful reading in the male report is the description of children who supposedly orgasm, a description supplied from adults who had sex with children, describing the children "groaning, sobbing, or more violent cries, sometimes with an abundance of tears (especially among younger children)" and also children who "will fight away from the partner."[26] This final description sounds like a terrified child being molested.[27]

What do we make of the data on children in *Sexual Behavior in the Human Male?* John Bancroft, former director of the Kinsey Institute, contends all of

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[23]Ibid., 177.

[24] Ibid., 501.

[25] Ibid., 178.

[26] Ibid., 161.

[27] Judith Reisman has strongly suggested that Kinsey's researchers were the ones guilty of perpetrating the violence on the children. *See* Judith A. Reisman and Edward W. Eichel, *Kinsey, Sex and Fraud: The Indoctrination of a People* (Lafayette, LA: Huntington House Publishers, 1990). Reisman's claims have been quite controversial and The Kinsey Institute itself categorically denies that Kinsey or his researchers participated in experiments on children. John Bancroft, director of the Kinsey Institute from 1995 – 2004, contends all the data in Kinsey's statistical tables regarding pre-adolescent orgasm came from one man who had sex with many adults and children beginning in 1917 until the time Kinsey interviewed him in the mid-1940s. Since Kinsey mentions gathering data from nine people who molested children, Bancroft says he does not know why Kinsey did not want to admit all the data came from one person, but suggests Kinsey "did not want to draw attention to this one man, or alternatively because he was particularly interested in this evidence and did not want to diminish its possible scientific credibility by revealing its single source." Bancroft further argues that Kinsey did not promote child molestation, did not train people to molest children, and was not in any sense a pedophile. John Bancroft, "Alfred C. Kinsey and the Politics of Sex Research," *Annual Review of Sex Research* 1.15 (2004): 16 – 17. At a bare minimum one would expect Bancroft to concede the lack of informed consent on the part of the children, but he does not do so.

Kinsey's data concerning adolescents came from one man. If Bancroft is correct, then Kinsey is *at least* guilty of lying in his research by asserting the data came from several people when in actuality it came from one man who can only be described as a serial child molester. Furthermore, Bancroft protests that Kinsey did not encourage child molestation, but this seems to be a weak defense. Recently, Joe Paterno was fired from Penn State because he did not report a child molester to the police, which is the same thing Kinsey failed to do. What is most disturbing is Kinsey's refusal to make any moral judgment concerning the "data" he obtained about children. Notice the terms he uses for child molestation: the observers were "technically trained," the molesters are called "adult observers," and the molesters are actually called the child's sexual "partner." Perhaps Kinsey's own distorted view of child sexuality is best found in *Sexual Behavior in the Human Female* in which he says, "It is difficult to understand why a child, except for its cultural conditioning, should be disturbed at having its genitalia touched, or disturbed at seeing the genitalia of other persons, or disturbed at even more specific sexual contacts."[28] Kinsey could not sympathize with the reaction of the children to being molested. The inability to sympathize with victims is a character trait associated with a person whose conscience is seared and non-functional.

Two aspects of Kinsey's research have had the most enduring impact in relation to homosexuality: The Kinsey Scale and the "10%" myth. As noted above, the Kinsey Scale is weighted to find any level of homosexual attraction and it is still used in research today. By using the Kinsey Scale, some assessments may have a built-in bias. Giving exclusive heterosexual attraction a score of "0" significantly skews the conclusions about the prevalence of homosexuality.[29] But perhaps the most enduring influence of Kinsey's report is the 10% myth -- the idea that 10% of people are homosexual. The true number of people who are homosexual is much lower than Kinsey suggests. The pro-homosexual Williams Institute at UCLA's School of Law reported in 2011 that about 3.5% of American adults self-identify as lesbian, gay, or bisexual and that a further .03% identify as transgender. Among the 3.5% who identify as gay, lesbian, or bisexual, bisexuals comprise a slight majority of 1.8% as opposed to 1.7% who identify as gay or lesbian.[30] About 1.1% of women and 2.2% of men self-identify as exclusively

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[28] Kinsey, *Sexual Behavior in the Human Female*, 121.

[29] Pro-homosexual author Fausto-Sterling comments, "In studies that search for a genetic link to homosexuality . . . the middle of the Kinsey scale disappears; researchers seek to compare the extreme ends of the spectrum in hopes of maximizing the chance that they will find something of interest." Anne Fausto-Sterling, *Sexing the Body: Gender Politics and the Construction of Sexuality* (New York: Basic Books, 2000), 10.

[30] In 1903, Magnus Hirschfeld (1868 – 1935) claimed 2.2% of people he surveyed were homosexual. *See* Vern L. Bullough, *Science in the Bedroom* (New York: Basic Books, 1994), 67.

homosexual[31] Although Kinsey's data and conclusions are flawed, his work opened the door for public discussion of homosexuality and helped set the stage for one of the most defining moments in the Gay Rights movement: The removal of homosexuality as a mental disorder from DSM-II in 1973.

II. American Psychiatric Association

The American Psychiatric Association (APA) is the world's largest professional organization for psychiatrists. It publishes the *Diagnostic and Statistical Manual of Mental Disorder*s (DSM), the standard professional reference work used by psychiatrists and mental health professionals for determining a diagnosis. The DSM's first edition was published in 1952 with subsequent revisions following in 1968 (DSM II), 1980 (DSM III), 1994 (DSM IV), and a new revision published in 2013 (DSM V).

Both DSM I[32] and II[33] listed homosexuality as a mental disorder, but soon after the publication of DSM II, homosexual activists began exerting strong pressure to remove homosexuality as a mental disorder. At a 1970 meeting of the APA held in San Francisco, homosexual activists used tactics similar to anti-war protests of the era and interrupted the meetings, yelling at psychiatrists and shouting down different program speakers. In particular, Irving Bieber (1911 – 1991), a noted psychotherapist working at New York Medical College, was presenting a paper on homosexuality and transsexualism when he was interrupted by protesters, one of whom shouted, "I've read your book, Dr. Bieber, and if that book talked about black people the way it talks about homosexuals, you'd be drawn and quartered and you'd deserve it."[34] The homosexual activists were even more animated during a panel discussion addressing issues in sexuality and asked one speaker, "Where did you take your residency, Auschwitz?"[35]

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[31] Gary J. Yates, "How Many People are Lesbian, Gay, Bisexual, and Transgender?" http://williamsinstitute.law.ucla.edu/wp-content/uploads/Gates-How-Many-People-LGBT-Apr-2011.pdf. (Accessed January 3, 2013). One is left to wonder why the Kinsey Institute can claim, "Interestingly, most statistics, such as homosexual behavior, did not change significantly from the original reports." This statement is plainly inaccurate and sounds self-serving. The Kinsey Institute, "Facts About *Kinsey,* The Film." www.kinseyinstitute.org/about/Movie-facts.html. (Accessed December 21, 2012).

[32] DSM I referred to homosexuality as "sexual deviation," a form of "sociopathic personality disturbance." The Committee on Nomenclature and Statistics of the American Psychiatric Association, *Diagnostic and Statistical Manual of Mental Disorders* (Washington, D.C.: American Psychiatric Association Mental Hospital Service, 1952), 38 – 39.

[33] DSM II listed homosexuality also listed homosexuality as a "sexual deviation." The Committee on Nomenclature and Statistics of the American Psychiatric Association, *DSM II / Diagnostic and Statistical Manual of Mental Disorders* (Washington, D.C.: The American Psychiatric Association, 1968), 44.

[34] Ronald Bayer, *Homosexuality and American Psychiatry: The Politics of Diagnosis*, 103.

[35] Ibid., 103. The question was directed at Australian Psychiatrist Nathaniel McConaghy.

Caught off guard by the fierce protests, the APA responded to the pressure and at the 1971 annual meeting held in Washington, DC, the APA sponsored a special panel discussion composed of homosexuals themselves. At this meeting, homosexual activists associated with the Gay Liberation Front stormed into the building and interrupted the meeting, demanding the APA change its stance on homosexuality. Change quickly followed, and on December 15, 1973, the board of trustees of the APA voted to remove homosexuality as a mental disorder from DSM II. In 1974, the membership at large of the APA was asked to vote to sustain or deny the change to the DSM II. Ten thousand psychiatrists participated in the referendum and approved the change by a 58% margin. Thus, via political pressure and protests, homosexual activists were able to force a change in the DSM. The revised seventh printing DSM II now only mentioned "sexual orientation disturbance," and added, "This diagnostic category is distinguished from homosexuality, which by itself does not constitute a psychiatric disorder."[36] When the DSM III was released in1980, "sexual orientation disturbance" was changed to "ego-dystonic homosexuality," a classification that was reserved for homosexuals who were distressed about their orientation.[37] The revised language essentially allowed psychiatrists to treat homosexuals who were uncomfortable with their lifestyle. But the language of "ego-dystonic homosexuality" was removed from the DSM-III in 1987. The APA went further in 1998 and rejected any attempt to help people change their sexual orientation, saying, "The American Psychiatric Association opposes any psychiatric treatment, such as "reparative" or "conversion" therapy, which is based upon the assumption that homosexuality *per se* is a mental disorder, or based upon a prior assumption that the patient should change his/ her homosexual orientation."[38] DSM V was released in May, 2013. In this edition, gender identity disorder was changed to gender dysphoria, a change viewed as a victory for transsexual individuals. Under the new paradigm a person intensely uncomfortable with their biological gender and who strongly identifies with, and wants to be, the opposite gender, may or may not be pathological. The DSM V states gender dysphoria is not meant to describe nonconformity to

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[36] The Committee on Nomenclature and Statistics of the American Psychiatric Association, *DSM II / Diagnostic and Statistical Manual of Mental Disorders*, 7th printing (Washington, D.C.: The American Psychiatric Association, 1974), 44. The American Psychological Association followed the American Psychiatric Association's lead and changed its stance on homosexuality in 1975.

[37] Tyger Latham, "Scientific Homophobia: When It Comes to Homosexuality, We Have Not Always Practiced What We Preach," *Psychology Today,* April 19, 2011. http://www.psychologytoday.com/blog/therapy-matters/201104/scientific-homophobia. (Accessed December 28, 2012).

[38] The American Psychiatric Association, "LGBT-Sexual Orientation." http://www.psychiatry.org/mental-health/people/lgbt-sexual-orientation. (Accessed December 28, 2012). This stance of the APA is reflected in a recent California law banning therapy to help minors overcome homosexual temptation and become heterosexual. As of this writing, implementation of the law is awaiting review by the Ninth Circuit Court of Appeals.

stereotypical gender role behavior such as "occasional cross-dressing in adult men."[39] Gender dysphoria is only for those who feel distressed about their desire to be the opposite gender.

Much of the conflict over homosexuality in the APA reflects a larger debate within the psychiatric community about psychoanalytic theory versus theories that see a biological basis for mental health disorders. The current trend in psychiatry and mental health is toward neurobehavioral or neurobiological solutions.[40] As a result, the psychoanalytic approach to treatment, with its origins in the work of Freud, has been largely replaced by a pharmacological approach. These battles within psychiatry were fueled by many new findings in the late Twentieth Century, including new discoveries in brain research as well as a general fall from favor of many Freudian concepts regarding sexuality. Several breakthroughs in our understanding of the brain resulted in new directions for scientific research and homosexuality.

III. Homosexuality and Brain Research

The change in the stance of the APA towards homosexuality was paralleled by rapid advances in brain research occurring at the same time. Initially, researchers discovered differences between the male and female brains in certain animals. This led scientists to ask the next logical question, "If there are differences in the brains of male and female animals, are there differences in the brains of male and female humans?" During the 1970s and 1980s, the conclusion was reached that human brains are somewhat sexually dimorphic, meaning the brains of males and females exist in two somewhat distinct forms. Then, some researchers began to speculate that homosexuals may also have brain differences as well which separate them from heterosexuals in a similar way to how male and female brains are distinguishable. The claims and counter-claims regarding findings in this area have often been controversial with significant implications in political arguments for homosexual rights.

This is a good point to mention a basic postulate of research: Representative samples are better than biased samples!

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[39] American Psychiatric Association, *DSM V/ American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders*, 5th ed. (Arlington, VA: American Psychiatric Association, 2013), 458.

[40] "Neurobehavioral" refers to the action of the nervous system and behavior. A "neurobiological" disorder is an illness of the nervous system caused by genetic or other biological factors. For example, bi-polar disorders and obsessive compulsive disorders are considered neurobiological disorders.

Initial research into the differences between the brains of male and female rodents sparked the concept of sexually dimorphic human brains. In 1971, two Oxford University scientists, Geoffrey Raisman and Pauline Field, discovered differences between male and female rat brains. They found that female rats had more synapse connections between brain cells in the hypothalamus than male rats.[41] In a subsequent study published in 1973, Raisman and Field demonstrated that neonatal exposure to testosterone organizes the male typical features in the rat brain.[42] Other studies soon followed confirming and expanding on Raisman and Field's findings.[43] Of special significance for future research into homosexuality, a team of neurobiologists led by Roger Gorski of UCLA identified differences between the hypothalamus in male and female rats. They observed a small cluster of cells (or a "nucleus" of cells) in the hypothalamus that was five times larger in the brains of male rats than in female rats. The difference between the male and female rat brains in this area was so pronounced that Gorski found he could determine the sex of rat brains using only the naked eye with near 100% accuracy.[44] The researchers named this observable area of difference in rat brains the "sexually dimorphic nucleus of the preoptic area" (SDN-POA). Hence, by the end of the 1970s, research confirmed sexually dimorphic differences between the male and female brains in rodents. The research in rodent brains set the trajectory for the following decades concerning scientific inquiry into gender-specific differences in the human brain and corresponding research concerning brain structure and homosexuality.

Building on the rapid advances in understanding animal brains, research now focused on finding an area in human brains corresponding to

[41] Geoffrey Raisman and Pauline Field, "Sexual Dimorphism in the Preoptic Area of the Rat," *Science* 173 (August 20, 1971): 731 – 733.
[42] Geoffrey Raisman and Pauline Field, "Sexual Dimorphism in the Neuropil of the Preoptic Area of the Rat and Its Dependence on Neonatal Androgen," *Brain Research 54* (1973): 1 – 29.
[43] In 1977, researchers observed differences in the shape of the stimulus-receiving ends of the nerve fibers (dendrites) between the male and female hamsters. W.T. Greenough, C.S. Carter, C. Steerman, and T.J. DeVoogd, "Sex Differences in Dentritic Patterns in Hamster Preoptic Area," *Brain Research* 126.1 (May, 1977): 63 – 72.
[44] Roger Gorski, J.H. Gordon, J.E. Shryne, and A.M. Southam, "Evidence for a Morphological Sex Difference Within the Medial Preoptic Area of the Rat," *Brain Research* 148 (1978): 333 – 346.

the SDN-POA discovered in rats. A 1985 study by Swaab and Fliers claimed to find just such a structure in humans. Swaab and Fliers asserted there is an area of the human hypothalamus corresponding to the SDN-POA in rats and it is larger in males than in females.[45] Subsequent studies have found these claims to be overstated. Most significantly, in 1989, Laura Allen, a postdoctoral assistant in Gorski's lab at UCLA, was not able to verify Swaab and Flier's claim that humans have a SDN-POA similar to rats. But Allen did make a new discovery and identified four cell groups in the preoptic area of the hypothalamus in human brains. Allen coined a new name for these cell groups: "Interstitial Nuclei of the Anterior Hypothalamus" (INAH 1-4). Furthermore, Allen and Gorski said the INAH 2 was twice as large in men as in women and that INAH 3 was 2.8 times as large in men as in women.[46] To summarize, we now know that humans do not have a SDN-POA like rats do.[47] But the area identified in humans as INAH 1 – 4 would become the focus of later studies concerning brain structure and homosexuality.

Around the same time other researchers began to look for dimorphism in one of the most popular subjects of brain research -- the corpus callosum, a thick bundle of nerves that connects the left and right brain. For a number of years, researchers asserted a significant difference between the thickness of the corpus callosum in males and females, typically asserting that the Corpus Callosum was thicker in females. A similar claim has also been made about the corpus callosum in transgender males. In 2005 one group of researchers from Japan used magnetic resonance imaging (MRI) to view the corpus callosum across what is known as the "midsagittal plane" -- a view of a brain which has been sliced between the left and right hemispheres. They claimed the corpus callosum

[45] D. F. Swaab and E. Fliers, "A Sexually Dimorphic Nucleus in the Human Brain," *Science* 228.4703 (May 1985): 1112 – 1115.

[46]Laura S. Allen, M. Hines, J.E. Shryne, and R.A. Gorski, "Two Sexually Dimorphic Cell Groups in the Human Brain," *Journal of Neuroscience* 9.2 (February 1989): 497 – 506. This study compared the brains of the cadavers of twenty-two adult men and women.

[47] Laura A. Freberg, *Discovering Biological Psychology*, 2nd ed. (Belmont, CA: Wadsworth / Cengage Learning, 2010), 301. Some researchers claim the INAH1 may be a human analogue for the SDN-POA, but this is debated. Others, including Wikipedia, claim INAH3 is analogous to the rat's SDN-POA, but this is also strongly debated.

was measurably different between males and females. Furthermore, they asserted that their subjects with gender identity disorder were more likely to have a corpus callosum more similar to the sex with which they identified than the sex with which they were born. In other words, males who identified as females were more likely to have a corpus callosum similar to females than males. Likewise, females who identified as males were more likely to have a corpus callosum similar to males than females. The researchers went so far as to conclude that their data "can be utilized for diagnosis of GID [Gender Identity Disorder] as an objective and quantitative criteria."[48]

Caution should be urged when forming opinions about homosexuality based on studies of the corpus callosum since several factors can affect the measurement of the corpus callosum or its individual areas. One group of researchers note "sex differences in any proportion measure[d] . . . must be interpreted with caution, since any association with sex and age and the ratio's denominator can create a substantial effect. As a result, significant sex differences in ratio measures have often been found to disappear when analysis of covariance is used on the same data."[49] Gorski and his colleagues attempted to find sexual dimorphism in the corpus callosum, but found "no conclusive evidence of sexual dimorphism in the area of the corpus callosum or its subdivisions."[50] Contradictory opinions abound concerning purported gender-specific differences in the corpus callosum, making inferences about sexual preference based on dimorphism in the corpus callosum even more speculative.

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[48] Y. Yokota, Y. Kawamura, and Y. Kameya, "Callosal Shapes at the Midsagittal Plane: MRI Differences of Normal Males, Normal Females, and GID," Conference Proceedings of the 2005 IEEE Engineering in Medicine and Biology 27th Annual Conference, Shanghai, China, September 1-4, 2005. 3:3058.

[49] Paul M. Thompson, Katherine L. Narr, Rebecca E. Blanton, and Arthur W. Toga, "Mapping Structural Alterations of the Corpus Callosum During Brain Development and Degeneration," in *The Parallel Brain: The Cognitive Neuroscience of the Corpus Callosum,* Eran Zaidel and Marco Lacobni, eds. (Cambridge: Massachusetts Institute of Technology Press, 2003), 98.

[50] Laura S. Allen, Mark F. Richey, Yee M. Chai, and Roger A. Gorski, "Sex Differences in the Corpus Callosum of the Living Human Being," *The Journal of Neuroscience* 11.4 (April 1991): 933. Even more amazingly, some people are actually born without a corpus callosum, a condition known as agenesis of the corpus callosum. As an example of contradictory evidence, in 2003 John Allen, et al performed MRI measurements of male and female brains and actually found the volume of males to be greater than females. *See* John S. Allen, Hanna Damasio, Thomas J. Grabowski, Joel Bruss. and Wei Zhang, "Sexual dimorphism and Asymmetries in the Gray–White Composition of the Human Cerebrum," *NeuroImage* 18.4 (April 2003): 880 – 894. For a recent study re-asserting the corpus callosum is larger in females, see Babak A. Ardekani, Khadija Figarsky, and John J. Sidtis, "Sexual Dimorphism in the Human Corpus Callosum: An MRI Study Using the OASIS Brain Database," *Cerebral Cortex* 23.10 (2013): 2514-2520.

Before moving forward, it is important to connect the study of gender-specific brain studies and research into biological causes of homosexuality. The argumentation of some homosexual activists is as follows: (1) Brains of males and females are sexually dimorphic. (2) In a similar way, brains of homosexuals are also sexually dimorphic. (3) Since our society says it is wrong to discriminate against someone because of their innate gender differences, it is also wrong to discriminate against homosexuals because they also have scientifically proven innate differences in sexual orientation.

There are several problems with this line of thinking, but I will mention four. First, attempts to find sexual dimorphism in human brains as clear as that found in animals has produced inconclusive results. Second, due to the wide variety of people on earth, a certain amount of deviation should be expected when measuring the sizes of various parts of the human brain any time two different groups are studied. This leads to the third criticism, which is that differences between male and female brains have sometimes been exaggerated. Males and females, both homosexual and heterosexual, have the same components and structures in their brains. Differences which have been identified are usually differences in volume or shape. One author goes so far as to say, "During the past century, numerous researchers have tried to find anatomic factors differentiating the male and female brain. To date, only one reliable difference has been found – regardless of sexual orientation, men have slightly larger brains than women."[51] While there seems to be some minor gender associated differences in the hypothalamus, the brains of men and women are basically identical. The fourth problem with the way brain studies have been used relates to brain plasticity. "Plasticity" of the brain, or neuroplasticity, is a way of describing the remarkable manner in which the human brain can form new neural pathways, strengthen existing neural pathways by repetitious use, or bypass damaged pathways. This is important for moral debates because these pathways can be strengthened, discarded, or by-passed based on volitional choices. In other words, some differences in the measurements of human brains are definitely the result of our response to the environment in which we live.

On occasion one may encounter a person struggling with issues of sexual temptation or gender identity who claims to have a "female brain in a male body" or a "male brain in a female body." In this way, brain research is used in a crude manner to validate transgender or homosexual behavior. This assertion is made on flawed assumptions about the differences between male and female brains.

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[51] Bertam J. Cohler and Robert M. Galatzer-Levy, *The Course of Gay and Lesbian Lives: Social and Psychoanalytic Perspectives* (Chicago: University of Chicago Press, 2000), 83.

Evangelical preachers sometimes add to this confusion by over-emphasizing the differences between male and female brains in well-intended efforts to affirm healthy distinctions between the genders. But we must remember male and female brains are overwhelmingly more alike than different, with the most robust difference being a 10% larger total brain size in males. Giedd, et al comment on other gender-related brain differences, saying, "Other brain morphometric differences depend on whether or how subcomponents are adjusted for total brain volume with the largest effect sizes reported for the caudate nucleus, amygdala, and hippocampus, and cerebellum. Non-linear scaling effects may lead to regional differences attributable to variation in brain size alone."[52] In other words, the differences in brain size may account for some of the differences in measurement of specific areas. Two small areas of the hypothalamus have consistently shown some minor differences between men and women: The preoptic area of the hypothalamus tends to be larger in males than in females (this is related to the INAH 1 – 4) and the Suprachiasmatic Nucleus of the Hypothalamus is shaped differently in males and females.[53]

Since the hypothalamus is the area of the brain where some differences between male and female brains have been identified, it also has been the focus of research concerning the possible difference in brain structures between homosexuals and heterosexuals. Because the hypothalamus connects the nervous system to the endocrine system by producing hormones, the hypothalamus affects the human sex drive. The hypothalamus is a hub of regions high in sex steroid receptors, having a high density of estrogen, androgen, and progesterone receptors.[54] This relationship between the sex-drive and the hypothalamus further explains why it has been the focus of research concerning a biological origin for homosexuality. Two studies have been the focus of interest in particular: Swaab and Hofman on the Suprachiasmatic Nucleus and Simon LeVay on the Interstitial Nucleus of the Anterior Hypothalamus.

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[52] Jay N. Giedd, Armin Raznahan, Kathryn L. Mills, and Roshel K. Lenroot, "Review: Magnetic Resonance Imaging of Male/Female Differences in Human Adolescent Brain Anatomy," *Biology of Sex Differences* 3.19 (2012): 7. These authors suggest the basal ganglia, amygdala, hippocampus, and cerebellum are sexually dimorphic.

[53] University of Washington, "Neuroscience for Kids: He Brains, She Brains." http://faculty.washington.edu/chudler/heshe.html. (Accessed December 29, 2012). The hypothalamus is a highly complex area of the brain below the thalamus that regulates any number of physiological processes and autonomic activities of the human body.

[54] Jay N. Giedd, Armin Raznahan, Kathryn L. Mills, and Roshel K. Lenroot, "Review: Magnetic Resonance Imaging of Male/Female Differences in Human Adolescent Brain Anatomy," 5.

Swaab and Hofman – the Suprachiasmatic Nucleus

One of the most influential researchers in the area of homosexuality and brain research has been Dutch scientist Dick F. Swaab. From 1978 – 2005, he served as the director of the Netherlands Institute for Brain Research. Swaab has published many articles and is widely quoted in literature concerning homosexuality and brain structure. Swaab's most frequently cited study was published in 1990 in the journal *Brain Research*. Co-written with Michael Hofman, the article was titled "An Enlarged Suprachiasmatic Nucleus in Homosexual Men." The Suprachiasmatic Nucleus (SCN) is a collection of nerve cell bodies on either side of the hypothalamus. It is thought to have an involvement in sexual desire because of the varying body rhythms associated with sexual desire as well as the sexual changes that come with aging.[55] Swaab and Hofman studied the SCN in thirty-four cadavers. Of these thirty-four cadavers, eighteen were a reference group of males who died of various causes, ten were homosexual men who died of AIDS, and six were heterosexuals who died of AIDS (two males, four females). Swaab and Hofman claimed to observe a larger SCN in the cadavers of homosexual men than those in the heterosexual reference group: "The SCN volume in homosexual males was 1.73 times larger than male subjects from the reference group . . . and contained 2.09 times as many cells."[56] They went on to say that this size difference means that homosexuals have a more elongated SCN than heterosexuals. Furthermore, they suggested this enlargement of the SCN might be a cause of homosexuality: "An association was found . . . between sexual orientation in men and SCN size, from which the functional implications are momentarily not clear."[57] It is important to reiterate that Swaab and Hofman were building on previous research which asserted differences between the male and female brains. Just as some suggested males and females have sexually dimorphic brains, Swaab and Hofman suggested homosexuals also have a dimorphic brain structure based on sexual orientation.

However, Swaab and Hofman's research on the Suprachiasmatic Nucleus was plagued by several problems. First, to date the findings about the SCN have

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[55] John Feinberg and Paul Feinberg, *Ethics for a Brave New World*, 2nd ed. (Wheaton, IL: Crossway Books, 2010), 368.

[56] D.F. Swaab and Michael A. Hofman, "An Enlarged Suprachiasmatic Nucleus in Homosexual Men," *Brain Research* 537 (1990): 145.

[57] D.F. Swaab and Michael A. Hofman, "An Enlarged Suprachiasmatic Nucleus in Homosexual Men," 146. It should also be noted that Swaab and Hofman claimed their research refuted the idea that male homosexuals have a "female brain." Swaab and Hofman, "An Enlarged Suprachiasmatic Nucleus," 145.

not been replicated.[58] Second, this study was very small consisting of only thirty-four cadavers and of these only ten were homosexuals. Third, the reference group identified as heterosexuals may not serve as a reliable comparison, since, as Swaab and Hofman state themselves, "Sexual preference of the subjects of the reference group was generally not known."[59] In other words, the researchers *assumed* the members of reference group were heterosexuals or arbitrarily assigned them the designation. In either case, if the reference group is not confirmed, all the data comes into question. Finally, the connection between the SCN and sex has been questioned.[60] If the SCN is not associated with sex, then it is difficult to see how it can be associated with same-sex desire.

Simon LeVay – Interstitial Nucleus of the Anterior Hypothalamus

Simon LeVay is a neuroscientist who worked for the prestigious Salk Institute for Biological Studies from 1984 – 1993. A practicing homosexual, LeVay was the co-founder of The Institute of Gay and Lesbian Education located in West Hollywood, CA, and served as the Institute's chairman of board of directors from 1992 – 1996.[61] Building on the earlier findings of Gorski and his colleagues that the INAH 2 & 3 are larger in males than in females, LeVay also focused on the INAH 1 – 4 to see if there were any corresponding differences based on sexual preference. Studying 41 cadavers, LeVay identified 19 of the subjects as homosexual men (including one man who self-identified as bisexual, but was placed in the category of "homosexual") – all of whom died of complications from AIDS, 16 as heterosexual men – 6 of whom died of AIDS related illnesses, and 6 as heterosexual women, 1 of whom died of AIDS. LeVay's findings were published in the August, 1991 issue of *Science*, and were widely reported as evidence for a biological cause for homosexuality. LeVay could not replicate Allen and Gorski's claim that INAH 2 is larger in males than in females. But he did find that the INAH 3 is larger in males than females. Furthermore, he claimed the INAH 3 cell group was more than twice as large in heterosexual men as opposed to homosexual men. LeVay's claims were profound not only because he claimed to replicate the finding that a certain amount of sexual dimorphism existed at INAH 3, but he was claiming that homosexual men have an INAH 3

[58] Simon LeVay, *Gay, Straight, And The Reason Why: The Science of Sexual Orientation* (New York: Oxford University Press, 2010), 200.
[59] Swaab and Hofman, "An Enlarged Suprachiasmatic Nucleus in Homosexual Males," 141.
[60] Simon LeVay, *Gay, Straight, and the Reason Why*, 200. Readers should note that Swaab and LeVay each argue for competing theories concerning the biological origin of homosexuality. LeVay promotes his own hypothesis while rejecting Swaab's.
[61] Simon LeVay, "My Resume." http://www.simonlevay.com/my-resume. (Accessed January 4, 2013).

more close in size to women than to heterosexual men.[62] While LeVay did assert that further interpretation of the results of his study must be considered speculative, he concluded by saying that it is more likely "the size of the INAH 3 is established early in life and later influences sexual behavior than that the reverse is true."[63] Hence, LeVay was saying the size of the INAH 3 – smaller in homosexual males – affects sexual orientation and was rejecting the idea that sexual behavior affects the size of the INAH 3.

LeVay's research was greeted with quite a bit of fanfare, but it is also plagued by some significant problems. First, much like Swaab and Hofman's research, LeVay's sample was very small, consisting of only 41 subjects. It is imprudent to make global assumptions about same-sex attraction based on a study consisting of such a small number. But a larger problem with LeVay's research is related to the manner in which he classified his research subjects as either heterosexual or homosexual. Satinover notes that LeVay's definitions for each were very imprecise, nor was there any way of verifying sexual orientation since the subjects being studied were dead.[64] Writing in *Technology Review*, Paul Billings and Jonathan Beckwith commented on LeVay's study, emphasizing that LeVay's "research design and subject sample did not allow others to determine whether it was sexual behavior, drug use, or disease history that was correlated with the observed differences among the subjects' brains."[65] The effect of AIDS on the subjects of LeVay's study is also not clear. Could AIDS have affected the area of the brain LeVay was studying, thus accounting for the differences? In 2000, Jones and Yarhouse asserted that AIDS and medications used to treat the HIV infection can affect the very part of the brain LeVay was studying. Jones and Yarhouse thus conclude, "We do not know whether his [LeVay's] findings are related to homosexuality or to the medications used to treat HIV." On top of these difficulties are questions that are more fundamental regarding the degree to which the hypothalamus does or does not affect sexual orientation. Cohler and Galatzer-Levy, authors favorable to homosexual rights, go so far as to say "there is little evidence that the hypothalamus plays any role in human sexual orientation."[66]

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[62] Van Wyk and Geist noted that the one subject listed as "bisexual" had a INAH 3 that resembled heterosexual men rather than the other homosexual men. Paul H. Van Wyk and Chrisann S. Geist, "Biology of Bisexuality: Critique and Observations," *Journal of Homosexuality* 28.3-4 (1995): 359. Van Wyk and Geist apparently base their comments on LeVay's "Figure 2" in his article. *See* Simon LeVay, "A Difference in Hypothalamic Structure Between Heterosexual and Homosexual Men," *Science* 253 (August 30, 1991):1036.

[63] Simon LeVay, "A Difference in Hypothalamic Structure Between Heterosexual and Homosexual Men," 1036.

[64] Jeffrey Satinover, *Homosexuality and the Politics of Truth* (Grand Rapids: Baker Book House, 1996), 78 – 79.

[65] Paul Billings and Jonathan Beckwith, "Born Gay?" *Technology Review* 96.5 (July 1993): 60.

[66] Cohler and Galatzer-Levy, *The Course of Gay and Lesbian Lives*, 82.

Another problem with LeVay's research is difficulty in precise measurement of the INAH 1 – 4, parts of the brain no bigger than a pinpoint.[67] Swaab and Hofman evaluated LeVay's approach of measuring the volume of the hypothalamic structures and said: "Volume is susceptible to various pre- and post-mortem factors, such as differences in agonal state and fixation time but also to histological procedures and methods, such as section thickness. Therefore, it is essential to include data on total cell numbers of hypothalamic nuclei, since this parameter is not influenced by such factors."[68] In order to study tissues, the tissues themselves must be preserved and cut into sections thin enough to be translucent. Measuring the volume of something as small as the INAH 3 is therefore difficult, but not impossible. However, volume is affected by other external factors, such as the process of death itself. Taken as a whole, these variables may have skewed LeVay's findings.

Further investigation has challenged LeVay's conclusions and suggests he did in fact incorrectly measure the volume of the INAH 3. In 2001, William Byne, director of the Neuroanatomy and Morphometrics Laboratory at the Icahn School of Medicine at Mount Sinai Hospital, led a research team that examined the INAH 1- 4 and its connection to sexual orientation. This particular study measured via autopsy the INAH from 34 men presumed to be heterosexual (10 of whom were HIV positive at death), 34 presumed heterosexual women (9 of whom were HIV positive at death), and 14 homosexual men, all of whom were HIV positive at death. Byne, et al asserted there does seem to be evidence that INAH-3 occupies a larger volume and contains more neurons in heterosexual men than in women, but there was no differentiation in the number of neurons between heterosexual men and homosexual men. The researchers stated, "The present study provides further evidence that INAH-3 occupies a larger volume and contains more neurons in presumed heterosexual men than women. The primary sexually dimorphic cellular characteristic of INAH3, neuronal number, did not vary as a function of sexual orientation."[69]

Two specific findings from Byne's research are worth noting concerning the relationship of the INAH 3 to homosexuality. First, while there was no difference

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[67] Michael R. Kauth, *True Nature: A Theory of Sexual Attraction* (New York: Kluwer Academic / Plenum Publishers, 2000), 126.

[68] D.F. Swaab and M.A. Hofman, "Sexual Differentiation Of The Human Hypothalamus in Relation to Gender and Sexual Orientation," *Trends in Neurosciences* 18.6 (January 1, 1995): 266 – 267. *Agonal state* refers to the state of a person during the time immediately preceding death.

[69] William Byne, Stuart Tobet, Linda A. Mattiace, Mitchell S. Lasco, Eileen Kemether, Mark A. Edgar, Susan Morgello, Monte S. Buchsbaum, and Liesl B. Jones, "The Interstitial Nuclei of the Human Anterior Hypothalamus: An Investigation of Variation with Sex, Sexual Orientation, and HIV status." *Hormones and Behavior* 40 (2001): 89.

in the number of neurons in INAH3 between heterosexual and homosexual men, the INAH3 of homosexual men did tend to occupy a smaller volume in homosexual males than in heterosexual males. Basically, the gay men and straight men had the same number of neurons in this region of the brain, but the neurons were packed more densely in gay men. Another finding of interest in the Byne study was that they found "no evidence for an influence of HIV on INAH3, lending credence to LeVay's (1991) contention that HIV infection did not account for the disparity of INAH3 volume he observed between homosexual and heterosexual men."[70] This is significant because, as noted earlier, some had questioned if LeVay's initial findings were flawed because of the influence of AIDS on the brains of the men he studied.[71]

So what does the Byne study tell us about LeVay's claim that the INAH-3 is different in heterosexual male and homosexual males? First, one of the more robust findings of brain research is that men tend to have more neurons in INAH-3 than women. However, there is no difference in the number of neurons in the INAH-3 of heterosexual males as compared to homosexual males. The neurons in INAH-3 seem to be more densely packed in homosexual males than in heterosexual males, thus leading to the finding that the "volume" of the INAH 3 is smaller in gay men than in straight men. Thus, Byne and his co-authors say, "Based on the results of the present study as well as those of LeVay, sexual orientation cannot be reliably predicted on the basis of INAH3 volume alone."[72] LeVay himself offers a nuanced response to Byne and says, "Byne's findings were in no way a refutation of the findings of my study, but neither were they a clear-cut confirmation."[73] In other words, LeVay is admitting that his claims have not been confirmed.

The comments of Byne, et al are even more fascinating because they actually discuss the relationship of environment to brain structure. To understand the importance of their comments, it is helpful to understand some basic concepts about the growth and development of the human brain. The basic building block of the brain and the central nervous system is the neuron, a highly specialized brain cell that communicates information throughout the body. When babies are born, they have just about all the neurons they will ever have, close to 100 billion. Unlike most other cells, neurons generally do not grow or repair after damage.

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[70] Ibid., 91.

[71] For example, Stanton A. Jones and Mark A. Yarhouse, *Homosexuality: The Use of Scientific Research in the Church's Moral Debate* (Downers Grove, IL: InterVarsity Press, 2000), 70.

[72] Byne, et al, "The Interstitial Nuclei of the Human Anterior Hypothalamus," 91.

[73] LeVay, *Gay, Straight, and the Reason Why,"* 199.

One source explains, "Brain development, or learning, is actually the process of creating, strengthening, and discarding connections among the neurons; these connections are called synapses. Synapses organize the brain by forming pathways that connect the parts of the brain governing everything we do—from breathing and sleeping to thinking and feeling."[74] Closely related to the growth of a child's brain is the concept of *brain plasticity* introduced earlier. The human brain has greater plasticity at younger ages and less plasticity when older.

Some researchers have suggested that people develop an orientation towards same-sex attraction based on hormones to which they are exposed prenatally. Byne suggests that "sex related differences may also emerge later in development as the neurons that survive become part of the functional circuits."[75] The upshot of the research is that these differences between the INAH3 of heterosexual males and homosexual males are not proof of prenatal, biological determination of sexual orientation. Instead, these differences could be the result of postnatal experience. Jones and Kwee explain the importance of Byne's findings and say, "In other words, if there are brain structure differences between homosexuals and heterosexuals, they could well be the result rather than the cause of sexual behavior and preference."[76]

A final word concerning LeVay's research concerning the INAH 3 is related to an evolutionary view of human origins. As was noted above, humans do not have a SDN-POA as has been found in rats. However, some researchers now contend that the INAH 3 in humans and the SDN-POA in the rat are homologous, meaning they are similar in evolutionary origin and share a common ancestor. Based on this premise, continuing research on the sexual behavior of rats is viewed as informative for research into human homosexual behavior.

Allen and Gorski – 1992

In 1992, Laura Allen and Roger Gorski published an article in the *Proceedings of the National Academy of Sciences*, arguing the anterior commissure (AC) of the brain is larger in homosexual men than in either heterosexual men or women. The AC is a bundle of nerve fibers connecting the two temporal lobes, and is quite a bit smaller than the corpus callosum. In this study, a postmortem

[74] U.S. Department for Health and Human Services, "Understanding the Effects of Maltreatment on Brain Development." https://www.childwelfare.gov/pubs/issue_briefs/brain_development/how.cfm. (Accessed January 7, 2013).
[75] Byne, et al, 91.
[76] Stanton L. Jones and Alex W. Kwee, "Scientific Research, Homosexuality, and the Church's Moral Debate: An Update," *Journal of Psychology and Christianity* 24.4 (2005): 307.

comparison was made between the anterior commissure of 30 homosexual males, 30 heterosexual males, and 30 heterosexual females. The researchers reported there was a significant difference in the area of the [Anterior Commissure] between the three groups: The AC of homosexual men was 18.0% larger than that of heterosexual women and 34% greater than that of heterosexual men.[77]

Though this Allen and Gorski study is still widely quoted as evidence for a biological aspect of homosexuality, there are several reasons this claim should be taken with caution. First, in the 20 years since it was published, the findings have not been replicated. Secondly, when one examines the graph in which Allen and Gorski plotted the average size of the AC in the three groups, it becomes clear that there is significant overlap in the size of the AC of many of the subjects that they studied. In other words, several homosexual men, heterosexual men, and heterosexual women had ACs very similar in size.[78] Not *every* homosexual subject had an AC that was 34% larger than *every* heterosexual male: the data reflects the average of all the subjects studied, and thus shows a *trend* and not an absolute reality in every case. Neither did the study prove causation, but Allen and Gorski only argued for a "correlation between sexual orientation and the midsaggital area of the AC," and added that their research "clearly argues against the notion that a single brain structure causes or results from a homosexual orientation."[79] In other words, Allen and Gorski seemed more cautious in their article while subsequent reporting of their findings amplified the idea of *causation.*

Savic and Lindstom – 2008

Ivanka Savic-Berglund is a neurologist at the Karolinska Institute in Stockholm. She led a team of researchers who investigated the degree of brain asymmetry in homosexuals compared to heterosexuals. Brain asymmetry refers to the differences in all humans between the two hemispheres of the brain.[80] Savic and her colleagues compared the asymmetry of the brains of 25 heterosexual men, 25 heterosexual women, 20 homosexual men and 20 homosexual women. Using both MRI and PET imaging to evaluate their sample, they made several important claims. First, they asserted that in heterosexual men the right hemisphere of the brain is slightly larger than the left while in heterosexual women two hemispheres

[77] Laura S. Allen and Roger A. Gorski, "Sexual Orientation and the Size of the Anterior Commissure in the Human Brain," *Proceedings of the National Academy of Sciences* 89.15 (1992): 7200.

[78] It is of some interest to note that two outliers in the data were two homosexual men who died of AIDs and whose anterior commissures were unusually large. Allen and Gorski acknowledged these two outliers.

[79] Laura S. Allen and Roger A. Gorski, "Sexual Orientation and the Size of the Anterior Commissure in the Human Brain," 7202.

[80] For example, in most people the right hemisphere is often larger and heavier than the left.

were about equal in size. Second, they claimed the brains of homosexual men looked more like heterosexual women with both hemispheres being equal in size and the brains of homosexual women looked more like heterosexual men with the right hemisphere being slightly larger. Finally, the researchers examined the amygdalae, the two almond-shaped structures in the brain that are closely connected to our emotions, such as fear and anger. They claimed to find that heterosexual men and women have somewhat different connections between the amygdalae and the rest of the brain. Again, the homosexual men in their study showed connections between the amygdalae and the rest of the brain more similar to heterosexual females and homosexual females had connections more similar to heterosexual males.[81]

Savic's findings were greeted with great fanfare, leading Qazi Rahman of the University of London to announce, "As far as I'm concerned there is no argument any more – if you are gay, you are born gay."[82] Savic and Lindstrom were more restrained and acknowledged that their findings need to be confirmed by additional research.[83] What should be kept in mind is that fundamental to their claims about homosexuality are claims about differences in brain asymmetry between men and women. While their research may be replicated, other claims about the differences between the brains of males and females were also greeted with initial enthusiasm only to be found less compelling in later research.

IV. Homosexuality and Twin Studies

When addressing the degree to which homosexuality is affected by genetic or environmental factors, it is easy to see why researchers would be drawn to twin studies since identical twins share a virtually identical genetic identity. If homosexuality does have a genetic component, then research focusing on the degree to which twins do or do not share a homosexual orientation should be quite informative. Put most simply, a twin study is a genetic study performed to determine the heritability of specific traits. Since identical twins (also known as "monozygotic twins") share the same DNA, the assumption is that any major differences between the twins must be the result of other factors.

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[81] Ivanka Savic and Per Lindstrom, "PET and MRI Show Differences in Cerebral Asymmetry and Functional Connectivity Between Homo- and Heterosexual Subjects," *Proceedings of the National Academy of Sciences* 105.27 (June 16, 2008): 9403 – 9408.

[82] BBC News, "Scans See Brain Differences." June 16, 2008. https://news.bbc.co.uk/2/hi/health/7456588.stm. (Accessed January 25, 2013).

[83] Rob Stein, "Brain Study Shows Differences Between Gays, Straights," *The Washington Post,* June 23, 2008. http://www.washingtonpost.com/wp-dyn/content/story/2008/06/22/ST2008062202006.html. (Accessed January 25, 2013).

Bailey and Pillard, 1991, 1993

Michael Bailey, a psychologist and professor at Northwestern University, and Richard Pillard,[84] a professor of psychiatry at Boston University, researched the concordance rate for homosexuality among male twins hoping to discern genetic linkage to homosexuality. They published their findings in 1991 in the *Archives of General Psychiatry* and asserted genetic factors are "important in determining individual differences in sexual orientation."[85] The claim to find a genetic link to homosexuality was greeted with great fanfare and Bailey and Pillard have been often-quoted since then as evidence for the constitutional nature of homosexuality.

Bailey and Pillard searched for people in the homosexual community who had a twin. To develop their sample base, they recruited participants via advertisements in gay-friendly magazines and publications, specifically asking for male homosexuals who had a male co-twin or an adopted brother. Their goal was to discover what percentage of siblings of homosexual males were also homosexual males. Eventually, 161 interviews of homosexual males were collected, 115 of whom had a male twin and 46 having adoptive brothers. Bailey and Pillard's most striking claim is that 52% (29/56) of the monozygotic co-twins were either homosexual or bisexual. Among homosexual males with a dizygotic (non-identical) twin brother, 22% (12/54) of their co-twins were also homosexual. They also found that non-twin biological brothers in the sample had a 9.2% (13/142) concordance rate for homosexuality compared to a 11% (6/57) concordance rate for non-twin adoptive brothers.[86]

Bailey and Pillard initiated a follow-on study of homosexual women with a female co-twin or adoptive sisters. Publishing their findings in 1993 in the *Archives of General Psychiatry*, they again claimed to find a strong genetic component for homosexuality. As in their male study, prospective participants were recruited from advertisements in gay-friendly publications, eventually gathering data from 147 interviews, 115 of whom were homosexual females with a female twin and 32 with adoptive sisters. They reported a concordance rate of 48% (34/71) for monozygotic twins, 16% (6/17) for dizygotic twins, 6% (2/35) for

[84] In a 2010 interview, Pillard said, "I have to say I'm a hard core atheist." Kimberly Cornuelle, "Nature vs. Nurture: The Biology of Sexuality," *BU Today,* November 16, 2010, accessed July 28, 2017, https://www.bu.edu/today/2010/nature-vs-nurture-the-biology-of-sexuality/.

[85] J. Michael Bailey and Richard C. Pillard, "A Genetic Study of Male Sexual Orientation," *Archives of General Psychiatry* 48 (December 1991): 1093.

[86] J. Michael Bailey and Richard C. Pillard, "A Genetic Study of Male Sexual Orientation," *Archives of General Psychiatry* 48 (1991): 1092 - 1093.

adoptive sisters, and 15% (10/73) for non-twin biological sisters.[87] Bailey and Pillard concluded their report on female twins by saying, "How do the findings of the present study compare with those of Bailey and Pillard's genetic study of male sexual orientation, which employed a similar method? The most important similarity is that both male and female sexual orientation appeared to be influenced by genetic factors. However, in neither study was an indicator of genetic loading found."[88]

There can be some confusion concerning Bailey and Pillard's claims, confusion caused by the fact they use *probandwise concordance rates* and not *pairwise concordance rates.* In genetic research and twin studies, a *proband* denotes a particular person with a specifc genetic trait serving as the starting point for the genetic study of a family. So, in their research on male homosexuality in twins, Bailey and Pillard had 161 male homosexual probands. In genetic research, the *pairwise* rate only counts a concordant pair once, but the *probandwise* rate counts such pairs twice. This distinction is important to remember and brings clarification to Bailey and Pillard's claims. When the average person hears a concordance rate of 52% (29/56) among monozygotic homosexual twins, the assumption is Bailey and Pillard found 56 separate twin pairs with at least one homosexual twin, and 29 of these pairs had two homosexuals. Actually, Bailey and Pillard had a total of 41 pairs of monozygotic twins and from this group they found 14 matched groups (13 twin pairs and one triplet trio – 29 total individuals). Among the 41 monozygotic twin pairs, they also identified 27 individuals whose co-twin was not homosexual. Notice carefully that both of the individuals in a twin set (or the triplets) are counted in the numerator. The formula used to develop the concordance rate can be presented as follows:

$$\frac{\text{13 pairs or 26 homosexual individuals + 1 matched triplet set or 3 individuals = 29}}{\text{29 matches + 27 failures to match = 56}}$$

The probandwise concordance rate is widely used in genetic research and is useful because, when addressing diseases, it forecasts risk at the individual rather than the

[87] J. Michael Bailey, Richard C. Pillard, Michael C. Neale and Yvonne Agyei, "Heritable Factors Influence Sexual Orientation in Women," *Archives of General Psychiatry* 50 (1993):219, 221.

[88] J. Michael Bailey, Richard C. Pillard, Michael C. Neale and Yvonne Agyei, "Heritable Factors Influence Sexual Orientation in Women," *Archives of General Psychiatry* 50 (1993):223. In genetic research, the term "genetic loading" is often used to describe harmful genes that are carried in the genome and that are transmitted to descendants causing disease or morbidity. In this study, the term is merely used to describe the heritability of homosexuality.

pair level.[89] At the same time, it is important to clarify exactly what Bailey and Pillard claimed and they *did not* claim that of 56 monozygotic twin pairs, they found 29 pairs where both brothers were homosexual.[90]

What are we to make of the Bailey and Pillard's studies? First, while the studies indicate a genetic component to homosexuality, neither study produced 100% concordance rates. Thus, if there is a genetic aspect to homosexual orientation, it is not equal to causation. Satinover rightly says, "If 'homosexuality is genetic,' as activists and their media supporters repeatedly claim, the concordance rate between identical twins – that is, the incidence of the two twins either both being homosexual or both being heterosexual – will be 100 percent. There would *never* be a *discordant pair* – a pair with one homosexual twin and one heterosexual twin."[91] The weakness of the genetic argument suggested by Bailey and Pillard is further demonstrated by close examination of their data. Bailey and Pillard claimed that there was a 22% concordance rate between non-identical twin brothers while non-twin biological brothers had a 9.2% concordance rate for homosexuality. Remembering that non-identical twins and their non-twin biological brothers share the same amount of genetic material, one would expect to see a similar concordance rate between the two groups, yet we do not. This led Byne and Parsons to conclude, "If we rely only on the data presented in their [Bailey and Pillard's] study, we must at least consider the possibility that the higher concordance rate for homosexuality in [non-identical] twins compared with nontwin biological brothers is due to increased similarity of the trait-relevant environment in the former."[92] Finally, the method in which Bailey and Pillard's sample was obtained by seeking respondents from gay-friendly publications poses another problem for this study. It is not hard to imagine that homosexuals with a twin who was also a homosexual would be more motivated to respond to such advertisements and participate in the study, thus skewing the sample.

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[89] Matt McGue, "When Assessing Twin Concordance, Use the Probandwise Not the Pairwise Rate," *Schizophrenia Bulletin* 18.2 (1992): -174.

[90] I am extremely thankful for Stanton L. Jones' work in clarifying the exact claims of Bailey and Pillard. This paragraph owes particular debt to Jones. *See* Stanton Jones, "Homosexuality: The Use of Scientific Research in the Church's Moral Debate," (Paper delivered to the Council of Christian Colleges and Universities, November 16, 2004).

[91] Satinover, *Homosexuality and the Politics of Truth*, 83. Another study claimed to find a 2/3 concordance rate for homosexuality in monozygotic twins, but no other recent study has found a concordance rate nearly this high indicating there were errors in the way the sample was chosen. *See* F.L. Whitman, M. Diamond, J. Martin, "Homosexual Orientation in Twins: A Report of 61 pairs and three Triplet Sets," *Archives of Sexual Behavior* 22 (1993): 187 -206.

[92] William Byne and Bruce Parsons, "Human Sexual Orientation: The Biologic Theories Reappraised," *Archives of General Psychiatry* 50.3 (March 1993): 229.

Bailey, Dunne, and Martin – 2000

Bailey and Pillard acknowledged that the method by which they obtained their samples for their 1991 and 1993 studies was problematic. In 1993 they stated, "Future studies of sexual orientation that avoid this bias, for example through the use of twin registries, are clearly desirable."[93] Bailey was able to do just such research and in 2000 published findings using a large population-based sample of twins recruited from the Australian National Health and Medical Research Council Twin Registry, ultimately gathering data from 4,901 participants. Using a much larger and more representative sample provided strikingly lower percentages of concordance rates for homosexuality among twins. Among identical twins, the Australian data revealed that if a male was homosexual, there was a 20% concordance rate for homosexuality and among female homosexuals with an identical twin, there was a 24% concordance rate. Commenting on the much lower concordance rate in the Australian study, the authors addressed the sample bias in the previous studies and said, "In those studies, twins deciding whether to participate in a study clearly related to homosexuality probably considered the sexual orientation of their co-twins before agreeing to participate."[94]

The significance of the Australian twin study should not be underestimated. The suspected sample bias of the previous studies was proven to be true. Furthermore, the genetic correlation towards homosexuality is much weaker than initially suggested.[95] In 2010, the results were published of study focusing on homosexuality among twins in Sweden. The Swedish study found concordance rates for homosexuality among twins very similar to the Australian research.[96] All twin studies experience certain limitations when addressing behavioral traits. A trait with a genetic characteristic should show higher concordance in Monozygotic than in Dyzgotic twins, and even the data from the Australian and Swedish samples shows this. However, a higher concordance in MZ twins than in DZ twins does not *prove* a genetic effect. For starters, half of DZ twins are of different

[93] Bailey and Pillard, "Heritable Factors Influence Sexual Orientation in Women," 222.
[94] J. Michael Bailey, Michael P. Dunne, and Nicholas G. Martin, "Genetic and Environmental Influences on Sexual Orientation and Its Correlates in an Australian Twin Sample," *Journal of Personality and Social Psychology* 78.3 (March 2000): 533.
[95] The findings of another twin study based on data gathered from the United States was also published in 2000 which produced results similar to the earlier Bailey and Pillaard studies, but the sample was smaller than the Australian one with strong evidence of sample bias similar to Bailey and Pillard. *See* Kenneth S. Kendler, Laura M. Thornton, Stephen E. Gilman, Ronald C. Kessler, "Sexual Orientation in a U.S. National Sample of Twin and Nontwin Sibling Pairs," *American Journal of Psychiatry* 157.11 (November 2000): 1843 – 1846.
[96] Niklas Langström, Qazi Rahman, Eva Carlstöm, and Paul Lichtenstein, "Genetic and Environmental Effects on Same-sex Sexual Behavior: A Population Study of Twins in Sweden," *Archives of Sexual Behavior* 39 (2010):75 – 80.

sexes, whereas all MZ twins are of the same sex. Even if the comparison is restricted to same-sex DZ twins, at least *for behavioral traits* the argument can be made that MZ twins are more likely to look similar, to be dressed and treated the same, and thus to share more of their common environment than DZ twins.[97] At the same time, the free choices of human beings are also contributing factors in the dispositions that people develop.[98]

V. Homosexuality and Genetics

While twin studies have attempted to trace the heritability of homosexuality via family histories, other studies have examined DNA itself in an attempt to prove genetic etiology for homosexual behavior. The most discussion has been about an area known as Xq28.

What is Xq28?

Xq28 is *not* a gene, but it is a section of the X chromosome which contains many genes and is well-known among geneticists for its gene density. While the Xq28 region only covers approximately 5% of the X chromosome, it contains almost 13% of the X chromosomal genes. The Xq28 region comprises 180 genes, of which 28 are associated with a phenotype (the observable physical or biochemical characteristics that are connected to a particular gene).[99] The gene density of this area is seen in the fact over 40 different diseases have been traced to abnormalities in the Xq28 region.[100] The X chromosome itself contains about 155

[97] This is a summary from Tom Strachan and Andrew Read, *Human Molecular Genetics*, 4th ed. (New York: Garland Science, 2011), 470. In context, Strachan and Read are not discussing homosexuality in particular.

[98] In his critique of astrology, Augustine points out that astrology cannot be deterministic of one's character since twins are born at the same time "astrologically," but often have quite different temperament and character. Augustine then says, "[Hippocrates] would say that because of differences of food and exercise (matters depending not on bodily constitution, but on the mind's free choice) they [twins] might very well experience different states of health." Augustine, *The City of God Against the Pagans,* vol. 2, The Loeb Classical Library, William Green, trans. (Cambridge, MA: Harvard University Press, 1963), 141 – 143, V.ii.

[99] B. Auber, P. Burfeind, C. Thiels, E.A. Alsat, M. Shoukier, T. Liehr, H. Nelle, I. Bartels, G. Salinas-Riester, F. Laccone, "An Unbalanced Translocation Resulting In a Duplication of Xq28 Causes a Rett Syndrome-Like Phenotype in a Female Patient," *Clinical Genetics* 77 (2010): 593.

[100] Anja Kolb-Kokocinski, Alexander Mehrle, Stephanie Bechtel, Jeremy C. Simpson, Petra Kioschis, Stefan Wiemann, Ruthe Wellenreuther, and Annemarie Poustka, "The Systematic Functional Characteristic of Xq28 Genes Prioritizes Candidate Disease Genes," *BMC Genomics* 7.29 (February 17, 2006): 1 – 2.

million base pairs and represents approximately 5 percent of the total DNA in human cells.[101]

Each male inherits one X chromosome from his mother and a Y chromosome from his father. Studies relating to Xq28 and homosexuality focus on the X chromosome men receive from their mothers. Thus, when researchers claim to have found something connecting "Xq28" to homosexuality, they are referring to a gene-dense region on the X chromosome men inherit from their mothers.

Genetic Linkage Analysis

To date, research on Xq28 and homosexuality has utilized a method known as Genetic Linkage Analysis. This method uses several DNA sequence polymorphisms (normal variants) that are near or within a gene of interest to track within a family the inheritance of a disease-causing mutation in that gene.[102] These identifiable polymorphisms are called "genetic markers."[103] A genetic marker can be compared to using a landmark when giving directions to an out of town friend. For example, if you want to attend Roswell Street Baptist Church in the Atlanta area, I might tell you to "take highway 41 north until you reach the "Big Chicken" [a famous Atlanta landmark] and turn left." Once you arrived at the "Big Chicken," you wouldn't be at Roswell Street Baptist Church, but you would be close. In a similar way, genetic markers don't identify a specific gene, but they place us in a general region and we then know which genes are close by.

In genetics, linkage refers to the tendency for genes and other genetic markers to be inherited together because of their location near one another on the same chromosome. Stated most simply, genetic linkage analysis is a gene-hunting technique which has been used to locate genes responsible for various diseases.

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[101] Genetics Home Reference, "What is the X Chromosome," October 16, 2015, accessed October 30, 2015, http://ghr.nlm.nih.gov/chromosome/X.

[102] "Linkage Analysis," Genetics Home Reference, November 2, 2015, accessed November 9, 2015, http://ghr.nlm.nih.gov/glossary=linkageanalysis.

[103] A genetic marker is an identifiable segment of DNA with a known physical location on a chromosome and with enough variation between individuals that its inheritance and co-inheritance with alleles of a given gene can be traced; markers are used in linkage analysis. DNA segments close to each other on a chromosome tend to be inherited together. Markers are used to track the inheritance of a nearby gene that has not yet been identified but whose approximate location is known. The marker itself may be a part of a gene or may have no known function. Genetics Home Reference, "Marker," October 26, 2015, accessed October 30, 2015, http://ghr.nlm.nih.gov/glossary=marker.

Linkage analysis has been very successful in mapping many Mendelian traits and is at its most powerful when the phenotype is due to a single gene.[104] Linkage analysis is less effective in cases where not everyone who has a particular gene expresses the trait (incomplete penetration). The effectiveness of linkage analysis is also diminished when many different genes can lead to the same expression of a trait and when there are non-genetic forms of the disease.[105] Linkage analysis needs very few genetic markers to deduce the chromosomal region shared between affected individuals, but it cannot "find the gene" on its own. Once a region is narrowed down, researchers can then move forward with more specific genetic sequencing of the region in question.[106]

In exploring genetic links to homosexuality, researchers have used genetic linkage analysis in attempts to trace the source of a particular trait – homosexuality – in the same way that they have traced down the genetic origins of various diseases. This does not mean they consider homosexuality a disease, but a particular method used to find genes which cause disease have been applied to the behavioral trait of homosexuality.

"Inherited" versus "Heritability"

What does it mean to say a particular trait is "inherited" or, more specifically, that one "inherits" homosexuality? Though the popular media touts the concept that homosexuality is "genetic" or "inherited," the somewhat straightforward proposition that there is some gene ("x") for homosexuality could have one of many meanings:

1. That everyone possessing gene "x" will definitely be homosexual.
2. That only those possessing gene "x" could possibly be homosexual.
3. A combination of (1) and (2) such that homosexuality will be apparent if and only if the person has gene "x."
4. That there seems to be some sort of statistical correlation between having gene "x" and being homosexual.

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[104] M. Dawn Teare and Mauro F. Santibanez Koref, "Linkage Analysis and the Study of Mendelian Disease In the Era of Whole Exome and Genome Sequencing," *Briefings in Functional Genomics* 13.5 (September 2014): 379.

[105] Ibid.

[106] Ibid.

5. There is some sort of statistical correlation between particular regions of DNA containing many genes and homosexuality. [107]

The preponderance of data to date supports something like proposition 5: there is possibly some correlation between certain regions of the human genome and homosexual behavior. In contrast, the cultural mindset asserts something very similar to proposition 1 even though *no gay gene has been found.*

Much of the misunderstanding about a purported "gay gene" emerges from confusion about the vital distinction between a trait being "inherited" and a trait's "heritability." To say a trait is "inherited" means it has no connection with choice, that it is completely predetermined, and – except in unusual circumstances – it cannot be prevented. For example, having five toes on each foot is an inherited trait. Some traits are directly related to one specific gene. For example, whether you have a straight hairline or a widow's peak is determined by one gene. Yet most traits are not the simple result of just one gene. Instead, they are the result of the interactions between several genes.

To say that a trait is "heritable" is not the same thing as saying the trait is "inherited." "Heritability" is a term used to describe the complex interaction between genes and environment which results in many traits we express. For example, someone may have a genetic predisposition to be taller than normal. However, if a child with this genetic trait is raised in a war-torn country in which his diet lacks essential nutrients, he will not grow as tall as he would have under better conditions. Both his genetics and his environment contribute to the final height he reaches in adulthood. Usually, heritable traits are those which demonstrate a lot of variation within the population as a whole. For example, someone who is two inches shorter than average is still within the normal deviation for height in a particular community. In contrast, a child born with three toes would be considered abnormal.

One definition for heritability is the extent to which individual genetic differences contribute to observed behavior. The degree to which a trait is heritable is typically measured on a scale of 0.0 (genes make no contribution to individual differences) to 1.0 (genes are completely responsible for individual differences). Furthermore, it is important to know that when scientists discuss a trait's heritability, they are talking about the degree to which a trait varies within the

[107] This list of possibilities is modified from Ozan Onay's discussion of criminality and genetics in "The True Ramifications of Genetic Criminality Research for Free Will in the Criminal Justice System," *Genomics, Society, and Policy* 2.1 (2006): 81. Option 5 is added by me.

population as a whole, and not an individual person. For example, if someone says depression has a heritability of .40, they are claiming that, on average, about 40% of the individual differences that we observe in depression may in some way be attributable to genetic individual difference. It does not mean that 40% of any specific individual's depression is due to his or her genes and the other 60% is due to his or her environment. For human behavior, almost all estimates of heritability are in the moderate range of .30 to .60.[108] Keeping the concepts of inherited and heritable distinct from each other is important to understanding born-this-way arguments and genetics.

Dean Hamer and Colleagues–1993

The first study claiming to discover a specific DNA link to homosexuality was published in the July 16, 1993 edition of *Science* and asserted a region on the X chromosome in males was associated with homosexuality. The primary author was Dean Hamer, an American geneticist who worked as a researcher for the National Institutes of Health for thirty-five years where he was director of the Gene Structure and Regulation section at the National Cancer Institute. Hamer and his colleagues claimed to find "a statistically significant correlation between the inheritance of genetic markers on chromosomal region Xq28 and sexual orientation in a selected group of homosexual males."[109] In the years following this article's publication it has been widely cited as evidence of a "gene" for homosexuality and is a popular component of born-this-way arguments. When the average person mentions that scientists have found a "gay gene," they usually have "Xq28" in mind.[110]

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[108] The information summarized here about inherited versus heritability is a composite from "Heritability: Introduction," accessed June 10, 2015, http://psych.colorado.edu/~carey/hgss/hgssapplets/heritability/heritability.intro.html; Razib Khan, "Genetic versus heritable Trait," *Discover,* August 30, 2007, accessed June 9, 2015, http://blogs.discovermagazine.com/gnxp/2007/08/genetic-vs-heritable-trait/#.VXh-IUa-Pwc; John S. Feinberg and Paul D. Feinberg, *Ethics for a Brave New World*, 2nd ed. (Wheaton, IL: Crossway, 2010), 366 – 367.

[109] Dean Hamer, Stella Hu, Victoria L. Magnuson, Nan Hu, and Angela M.L. Pattatucci, "A Linkage Between DNA Markers on the X Chromosome and Male Sexual Orientation," *Science* 261 (July 16, 1993): 321.

[110] The issue of *Science* immediately following publication of Hamer's research included an article critical of Hamer's methods and conclusions. N. Risch, E. Squires-Wheeler, and J.B.K. Bronya, "Male Sexual Orientation and Genetic Evidence," *Science* 262 (December 1993): 2063–65.

Hamer and his colleagues gathered their data by investigating 114 families of homosexual men. 76 of the families were recruited from two Washington, DC area sources: An HIV outpatient center operated by the National Institutes of Health Clinical Center in Washington, DC and local pro-homosexual organizations in the Washington, DC area. The other 38 families were recruited via advertisements in pro-homosexual publications. Preliminary research into the family histories of homosexual men in their sample indicated increased rates of homosexual orientation in the maternal uncles (the mothers' brothers) and male cousins through maternal aunts (the mothers' sisters). Thus, Hamer and his team were curious to know if any genetic markers consistent with male homosexuality were common in homosexual sons via the X chromosome inherited from their mothers. They suspected that in some cases there might be a gene or genes inherited from mothers that predisposed their sons to homosexuality.

From their original sample of 114 families, the researchers selected a sub-group of 40 families in which there were two gay brothers, no more than one gay sister, and no indication of paternal transmission of homosexuality. From this select population, Hamer claimed 33 out of 40 (82.5%) pairs of homosexual brothers had co-inherited (or "shared") genetic information from their mothers in the Xq28 region. Specifically, they identified five markers in the Xq28 region which the 33 pairs of brothers had inherited exclusively from their mother. Hamer concluded, "We have now produced evidence that one form of male homosexuality is preferentially transmitted through the maternal side and is genetically linked to chromosomal region Xq28."[111] He also added "it appears that Xq28 contains a gene that contributes to homosexual orientation in males."[112]

Stella Hu, Dean Hamer and Colleagues – 1995

In 1995, Hamer's team conducted new research expanded their work to include lesbians as well. In this new sample, they did not discover any link between Xq28 and female homosexuality. But they did claim to replicate some of the findings regarding males and Xq28, but at a much lower level of statistical significance: In 1993, 33/40 pairs of gay brothers (82.5%) co-inherited the distinct markers while in 1995 22/33 pairs of gay brothers (67%) co-inherited the distinct markers.[113] The 1995 study was also interesting because it documented some non-

[111] Dean Hamer, et al, "A Linkage Between DNA Markers on the X Chromosome and Male Sexual Orientation," 325.

[112] Ibid, 326.

[113] In 1993, Hamer asserted an LOD score of 4.02 for Xq28.

homosexual men who shared the Xq28 marker with a homosexual brother. In other words, both brothers shared the marker at Xq28, but only one of them was homosexual. The authors thus concluded, "Even within the selected population that was studied, the Xq28 region was neither necessary nor sufficient for a homosexual orientation."[114] Finally, it is important to remember that Hamer himself is not claiming that the Xq28 findings explain all forms of homosexuality, but is specifically related to cases involving maternal heritability.

Understanding what Hamer claimed can be confusing. First, as noted earlier, Xq28 is *not* a gene, but it is a section of the X chromosome which contains many genes and is well-known among geneticists for its gene density. Hamer and his colleagues were *not* claiming to have identified a specific "gay" gene, but they were asserting such a gene or genes may exist in the Xq28 region.[115]

Furthermore, not all of the men in Hamer's study shared the exact genetic sequence. When the average person reads the popular reports regarding Xq28, they assume all 66 men from the 33 pairs of gay brothers in his 1993 study and all 44 men from the 22 pairs of gay brothers in his 1995 study had exactly the same DNA sequence at chromosome Xq28. This is not what Hamer claimed. As was noted in *The Hastings Center Report*, "In fact, all [Hamer] showed was that each member of the thirty-three concordant pairs shared his Xq28 region with his brother but not with any of the other sixty-four men. No single specific Xq28 sequence was common to all sixty-six men."[116] The sequence of genetic information at Xq28 was different among each of the pairs of brothers. What was common was the *location* of the sequences.

In the years following Hamer's research, several attempts to replicate his findings were unsuccessful. An unpublished 1998 study did not find significant Xq28 linkage for homosexuality in 54 pairs of homosexual brothers from the

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[114] Stella Hu, Angela M.L. Pattatucci, Chavis Patterson, Lin Li, David W. Fulker, Stacey S. Cherny, Leonid Kruglyak, and Dean H. Hamer, "Linkage Between Sexual Orientation and Chromosome Xq28 in males but not in females," *Nature Genetics* 11 (1995): 253. Hu and Hamer also said, "Therefore it is highly unlikely that any single genetic variation or allele will be present in all homosexual individuals or absent from all heterosexual individuals." Ibid., 252.

[115] In 1993, Hamer said, "Rather, it appears that Xq28 contains a gene that contributes to homosexual orientation in males." Hamer, et al, "A Linkage Between DNA Markers on the X Chromosome and Male Sexual Orientation," 325.

[116] Udo Schüklenk, Edward Stein, Jacinta Kerin, and William Byne, "The Ethics of Genetic Research on Sexual Orientation," *Hastings Center Report* 27.4 (July–August, 1997): 7.

U.S.[117] In 1999, Bailey, et al reported no definitive linkage between Xq28 and male homosexuality.[118] Also in 1999, Canadian researchers failed to replicate Hamer's findings, asserting that homosexual brothers are no more likely than their heterosexual brother to have the marker at Xq28.[119]

The difficulties in replicating Hamer's research may point to a fundamental weakness in the structure of his 1993 study: the lack of a control group. Anne Fausto-Sterling and Evan Balaban, two authors very favorable to homosexual rights, commented on Hamer's 1993 work and said:

> Despite our praise for aspects of Hamer, *et al.*'s work, we feel it is also important to recognize some of its weaknesses. The most obvious of these is the lack of an adequate control group. Their study demonstrates cosegregation of a trait (which Hamer, *et al*. have labeled "homosexuality") with X chromosome markers and the trait's concordance in homosexual brothers. This cosegregation is potentially meaningful if the mother is heterozygous for the trait. In this case, segregating chromosomes without the markers should show up in nonhomosexual brothers, but Hamer, *et al* present no data to that effect.[120]

With this in mind, it is of interest that when Hamer did include some controls in his 1995 study, the statistical significance of his findings dropped significantly.[121]

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[117] Sanders, A.R., Q. Cao, J. Zhang, J.A. Badner, L. R. Goldin, J.J. Guroff, E.S. Gershon, P.V. Gejman, "Genetic Linkage Study of Male Homosexual Orientation. Poster Presentation at the 151st Annual Meeting of the American Psychiatric Association. Toronto, Ontario, Canada, 1998; Cited in Khytam Dawood, Michael Bailey, and Nicholas G. Martin, "Genetic and Environmental Influences on Sexual Orientation," in *Handbook of Behavioral Genetics*, Yong-Kyu Kim, ed. (New York: Springer Science Media, 2009), 273.

[118] J. M. Bailey, R.C. Pillard, K. Dawood, Michael B. Miller, Lindsay A. Farrer, Shruti Trivedi, and Robert L. Murphy, "A Family History Study of Male Sexual Orientation Using Three Independent Samples," *Behavior Genetics* 29.2 (1999): 79–86.

[119] George Rice, Carol Anderson, Neil Risch and George Ebers, "Male Homosexuality: Absence of Linkage to Microsatellite Markers at Xq28," *Science* 284 (April 23, 1999): 665–67.

[120] Anne Fausto-Sterling and Evan Balaban, "Genetics and Male Sexual Orientation," *Science* 261 (September 1993): 1257.

[121] Hamer was actually accused by a research assistant of intentionally omitting data from his 1993 study which was inconsistent with the claim that Xq28 was connected with homosexuality. *See* Eliot Marshall, "NIH's Gay Gene Study Questioned," *Science* 268 (June 30, 1995): 1841. Hamer was cleared by the Office of Research Integrity in 1997. *See* J. Kaiser, "No Misconduct in "Gay Gene" Study," *Science* 275 (February 28, 1997): 1251.

As was noted by Hamer and his colleagues in 1995, their findings did not prove that a chromosomal pattern at Xq28 is either necessary or sufficient to cause homosexuality. Evangelical authors Jones and Yarhouse comment on this and say:

> If [Xq28] was necessary to the homosexual condition, then [Hamer, et al] would not have found the 7 out of 40 homosexual brother pairs who did not share this characteristic (these 7 brothers did not have the chromosomal pattern but were gay anyway). If it was sufficient to cause homosexuality, then they would not have found, in their second study, nonhomosexual brothers who shared the genetic characteristic but not the sexual orientation. Having the genetic marker does not mean you are a homosexual (not sufficient), and not having the genetic marker does not mean you are a homosexual (not necessary).[122]

A more restrained analysis of Hamer's research would say that the Xq28 region *may possibly* contain genes with a role in sexual orientation, but that role, if it exists, is unclear and the association between homosexuality and Xq28 is not nearly as strong as Hamer initially proposed in 1993.[123] In spite of weaknesses in Hamer's research, the inaccurate idea of a "gay gene" has taken on a life of its own and is now commonly accepted by many people as proof homosexuals are "born this way."

Mustanski, et al.–2005

A 2005 study led by Brian Mustanski of Northwestern University's Feinberg School of Medicine also failed to replicate Hamer's findings concerning Xq28. The sample for this study was drawn from 73 families previously studied in Hamer's 1993 and 1995 reports along with 73 new families which had never been researched. When all the families were taken together, it resulted in a sample of 456 individuals from 146 unrelated families, of which 137 families had 2 gay brothers and 9 families had 3 gay brothers. Instead of focusing only on the Xq28 region, this study was a genome-wide scan, meaning all 46 chromosomes were examined. Studying the entire sample of 146 families, no statistical significance associated with chromosome Xq28 was discovered, thus Hamer's earlier findings were not confirmed.

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[122] Jones and Yarhouse, *Homosexuality: The Use of Scientific Research in the Church's Moral Debate*, 81.

[123] My comments here are influenced by Gareth Jones, "A Neurobiological Portrait of the Human Person," *What About the Soul? Neuroscience and Christian Anthropology,* Joel B. Green, ed. (Nashville: Abingdon Press, 2004), 44.

The 2005 study did report three new regions of genetic interest concerning male homosexuality on chromosomes 7, 8, and 10. But a careful reading of the report shows a weak linkage between male homosexuality and these chromosomes. The researchers' strongest finding was on chromosome 7 at 7q36. However, Mustanski and colleagues admitted that the connection they found falls just short of "criteria for genomewide significance."[124] They went on to say that certain regions on chromosomes 8 and 10 "approached criteria for suggestive linkage."[125] Their report shows that none of the new data met professionally accepted criteria for statistical significance. The weakness of the findings make it frustrating that the University of Illinois at Chicago issued a press release about the study saying Mustanksi "has identified several areas that appear to influence whether a man is heterosexual or gay." In fact, the press release is vast overstatement: The genome-wide scan had low resolution and did not find any region with a statistically significant linkage using established criteria.[126] Simon LeVay summarizes Mustanki's study and says, "The statistical power of these findings was low, however, and the findings should be thought of as pointers for future research rather than as actual identifications of regions containing 'gay genes.'"[127]

Ramagopalan, et al, 2010

In 2010, researchers associated with Oxford University and the University of Western Ontario examined 55 Canadian Caucasian families with two or more homosexual male siblings, a sample derived from Rice, et al's 1999 study. In light of Mustanksi's research in 2005, they wanted to examine the genome beyond the X chromosome. They could not replicate Mustanksi's findings and suggested that genes contributing to

[124] Brian S. Mustanski, Michael G. DuPree, Caroline M. Nievergelt, Sven Bocklandt, Nicholas, J. Schork, Dean H. Hamer, "A Genomewide Scan of Male Sexual Orientation," *Human Genetics* 116 (2005): 276.
[125] Ibid.
[126] Fernando Saravi, "The Elusive Search for a 'Gay Gene,'" in *Tall Tales About the Mind and Brain: Separating Fact From Fiction*, Sergio Della Sala, ed. (New York: Oxford University Press, 2007), 470.
[127] LeVay, *Gay, Straight, and the Reason Why*, 172.

homosexuality, "if they do exist" actually exert "more modest effects than detectable by linkage."[128]

Summary of Xq28 Research Prior to 2015

Beginning in 1993, several studies were conducted regarding male homosexuality and Xq28. The results can be summarized as follows:

1993, Hamer: Claimed a linkage between Xq28 and homosexuality in 33 of 40 pairs of homosexual brothers.

1995, Hamer / Hu: Claimed a linkage was further supported, but at a lower level than 1995 (22 of 33 brother pairs). No linkage found for females.

1998, Sanders, et al: No Xq28 linkage supported. 54 brother pairs.

1999, Bailey, et al: No significant X128 linkage discovered.

1999, Rice, et al: No Xq28 linkage supported. 52 brother pairs.

2005, Mustanski: No Xq28 linkage supported. Some interest at 7q36. 73 brother pairs.

2010, Ramagopolan, et al: Unable to replicate Mustanksi's findings regarding chromosome 7.

Sanders/Bailey 2015: Hamer's Research Confirmed?

In 2015, Alan Sanders and Michael Bailey, also of Northwestern research which claims to have replicated Hamer's findings concerning homosexuality and the Xq28 region in addition to an area of interest at chromosome region 8q12 which was previously identified by Mustanski in 2005.[129] In the largest study to date on the topic, Sanders and Bailey examined the genetics of 908 individuals from 384 different families, with special emphasis on 409 pairs of homosexual

[128] Sreeram V Ramagopalan, David A Dyment, Lahiru Handunnetthi, George P Rice and George C. Ebers, "A Genome-Wide Scan of Male Sexual Orientation," *Journal of Human Genetics* 55 (2010): 132.

[129] Sanders and Bailey began announcing their findings in late 2013 and early 2014.

brothers in their sample. 21 of the families actually had three homosexual brothers and two families had four homosexual brothers, leaving an actual number of 793 homosexual males in their sample. Much like Hamer, they were searching for families which had a large number of homosexuals on the mother's side. The researchers concluded they had found significant linkage in chromosomal regions 8q12 and Xq28 and said, "In context with the previous linkage scans, it seems likely that genes contributing to variation in male sexual orientation reside in these regions."[130] The authors also added, "While our study results provide further evidence for early (prenatal) biological influences on variation in male sexual orientation, we also emphasize that genetic contributions are far from determinant but instead represent a part of the trait's multifactorial causation, both genetic and environmental."[131]

While the new research from Sanders and Bailey is intriguing, it must be stressed that in their own data only 8q12 met the standard criteria for statistical significance. In genetics, an LOD score ("logarithm of the odds") is a standard way of measuring data. Typically, a score of 3.0 or higher is deemed to indicate significant linkage. In Sanders and Bailey's research, 8q12 had an LOD score of 4.08 while Xq28 had an LOD score of 2.99. Sanders himself admits data from Xq28 does not clear the threshold for significance.[132] Why then do they emphasize they have found something at Xq28? Because some of their other data clustered in regions neighboring Xq28. Other researchers also question the strength of these new findings. Neil Risch, a statistical geneticist at the University of California, San Francisco believes the Sanders and Bailey data are statistically too weak to demonstrate any genetic link.[133] Another way of stating the data is to

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[130] A.R. Sanders, E.R. Martin, G.W. Beecham, S. Guo, K. Dawood, G. Rieger, J.A. Badner, E.S. Gershon, R.S. Krishnappa, A.B. Kolunddzija, J. Duan, P.V. Gejman, and J.M. Bailey, "Genome-Wide Scan Demonstrates Significant Linkage for Male Sexual Orientation," *Psychological Medicine* 45 (2015): 1384. Mustanski identified the 8p12 region as an area of interest regarding homosexuality, but his findings regarding this area did not reach the standard for statistical significance. Sanders, et al say 8q12 and 8p12 "overlap."

[131] Ibid., 1386.

[132] Kelly Servick, "Study of Gay Brothers May Confirm X Chromosome Link to Homosexuality," *Science,* November 17, 2014, accessed March 2, 2015, http://news.sciencemag.org/biology/2014/11/study-gay-brothers-may-confirm-x-chromosome-link-homosexuality.

[133] "A Large Study of Gay Brothers Adds to Evidence That Genes Influence Men's Chances of Being Homosexual, But the Results Aren't Strong Enough to Prove It," *The Associated Press*, November 17, 2014, accessed May 5, 2015, http://www.nydailynews.com/life-style/health/study-suggests-genes-influence-men-chances-gay-article-1.2013597.

say that not all of the men in the study shared something significant at either Xq28 or 8q12. In other words, co-inherited genetic information at either of these chromosomes is not necessary to cause homosexuality.[134]

Another limitation in the Sanders/Bailey research is their use of older methods which have been surpassed by newer and more precise techniques. Sanders and Bailey performed a genetic linkage study using an approach similar to Hamer in 1993. In genetics, a linkage study only identifies wide regions of a chromosome containing many genes. As used by Sanders/Bailey, a linkage analysis seeks to identify chromosomal segments shared by affected family members (expressing the trait of homosexuality), without having to specify exactly how any susceptibility factors carried on those shared segments contribute to the trait.[135] However, when dealing with diseases, linkage studies are generally not performed for non-Mendelian traits. In fact, standard LOD score analysis / Linkage Studies are usually inappropriate for non-Mendelian traits.[136] Since homosexuality is clearly *not* a Mendelian trait, it is imprudent to make global assertions about homosexuality based on linkage analysis alone. In contrast, the preferred method now is called a "genome wide association study," a technique which can often identify a specific gene responsible for a particular trait.[137] Sanders and Bailey indicated they wanted to do a linkage study in order to replicate Hamer's findings. They have indicated they will do a genome wide association study in the future. But their current data was derived from an older and less precise technique.

Sanders and Bailey also lack a sufficient control group, a weakness in Hamer's 1993 study as well. While they sampled data from 33 heterosexual brothers, this is an insufficient control group when compared to 793 homosexuals.

Bailey insists the new findings about Xq28 and 8q12 support a born-this-way argument and yet sounds contradictory when he says, "Sexual orientation has nothing to do with choice. . . . We found evidence for two sets [of genes] that affect whether a man is gay or straight. But it is not completely determinative;

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[134] This can be clearly seen by examining the plot diagrams on page 1385 of the Sanders/Bailey study. Sanders, et al, "Genome Wide Scan," 1385.

[135] My wording here is influenced by Tom Strachan and Andrew Read, *Human Molecular Genetics*, 4th ed. (New York: Garland Science, 2011), 473. Strachan and Read are referring to diseases.

[136] Ibid. In context, Strachan and Read are not discussing homosexuality, but the observations are quite relevant.

[137] Kelly Servick, "Study of Gay Brothers May Confirm X Chromosome Link to Homosexuality."

there are certainly other environmental factors involved."[138] Bailey sounds triumphant when he says sexual orientation "has nothing to do with choice," but this assertion seems confusing when compared to his follow-on statement that the genes in question "are not completely determinative." Perhaps part of the confusion is caused by Bailey's use of the term "environment." For born-this-way arguments, environmental influences include not only a person's family and culture, but the prenatal environment of the womb. It is not clear how Bailey is using the term here.

Sanders and Bailey somewhat cautiously suggest the genes in question may affect the development of the brain *in utero*. They comment, "As usual with linkage peaks for complex traits, there are a number of genes of potential relevance under each broad peak, such as transcription factors, microRNAs, and various brain-expressed genes including some with neurodevelopment, neuroendocrine, and/or neurotransmission."[139] In this way, Sanders and Bailey are connecting their research to the popular claim that an inordinate exposure to the wrong hormones *in utero* "feminizes" the brains of male homosexuals, thus explaining their same-sex attraction.[140] With this in mind, research regarding people with Disorders of Sexual Development (DSD), who are indeed exposed to hormones discordant for their sex in utero, demonstrates that people with DSDs do have a higher percentage of homosexuals than the average population. However, the data also indicates that

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[138] Ian Sample, "Male Sexual Orientation Influenced by Genes, Study Shows," *The Guardian,* February 14, 201, accessed April 23, 2014, http://www.theguardian.com/science/2014/feb/14/genes-influence-male-sexual-orientation-study. Simon LeVay apparently finds the new research convincing. In 2011, he said "Unfortunately, Hamer's report has not been robustly confirmed." Simon LeVay, *Gay, Straight, and the Reason Why: The Science of Sexual Orientation* (New York: Oxford University Press, 2011), 171. However, he comments on the new research and says, "This study knocks another nail into the coffin of the 'chosen lifestyle' theory of homosexuality." Andy Coghlan, "Largest Study of Gay Brothers Homes in on 'Gay Genes,'" *New Scientist,* November 17, 2014, accessed May 5, 2015, http://www.newscientist.com/article/dn26572-largest-study-of-gay-brothers-homes-in-on-gay-genes.html#.VVKNn_lViko.

[139] Sanders, et al, "Genome-Wide Scan Demonstrates Significant Linkage for Male Sexual Orientation," 1384.

[140] Likewise, it is claimed female homosexuals have "masculinized" brains, explaining their attraction to other females.

prenatal hormones are only a contributing factor in some cases and are not completely determinative.[141]

But Sanders and Bailey's own comments in their published research contradicts the idea they have found a particular gene or genes which cause a person to be born a homosexual. In their conclusion, they address concerns related to a hypothetical scenario where a parent with a strong animosity towards homosexuality might have a genetic test performed on a pre-born infant to determine if it would be homosexual. The fear among some homosexuals and their allies is that if such a test existed, then homosexual children would be aborted. But Sanders and Bailey say this fear is unwarranted because "the small magnitude of effects suggested herein are inconsistent with a test that those motivated to influence their children's sexual orientation would find useful."[142] They then conclude by saying, "While our study results provide further evidence for early (prenatal) biological influences on variation in male sexual orientation, we also emphasize that genetic contributions are far from determinant but instead represent a part of the trait's multifactorial causation, both genetic and environmental."[143] So the authors of the new study admit the purported influences they find are of a small magnitude and are not determinant. With these statements in mind, Bailey's claim that "sexual orientation has nothing to do with choice" seems perplexing, overstated, and inconsistent with his own research.

Sanders and Bailey's research is actually touching on the *heritability* of homosexuality as opposed to it being an *inherited* trait. Sanders himself commented, "When people say there's a gay gene, it's an oversimplification. There's more than one gene, and genetics is not the whole story. Whatever gene contributes to sexual orientation, you can think of it as much as contributing to heterosexuality as much as you can think of it contributing to homosexuality. It contributes to a variation in the trait."[144] Sanders has suggested 30 – 40% of the variation of the trait of male homosexuality can be connected with genetics.[145] As

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[141] See my paper delivered at the 2014 ETS meeting: ""Pro-Homosexual Arguments Regarding Congenital Adrenal Hyperplasia, Androgen Insensitivity Syndrome and Homosexuality as an Innate Trait."

[142] Sanders, et al, Genome-Wide Scan Demonstrates Significant Linkage for Male Sexual Orientation," 1386.

[143] Ibid.

[144] Ian Sample, "Male Sexual Orientation Influenced by Genes, Study Shows."

[145] Alan Sanders Interview, accessed April 24, 2014, https://www.youtube.com/watch?v=WANgHtb-NT8#t=29. The webpage home for the Sanders/Bailey study can be found at http://www.gaybros.com/index.html.

we saw earlier, virtually all human behavior falls into the range of 30 – 60% for heritability.

Bailey's claim that sexual orientation has "nothing to do with choice" is a statement reflecting his own personal worldview and not a necessary inference from his findings. Bailey is actually stating a version of biological determinism in which people are automatons driven on to their destiny by irresistible biological urges. Bailey seems willing to admit that environment plays a role in development of one's orientation, but is unwilling or unable to admit that our choices in response to our environment affect our own sexual desires and cravings. Ian Sample of *The Guardian* is favorable to homosexual rights, but even he sounded a more restrained note and said, "The gene or genes in the Xq28 region that influence sexual orientation have a limited and variable impact."[146]

To summarize, Sanders and Bailey found one area of interest at Chromosome 8q12 which reached the accepted criteria for statistical significance. They also claimed their data for Xq28 came very close to being statistically significant. However, there were homosexuals in their sample who did not share the markers at either region. Thus, a specific genetic link to either 8q12 or Xq28 is not necessary for homosexuality. By their own admission, the effects for specific areas of DNA are of a small magnitude and do not mean someone with a specific genetic pattern will inevitably become homosexual.

August, 2019: Hamer Refuted

The largest study to date regarding genetics and homosexuality was published in *Science* on August 30, 2019. These researchers used a sample of 477,522 people. Using the better and more precise genome wide association method, they discovered no gay gene. The study found no connection between the X chromosome and homosexuality, saying, "In contrast to linkage studies that found substantial association of sexual orientation with variants on the X-chromosome, we found no excess of signal (and no individual genome-wide significant loci) on the X chromosome."[147] This is a profound statement: Hamer's claims regarding Xq28, widely repeated for the previous 25 years, have no validity when a larger sample and more precise methods for doing research are used. This study found 5 single nucleotide polymorphisms of interest, but no gay gene.

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[146] Ibid.

[147] Andrea Ganna, Karin J.H. Verweij, et al, "Large-scale GWAS Reveals Insights into the Genetic Architecture of Same-sex Sexual Behavior," *Science* 365 (August 30, 2019), 3 – 4.

No one has discovered a gay gene. Genetics is a fabulously complex field and our knowledge of the mechanisms of inheritance have exponentially expanded in recent decades with new discoveries being made quite regularly. When we think of inheritance, we often think in terms of *Mendelian* traits, meaning a particular genotype at one locus on the genome is both necessary and sufficient for a trait to be expressed, given the normal range of human genetic and environmental backgrounds. But most human genetic or partly genetic traits *are not* Mendelian. They are governed by genes at more than one locus. The more complex the path between a DNA sequence and an observable trait, the less likely it is that the trait will show a simple Mendelian pedigree pattern.[148] The point of these facts for our discussion is that research to date *does not* indicate that homosexuality is a Mendelian trait; instead, genetics *may* play a part as a contributing factor, but they are not strictly causative for a homosexual identity. Thus, when people speak about a "gay gene," they are incorrectly oversimplifying findings to date.

VI. Homosexuality and Epigenetics

One of the most fascinating and burgeoning fields of scientific research is epigenetics. *Epigenetics* refers to chemical modifications of the human genome that alter gene activity without changing the DNA sequence. While many are familiar with arguments regarding *genetics* and homosexuality, epigenetics is now a growing focus of research into possible avenues of biological determination regarding sexual identity.

In October, 2015, Dr. Tuck Ngun of UCLA presented a paper at the annual meeting of the American Society of Human Genetics which suggested epigenetics may have a major influence on sexual orientation. Ngun claimed applying certain algorithms to data gathered from a specific sample of identical male twins allowed him to achieve a high degree of predictive accuracy regarding a person's sexual orientation based on DNA methylation patterns. In other words, he claimed to have discovered a fairly accurate method of determining if someone is a homosexual by merely examining the epigenome. Ngun's research is related to previous suggestions by researchers associated with the National Institute for Mathematical and Biological Synthesis and led by William Rice of the University of California, Santa Barbara. In 2012, Rice and his colleagues proposed epigenetics may explain the heritability of some forms of homosexuality. These claims are startling and debatable to some while they provide a satisfying

[148] My comments to this point in the paragraph are summarized from Tom Strachan and Andrew Read, *Human Molecular Genetics*, 4th ed. (New York: Garland Science, 2011), 62.

explanatory force to others. However, a review of current research into epigenetics demonstrates certain epigenetic tags may possibly be a contributing, but not a causative, factor in the development of a homosexual orientation.

Epigenetics

Epigenetics – a word with a rough literal meaning of "on genes" – refers to chemical modifications of the human genome that alter gene activity without changing the DNA sequence.[149] DNA is wrapped around proteins called histones and both DNA and the histones are covered with chemical "tags." These histones and chemical tags (or "epi-marks") are part of each person's *epigenetics* and constitute an extra layer of information attached to our genes' backbones that regulates their expression.[150] As science has discovered more and more about genetic traits, we have learned that these epigenetic structures regulate genome activity and govern which genes in the DNA of any given cell will be active. These epigenetic structures can be thought of as switches and knobs which turns things "on or off" or "up and down." Perhaps the most fascinating difference between DNA and epigenetics is that the genome does not change during cell division throughout a person's lifetime, but the epigenome can change.

A helpful analogy for understanding epigenetics is to think of actors reading a script for a movie. For example, Director Baz Luhrmann hands Leonardo DiCaprio his shortened version of Shakespeare's script for *Romeo and Juliet*, on which the director has written or typed various notes – such as directions for camera placements and other technical information. Whenever DiCaprio's copy of the script is photocopied, Luhrmann's additional information is copied along with it. Claire Danes, playing the part of Juliet, also has a script for Romeo and Juliet. While the notes on her copy are different from those on DiCaprio's, Danes' notes will also survive photocopying. Nessa Carey explains the analogy and says, "That's how epigenetic regulation of gene expression occurs – different cells have the same DNA blueprint (the original author's script) but carrying varied molecular

[149] Or stated slightly differently, epigenetics refers to all modifications to genes other than changes in the DNA sequence itself which alter gene expression. Joanna Downer, "Backgrounder: Epigenetics and Imprinted Genes," accessed April 24, 2014, http://www.hopkinsmedicine.org/press/2002/november/epigenetics.htm; and William B. Dobyns, Susan L. Christian, and Soma Das, "Introduction to Genetics," in *Swaiman's Pediatric Neurology*, 5th ed., vol. 1, *Principles and Practice*, Kenneth Swaiman, Stephen Ashwal, Donna M. Ferriero, and Nina Schor, eds. (New York: Elsevier, 2012), 277.

[150] "Study Finds Epigenetics, Not Genetics, Underlies Homosexuality," National Institute for Mathematical and Biological Synthesis, December 11, 2012, accessed April 24, 2014, http://www.nimbios.org/press/FS_homosexuality.

modifications (the shooting script) which can be transmitted from mother cell to daughter cell during cell division."[151]

Homosexuality and Epigenetics

Suggestions that homosexuality may have an epigenetic origin are rather recent. The two most well-known statements of this argument to date are from an article by William R. Rice and colleagues in 2012 and a paper delivered Dr. Tuck Ngun of UCLA presented at the annual meeting of the American Society of Human Genetics in October, 2015.

Rice, Gavrilets, & Friberg, 2012

In 2012, a team of researchers associated with the National Institute for Mathematical and Biological Synthesis and led by William Rice an evolutionary geneticist at the University of California, Santa Barbara, joined by Sergey Gavrilets, a mathematician at the University of Tennessee, and Urban Friberg, an evolutionary biologist at the University of Uppsala, suggested epigenetics may explain the heritability[152] of some forms of homosexuality. Published in December, 2012 in *The Quarterly Review of Biology*, they argued that epigenetic changes to the early embryo can affect the expression of genes related to androgen signaling which then influences later sexual orientation.

Two major theoretical premises undergird the claims of Rice and his colleagues. The first premise is that androgen levels drive sexual orientation in a manner similar to the way they drive the development of genitalia. The entire process of prenatal gender-specific growth is driven by the release of hormones at specific junctures. As children grow in the mother's womb, certain sex hormones are produced in quantity at specific times to help their tiny bodies grow in a gender-specific direction. Testosterone, an androgen, is especially important in this process. Both boys and girls produce testosterone, but testosterone production

[151] I got this analogy from Nessa Carey, *The Epigenetics Revolution*: *How Modern Biology is Rewriting Our Understanding of Genetics, Disease, and Inheritance* (New York: Columbia University Press, 2012), 55.

[152] "Heritability" is a term used to describe the complex interaction between genes and environment which results in many traits we express. For example, someone may have a genetic predisposition to be taller than normal. However, if a child with this genetic trait is raised in a war-torn country in which his diet lacks essential nutrients, he will not grow as tall as he would have under better conditions. Both his genetics and his environment contribute to the final height he reaches in adulthood. Usually, heritable traits are those which demonstrate a lot of variation within the population as a whole. For example, someone who is two inches shorter than average is still within the normal deviation for height in a particular community. In contrast, a child born with three toes would be considered abnormal.

peaks in male babies at around 16 weeks of gestation, but after this declines to around the same level as in prenatal females. Sexual development in females is also driven by hormones, or more specifically the absence of male hormones. Since girls do not have testes, not enough testosterone is produced to masculinize genitalia and, thus, the external genitalia develop in a female manner.[153] In humans, the process of sex determination and forming of the external genitalia is virtually complete by the 13th week of gestation.[154] The theory of Rice, et al assumes that androgens are also central to the development of sexual orientation. Mainly, they argue that homosexuals received the correct hormones to guide their genitalia in proper development, but later in prenatal development – especially in the brain – they received the incorrect level of hormones or the wrong hormones, resulting in a homosexual orientation.[155]

A second major premise is that a mother or father could pass down the wrong epigenetic marks to their children. Usually, epigenetic "tags" or "marks" develop very early soon after conception. The parents' epigenetic tags are erased and replaced by unique ones for the child. But if epigenetic marks that direct sexual development are not erased correctly, a mother could pass down epi-marks consistent with female development to her son, resulting in an attraction to men, and vice versa for a father and his daughters.[156] In other words, a young fetus

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[153] Since females do not have the SRY gene, the primitive gonads become ovaries and not testes. Female ovaries actually produce a small amount of testosterone. Both males and females produce testosterone and estrogen, but males produce far more testosterone and females produce far more estrogen.

[154] Margaret M. McCarthy, "Estradiol and the Developing Brain," *Physiological Review* 88.1 (January 2008): 91 – 124, accessed July 9, 2014, http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2754262/pdf/nihms117872.pdf, 7.

[155] The authors say, "The androgen signaling pathways differ among organs and tissues, the same inherited sexually antagonistic-epi mark can affect only a subset of sexually dimorphic traits, e.g., no effect on the genitalia, but a large effect on a sexually dimorphic region of the brain." William R. Rice, Urban Friberg, and Sergey Gavrilets, "Homosexuality As A Consequence of Epigenetically Canalized Sexual Development," *The Quarterly Review of Biology* 87.4 (December 2012): 358. This central premise – that androgens are in fact the driving factor in the development of sexual orientation – has not yet been proven. Eric Vilain, the lab supervisor for Tuck Ngun, agrees with the basic trajectory of Rice's model. Vilain acknowledges that girls with Congenital Adrenal Hyperplasia were exposed to very high levels of androgens in utero and have masculinized genitalia and report a higher incidence of same-sex attraction. But he then adds, "It remains to be seen whether smaller variants of testosterone that do not result [in masculinized genitalia] also lead to attraction of same sex partners." Vilain does not agree with my moral stance regarding homosexuality. *See* Sabrina Richards, "Can Epigenetics Explain Homosexuality?," *The Scientist* January 1, 2013, accessed August 9, 2016, http://www.the-scientist.com/?articles.view/articleNo/33773/title/Can-Epigenetics-Explain-Homosexuality-/. Elsewhere, Vilain and Ngun concur with one of Rice's core assertions and say, "We believe it is very likely that sex-specific epigenetic marks are at least (partly) responsible for sexually dimorphic traits including sexual orientation." Tuck Ngun and Eric Vilain, "The Biological Basis of Human Sexual Orientation: Is There a Role for Epigenetics?," *The Epigenetic Shaping of Sociosexual Interactions: From Plants to Humans* 86 (2014): 175.

[156] Sabrina Richards, "Can Epigenetics Explain Homosexuality?"

inherits epigenetic marks that are not consistent with the baby's gender. They then hypothesize these sexually-antagonistic (opposed to the child's gender) epigenetic marks "influence androgen signaling in the part of the brain controlling sexual orientation, but not the genitalia nor the brain region(s) controlling gender identity."[157] In other words, the epigenetics cause a child to process the wrong sex hormones or sex hormones in the wrong amounts into the brain. Thus, they hypothesize this causes the child to experience same-sex attraction as he or she matures. But determining whether or not these proposed epigenetic marks affecting sexual orientation exist has not been proven. Furthermore, to prove they have not been erased will be difficult to test because such marks, if they exist, will probably be in the brain.[158]

Rice, et al make a fascinating admission and say, "*Although we cannot provide definitive evidence that homosexuality has a strong epigenetic underpinning*, we do think that available evidence is fully consistent with this conclusion."[159] While they admit they cannot provide definitive evidence, they say in their conclusion, "If our model is wrong, it can be rapidly falsified and discarded."[160] The work by Rice and his team is a specific type of academic research called "meta-analysis, a quantitative, formal study design used to assess systematically previous research studies in order to derive conclusions about a particular body of research.[161] Such work is also called a *review* article, meaning it is an article that synthesizes other research already in print and suggests possible implications. Meta-analysis is often the first step in defining avenues for future research by summarizing what has been done, what conclusions have been reached, and providing suggestions for future research. Essentially, Rice, et al. are saying to other researchers, "Hey, you might look over here." In 2015, Tuck Ngun claimed to have discovered some findings quite consistent with the Rice model for epigenetics and homosexuality.

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[157] William R. Rice, Urban Friberg, and Sergey Gavrilets, "Homosexuality as a Consequence of Epigenetically Canalized Sexual Development," 358.
[158] This is Vilain's opinion. *See* Sabrina Richards, "Can Epigenetics Explain Homosexuality?" But again, Vilain finds a great deal of Rice's work compelling.
[159] William R. Rice, et al, "Homosexuality as a Consequence of Epigenetically Canalized Sexual Development," 357. Emphasis added.
[160] Ibid., 362.
[161] This definition is from A. B. Haidich, "Meta-Analysis in Medical Research," *Hippokratia* 14 (2010): 29.

Tuck Ngun, 2015

On October 8, 2015, Tuck Ngun, a post-doctoral scholar in the Department of Genetics at UCLA's David Geffen School of Medicine, presented a paper titled "A Novel Predictive Model of Sexual Orientation Using Epigenetic Markers" at the annual meeting of The American Society of Human Genetics. The lead researcher was Eric Vilain (Ph.D., M.D.), associate professor and Chief of the Division of Medical Genetics at UCLA.[162]

Ngun and Vilain published a paper in 2014 in which they evaluated and critiqued the epigenetic model proposed by Rice and colleagues in 2012. Ngun and Vilain agreed with much of Rice's model, but disagreed that "sex-reversing sensitivity to androgen signaling via epigenetic markers will result in homosexuality in both sexes."[163] Ngun and Vilain reject this premise because they think the different biological and genetic factors affect homosexuality in men and women.[164] Essentially, they argue there are different types of homosexuality while Rice, et al appear to be striving at a model which is universally applicable to all homosexuals. Nonetheless, the suggestions of Rice, Friberg, and Gavrilets gave a trajectory for the research by Ngun and Vilain.[165]

Ngun claimed an algorithm his team developed can predict sexual orientation in males at a rate of 67% accuracy using epigenetic information from

[162] "Eric Vilain, M.D., Ph.D.," David Geffen School of Medicine, accessed August 10, 2016, https://people.healthsciences.ucla.edu/institution/personnel?personnel_id=9435. Ngun presented his findings earlier on March 21, 2015 in Philadelphia at the meeting of the Society for Research in Child Development. Accessed October 10, 2016, https://gendercenter.genetics.ucla.edu/node/75.
[163] Tuck Ngun and Eric Vilain, "The Biological Basis of Human Sexual Orientation: Is There a Role for Epigenetics?," 175.
[164] Part of their reasoning is based on claims related to Xq28 and homosexuality in males while no genetic region whatsoever has been connected to homosexuality in females. I critiqued claims related to Xq28 in a 2015 paper delivered at ETS, "Xq28 and Homosexuality: An Update on Current Research."
[165] Ngun and Vilain said, "Rice, Friberg, and Gavrilets (2013) have proposed steps to test their epigenetic hypothesis. Our group is currently testing the hypothesis that discordance in sexual orientation between [monozygotic] twins is related to discordance in epigenetic traits." [165] Tuck Ngun and Eric Vilain, "The Biological Basis of Human Sexual Orientation: Is There a Role for Epigenetics?," 178.

five to nine regions of the human genome.[166] The data was generated using a sample composed of DNA derived from the saliva of 37 pairs of identical twins who were discordant for sexual orientation (one was homosexual and one was not) along with a control group of 10 pairs of identical twins who were concordant for homosexuality (both were homosexual). Ngun and his colleagues looked for epigenetic modifications made to the genes of the 47 sets of male twins. Specifically, they analyzed 140,000 regions in the genomes of the twins and looked at 400,000 methylation marks, which can be thought of as "chemical Post-It notes" that dictate when and where genes are activated.[167] The team then used an algorithm they developed to search out gene regions in which methylation patterns differed significantly between the two groups. They found five sites of particular interest– three in regions of intergenic DNA, the role of which is unclear, and two in genes whose roles are relatively well established. One of the genes the Ngun team identified as having epigenetic changes is involved with the production of MHC II molecules which are important for a healthy immune system, but are also thought to affect sexual attraction by affecting response to odor.[168]

The Vilain-Ngun team then split their sample of 37 discordant twin pairs into two groups. Using the test results from 20 of these pairs, they developed a model to predict if a person in one of the seventeen remaining pairs is straight or gay based on the methylation patterns of their genes. When they tested their model on the remaining pairs of male twins using their algorithm, they claimed it correctly predicted sexual orientation 67 per cent of the time.

In their 2014 article, Ngun and Vilain suggested that slight variations in the uterine environment may explain why some twin pairs are discordant for homosexuality. For example they suggested the twins may receive different

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[166] T. C. Ngun, W. Guo, N. M. Ghahramani, K. Purkayastha, D. Conn, F. J. Sanchez, S. Bocklandt, M. Zhang, C. M. Ramirez, M. Pellegrini, Eric Vilain, "Program Number 95: A novel predictive model of sexual orientation using epigenetic markers." A Paper Delivered October 8, 2015 at the Annual Meeting of The American Society of Human Genetics, accessed February 26, 2106, https://ep70.eventpilotadmin.com/web/page.php?page=IntHtml&project=ASHG15&id=150123267. One of the frustrating aspects of Ngun's research is that the paper has not been published. I personally wrote to Ngun via the USPS asking for a copy, but received no reply.

[167] Ed Yong, "No, Scientists Have Not Found the 'Gay Gene,'" *The Atlantic* October 10, 2015, accessed July 15, 2016, http://www.theatlantic.com/science/archive/2015/10/no-scientists-have-not-found-the-gay-gene/410059/.

[168] Much of the information in this summary is found in Jessica Hamzelou, "Gay or Straight? Saliva Test Can Predict Sexual Orientation," *New Scientist* October 8, 2015, accessed August 10, 2016, https://www.newscientist.com/article/dn28307-gay-or-straight-saliva-test-can-predict-male-sexual-orientation/. *See also* Claus Wedekind and Dustin Penn, "MHC Genes, Body Odours, and Odour Preferences," *Nephrology, Dialysis, and Transplantation* 15.9 (2000): 1269 – 1271.

nutrients even though they share the same uterus, saying, "Although the nutrient bath in which both twins develop may be highly similar, there could be differences that could affect epigenetic markers on genes relevant to sexual orientation."[169]

The burgeoning field of epigenetics has provided a new avenue of research for people seeking a biological basis for homosexuality. Does Ngun's research into epigenetics provide compelling reasons to believe homosexuality is an innate trait caused by epigenetic modifications?

Evaluation

An evaluation of the data regarding homosexuality and epigenetics reveals some fascinating insights into the way we as humans function and the manner in which the human genome expresses particular traits. However, data to date does not substantiate the claim that prenatal epigenetic changes "hard-wire" someone for homosexuality. To demonstrate this claim, I will review some of the data about epigenetics in general, Ngun's research in particular, data regarding epigenetics and drug addictions, and then move to a Scriptural-theological evaluation of the data.

The data presented by Ngun[170] in October, 2015 has received a fair amount of criticism from others in the research community. The fact that the report does not appear headed to publication is further confirmation of a rather lukewarm reception to his findings. The most glaring problem with the study is its size: the sample is tiny. Ed Yong of *The Atlantic* comments on this weakness in the Ngun paper and says, "The field of epigenetics is littered with the corpses of statistically underpowered studies like these, which simply lack the numbers to produce reliable, reproducible results."[171] Furthermore, remember that the team split their sample into two sets: One was a "training set" whose data they used to build their algorithm, and a "testing set" whose data they used to verify it. While this is standard practice in research, Ed Yong says the result here is to weaken further this

[169] Tuck Ngun and Eric Vilain, "The Biological Basis of Human Sexual Orientation: Is There a Role for Epigenetics?," 173.
[170] Ngun is himself a homosexual. He received his PhD. In December, 2012, writing on the molecular mechanisms underlying sexual differentiation in the brain. Ngun claimed he was not afraid of critiques of his work and said, "Trust me, I've had to deal with a lot worse as someone who grew up gay and an outsider. Dealing with critiques about my work are nothing compared to dealing with people telling me I'm going to hell." October 9, 2015, accessed August 10, 2016, http://vizbang.tumblr.com/post/130817769270/a-brief-digression-from-pretty-pictures.
[171] Ed Yong, "No, Scientists Have Not Found the 'Gay Gene,'" *The Atlantic* October 10, 2015, accessed July 15, 2016, http://www.theatlantic.com/science/archive/2015/10/no-scientists-have-not-found-the-gay-gene/410059/.

underpowered study and says, "But splitting the sample means that the study goes from underpowered to *really* underpowered."[172]

Andrew Gelman, a statistician at Columbia University, claimed the Ngun study inaccurately presented results as statistically significant. Gelman roundly critiqued Ngun's methodology and said, "Now let me say right here that I think the whole training/test-set idea has serious limitations, especially when you're working with n=47."[173] Gelman also added, "In general it seems like you're asking for trouble when you start publicizing technical claims without supplying the accompanying evidence."[174] Ngun himself acknowledged that the study was underpowered in social media, but blamed his small sample on lack of funding and said, "Yes, we were underpowered. The reality is we had basically no funding. . . . the sample size was not what we wanted. But do I hold out for some impossible ideal or do I work with what I have? I chose the latter."[175] This seems like a bad case of special pleading. Essentially, Ngun is saying, "I know that in research it is important to have a good sample size. I couldn't afford that because I had no funding. But I still want you to take my research seriously because I'm sincere and genuine and doing the best I can with what I have." Earnestness and a strong desire to do research cannot compensate for an underpowered study.

John Greally of the Albert Einstein College of Medicine also noted that deriving the DNA sample from saliva could lead to misleading results for the type of research the Ngun team was doing. The epigenetic marks iin the saliva could be quite different from those in the brain, which is the area of Ngun's focus. Greally also pointed out that the team developed a "new" algorithm to evaluate the data and asks, "Why use a new algorithm to identify these predictive markers, did current approaches not yield any results?"[176] Greally also says the authors tried to give their report an air of plausibility by noting specific roles played by the genes they identified, subtly suggesting they may influence sexual orientation. The problem with epigenetics research in general and the Ngun study in particular is that while it may be plausible that epi-marks on these genes affect someone's

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[172] Ibid. Emphasis in original. Sten Linnarsson, professor of Molecular Systems biology at the Karolinska Institute in Sweden (and no fan of conservatives!), tweeted about the Ngun study, "This is terrible science in so many ways I lost count." October 8, 2015, @slinnarsson.
[173] Andrew Gelman, "Gay Gene Tabloid Hype Update," Statistical Modeling, Causal Inference, and Social Science, October 10, 2015, http://andrewgelman.com/2015/10/10/gay-gene-tabloid-hype-update/.
[174] Ibid.
[175] Tuck Ngun, "A Brief Digression from Pretty Pictures," October 9, 2015, accessed February 26, 2016, http://vizbang.tumblr.com/post/130817769270/a-brief-digression-from-pretty-pictures.
[176] John Greally, "Over-Interpreted Epigenetics Study of the Week," October 9, 2015, http://epgntxeinstein.tumblr.com/post/130812695958/over-interpreted-epigenetics-study-of-the-week-2.

sexual orientation, it is also possible that sexual orientation affects the epi-marks. In other words, what Ngun demonstrated was a correlation in his data between sexual orientation and the epi-marks. His data does not demonstrate which direction, if any, causation is moving.

Other scientists have suggested the Ngun data may be an example of a "false positive." Johnjoe McFadden, a molecular geneticist at the University of Surrey, said, "Studies that associate biomarkers with particular traits are notoriously prone to false positive results due to the tendency of these studies to find spurious associations that are down to sheer chance."[177]

Of some interest is that a paper Eric Vilain co-authored in the Spring of 2016 did not mention the findings of his own research team. Vilain and Ngun's 2014 paper was cited, but not their findings delivered in the Fall of 2015. In fact, the only data cited in the paper Vilain co-authored in 2016 was from a 2011 study of 34 identical twin pairs which revealed no support for the hypothesis that epigenetics influences male sexual-orientation![178]

There seem to be contradictory claims about how many regions of interest were discovered in the epigenome. For example, Michael Balter in *Science* said Ngun had found "five regions" while Ngun's abstract refers to nine regions of interest. I suspect he started with nine regions of interest, but narrowed it down to a subset of five.[179]

Ngun's summary of research regarding genetic and biological factors associated with increased rates of homosexuality is also misleading. For example, he states, "Male sexual orientation has been linked to several genomic loci, with Xq28 and 8p12 being the most replicated."[180] Ngun is referring to Dean Hamer's

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[177] Jessica Hamzelou, "Gay or Straight? Saliva Test Can Predict Male Sexual Orientation," *New Scientist,* October 8, 2015, accessed August 10, 2016, https://www.newscientist.com/article/dn28307-gay-or-straight-saliva-test-can-predict-male-sexual-orientation/. A false positive is a result that indicates a given condition or attribute is present when it is not.

[178] *See* J. Michael Bailey, Paul L. Vasey, Lisa M. Diamond, S. Marc Breedlove, Eric Vilain, and Marc Epprecht, "Sexual Orientation, Controversy and Science," *Psychological Science in the Public Interest* 17.2 (April 25, 2016): 77. The authors cite S. Bocklandt, W. Lin, M.E. Sehl, F.j. Sanchez, J.S. Horvath, and Eric Vilain, "Epigenetic Predictor of Age," *PLoS One* 6.6 (2011): e14821 http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0014821.

[179] Michael Balter in *Science* said Ngun had found "five regions" while Ngun's abstract refers to nine regions of interest. *See* Michael Balter, "Can Epigenetics Explain Homosexuality Puzzle?" *Science* 350. 6257 (October 9, 2015): 148.

[180] Tuck C. Ngun, W. Guo, N.M. Ghahramani, K. Purkayastha, D. Conn, F.J. Sanchez, S. Bocklandt, M. Zhang, C.M. Ramirez, M. Pellegrini, and Eric Vilain, "Program Number 95: A Novel Predictive Model of Sexual Orientation Using Epigenetic Markers," Paper Presented at the American Society of Human

1993 claim to have found co-inherited genetic information among homosexual brothers in the gene-dense Xq28 region. Actually, several attempts to replicate Hamer's findings have resulted in conflicting data. In 2015, Alan Sanders and Michael Bailey claimed to have replicated Hamer's findings concerning homosexuality and the Xq28 region in addition to discovering an area of interest at chromosome region 8q12. First, Ngun incorrectly identifies the region as *8p12***,** when Sanders and Bailey's research clearly says 8q12.[181] But more importantly, Ngun overstates the strength of the findings regarding each of these regions, with all research demonstrating that the findings to date in these areas have a very weak predictive power.

Ngun also says "each male pregnancy a woman has increases the chance that her next son will be homosexual by 33% (the fraternal birth order effect)."[182] But this oft-repeated claim has many weaknesses, including the fact that around half of all homosexual males have *no* brothers, data from other sources which questions the existence of the fraternal birth order effect altogether, and the fact that the fraternal birth order effect (if it exists) can only account for homosexuality in one out of every seven homosexual men.[183] Finally, Ngun makes a passing reference to early life androgen exposure being associated with more homosexuality among women. Apparently, he has women with Congenital Adrenal Hyperplasia in mind, but doesn't mention that most of these women have a heterosexual identity. Ngun implies these findings – Xq28 and 8q12, the fraternal birth order effect, and prenatal androgen exposure in women – have a stronger influence than the data actually allows. In fact, findings in each of these areas only demonstrate a lower level of correlation between certain variables and a higher level of self-reported same-sex attraction. And there is still possibility that the Xq28 and fraternal birth order claims may yet be disproved. Ngun simply over-states the data to make his own claim sound more plausible.

Ngun's own response to the data and critiques of it is a bit confusing. On one hand, he said that the researchers want to replicate the study in a different group of twins and also determine whether the same marks are more common in

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Genetics 2015 Annual Meeting, Balitmore, MD, October 8, 2015, accessed February 26, 2016, https://ep70.eventpilotadmin.com/web/page.php?page=IntHtml&project=ASHG15&id=150123267.

[181] The centromere divides each chromosome into two major regionis: the smaller "P" region and the larger "Q" region.

[182] Ngun, et al, "A Novel Predictive Model of Sexual Orientation Using Epigenetic Markers."

[183] James M. Cantor, Ray Blanchard, Andrew D. Paterson, and Anthony Bogaert, "How Many Gay Men Owe Their Sexual Orientation to Fraternal Birth Order?" *Archives of Sexual Behavior* 31.1 (February 2002): 63 – 71.

gay men than in straight men in a large and diverse population.[184] But Ngun told another source he had quit the lab at the Geffen School of Medicine out of fear of how the data they were generating might be used. He said, "I don't believe in the censoring of knowledge, but given the potential for misuse of the information, it just didn't sit well with me."[185] Ngun seemed concerned that his research could be used by evil people or governments to identify homosexuals for the purpose of persecuting them. Yet, the weak and flawed nature of his findings make this fear sound quite unreasonable.

Epigenetics and Drug Addiction

As was noted above, one problem with Ngun's data is that he assumes the epigenetic tags he identified *caused* homosexuality, when it may in fact be the case that homosexuality caused a difference in the epigenetic tags. Research into alcoholism indicates this is at least a plausible scenario.

A robust body of evidence strongly indicates that alcoholism can lead to epigenetic changes which actually strengthen the alcoholism itself. An emerging model suggests that some genetic factors may predispose some people to alcoholism. These genetic factors are accentuated because expression of certain genes can be modified by excessive alcohol consumption – epigenetic changes can be induced by alcohol which modifies gene expression. These changes encourage further alcohol use and ultimately contribute to addiction.[186] One source says, "Although researchers are still piecing together all the details, findings to date suggest that epigenetic changes in gene expression induced by alcohol consumption may be the source or contributing factor in the brain pathology and adaptations in brain functioning associated with alcohol abuse and alcohol dependence and may contribute to alcohol relapse and craving."[187]

One group of researchers in 2012 studied the brains of 17 alcoholics along with a control group of 15. In their small sample, alcohol abuse was associated with widespread changes in brain gene expression.[188] In other words, consumption

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[184] Sara Reardon, "Epigenetic Tags Linked to Homosexuality," *Nature* October 12, 2015, accessed August 10, 2016, http://www.nature.com/news/epigenetic-tags-linked-to-homosexuality-in-men-1.18530.
[185] Jessica Hamzelou, "Gay or Straight? Saliva Test Can Predict Male Sexual Orientation."
[186] For a summary of the findings on alcoholism and epigenetics, see Harish R. Krishnan, Amul J. Sakharkar, Tara L. Teppen, Tiffani D.M. Berkel and Subhash C. Pandey, "The Epigenetic Landscape of Alcholism," *International Review of Neurobiology* 115 (2014): 75 – 116.
[187] U.S. Department of Health and Human Services: National Institute on Alcohol Abuse and Alcoholism, "Epigenetics – A New Frontier for Alcohol Research," *Alcohol Alert* 86 (November 1, 2013): 4.
[188] Igor Ponomarev, Shi Wang, Lingling Zhang, R. Adron Harris, and R. Dayne Mayfield, "Gene Coexpression Networks in Human Brain Identify Epigenetic Modifications in Alcohol Dependence," *The Journal of Neuroscience* 32.5 (February 1, 2012): 1884 – 1897. I acknowledge this is a small sample.

of alcohol was associated with a change in the epigenome which subsequently altered the manner in which genes were expressed in the brain, probably contributing to alcoholism.

With some caution, I suggest that we should at least be open to the possibility that something similar may occur in homosexuality. People who engage in homosexual behavior may find that the behavior itself is reinforced by epigenetic changes brought on by the homosexual behavior. In this way, the behavior may become compulsive and feel quite "natural." Such an epigenetic mechanism may also partially explain the higher rate of the experience of childhood sexual abuse experienced by homosexuals, a trend admitted by most pro-homosexual authors.[189] We know the age of sexual debut, the context in which it occurred, and the age and gender of the person with whom the sexual debut occurred have a strong organizing effect on later sexual identity. It is at least plausible that in some cases of child abuse, the abuse itself initiates a cascade of epigenetic changes which contribute to same-sex attraction in adulthood. Such a hypothesis has limited explanatory power since the majority of homosexuals do not report being abused as children.

It is important to remember that epigenetics is a somewhat new sub-discipline within genetics, so the exact mechanisms of epigenetic function are still being unraveled at a broad level, much less in the specific case of homosexuality. The degree to which sexual behavior affects the epigenetic signals within a person are speculative at present, but it is at least plausible that participation in homosexual behavior may alter one's epigenetics.

VII. Homosexuality and Other Factors

Several other issues are often mentioned in relation to scientific research and homosexuality. I will briefly summarize some of the most common ones.

Prenatal Hormones and Homosexuality

A standard argument related to the origin of homosexuality is that exposure to certain hormones in the womb can cause children to have a homosexual orientation. As is well known, estrogen and testosterone are closely related to human sexuality. In males, testosterone levels peak at three different times. During

[189] Because this is commonly admitted, I find it odd that Ngun and Vilain take issue with the idea that childhood abuse can contribute to a later homosexual identity, a claim they call "discredited." Ngun and Vilain, "The Biological Basis of Human Sexual Orientation: Is There a Place for Epigenetics?," 172.

the early part of pre-natal development – from around weeks 6 – 20 -- in male babies, testosterone drives the development of male genitalia. At birth, babies experience a "mini-puberty," with elevated gonadotropin levels and steroid hormone levels reaching the adult stage. Males are born with an elevated testosterone level that rapidly decreases in the first day of life and then rises again after about one week. After about three months age, a male infant's testosterone levels begin to fall back to pre-pubertal levels.[190] This second rise in testosterone in males which follows soon after birth continues until around 6 – 12 months. A final rise in testosterone in males occurs at puberty and continues throughout life though the levels decrease as men age. Some suggest that if male's testosterone levels are negatively affected in either phase 1 or phase 2, then the child may subsequently be homosexual. In recent years Gorski has argued the brain is basically female during early prenatal development. For a male to develop structural and functional characteristics typical of his species, his brain must be exposed to testicular hormones during a critical period, or critical phases, of development.[191] Research ethics prevent researchers from proving whether this same effect is observable in humans, but a common assertion is that a person's sexual identity is affected by the timing and amount of hormones released either prenatally or postnatally.

A syndrome called Congenital Adrenal Hyperplasia (CAH) occurs once out of every 10,000 – 18,000 births and has also provided some evidence that prenatal hormone levels may affect a child's sexuality. CAH can occur in both males and females and is caused when a pre-born child has an enzyme defect that makes it impossible for the baby's adrenal gland to make cortisol, a hormone that is vital for proper pre-natal development. At the same time, in a baby with CAH, the body produces more androgen, a male sex hormone. For males, CAH often results in an early on-set of puberty. In females, the symptoms seem to be more pronounced. In some cases, females are born with abnormal genitalia at birth, presenting aspects of both male and female genitalia.[192]

CAH is often cited as evidence that abnormal hormone levels affect not only genitalia but also the brain. Why so? Research indicates that as a group, CAH females are more likely to be sexually attracted to women than are their unaffected sisters, with up to one-third self-identifying as homosexual or bisexual. Nevertheless, a majority of women with CAH report themselves to be exclusively

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[190] Patricia Y. Fechner, "The Biology of Puberty: New Developments in Sex Differences," in *Gender Differences at Puberty*, Chis Hayward, ed. (Cambridge: Cambridge University Press, 2003), 18.
[191] Roger A. Gorski, "Hypothalamic imprinting by gonadal steroid hormones," *Advances in Experimental Medical Biology* 511 (2002): 57 – 70.
[192] Medline Plus, "Congenital Adrenal Hyperplasia," http://www.nlm.nih.gov/medlineplus/ency/article/000411.htm (Accessed January 9, 2013).

heterosexual, and it is unclear what differentiates them from others who are not. In contrast, males with CAH have generally been found to be similar to their unaffected brothers. Specifically, they do not seem to differ in sexual orientation.[193] Based on the higher incidence of homosexuality among girls with CAH, some have argued that prenatal hormone levels affect the development of sexual identity. A 1985 study comparing 30 women exposed to DES in utero with a control group of 30 women not exposed to DES found a higher rate of homosexuality among the women exposed to DES, along with a subsample control group of sisters not exposed to DES. In comparison to both control groups, the DES women showed increased bisexuality and homosexuality. However, about 75% of the DES women were exclusively or nearly exclusively heterosexual.[194]

Another argument sometimes used to support the pre-natal hormone theory is the histories of children exposed to diethylstilbestrol (DES) in utero. DES is a synthetic, not-steroidal estrogen which was prescribed to pregnant women between 1940 – 1971 under the mistaken belief DES would prevent miscarriage, premature labor, and related complications of pregnancy.[195] A 1985 study comparing 30 women exposed to DES in utero with a control group of 30 women not exposed to DES found a higher rate of homosexuality among the women exposed to DES, along with a subsample control group of sisters not exposed to DES. In comparison to both control groups, the DES women showed increased bisexuality and homosexuality. However, about 75% of the DES women were exclusively or nearly exclusively heterosexual.[196] These findings were surprising because previous research by some of the same authors had found increased femininity among DES-exposed women. While these findings raised some interest about the effects of prenatal hormones on sexual orientation, a 1993 review of research concerning DES-exposed women and

[193] Celina C.C. Cohen-Bendahan, Cornelieke van de Beek, and Sheri A. Berenbaum, "Prenatal Sex Hormone Effects on Child and Adult Sex-Typed Behavior: Methods and Findings," *Neuroscience and Behavioral Research Reviews* 29.2 (April 2005): 359.

[194] A. A. Ehrhardt, H. F. Meyer-Bahlburg, L. R. Rosen, J. F. Feldman, N. P. Veridiano, I. Zimmerman, and B. S. McEwen, "Sexual Orientation after Pre-natal Exposure to Exogenous Androgen," *Archives of Sexual Behavior* 14.1 (February 1985): 57 – 77.

[195] National Cancer Institute, "Diethylstilbestrol (DES) and Cancer," October 5, 2011, accessed March 28, 2017, https://www.cancer.gov/about-cancer/causes-prevention/risk/hormones/des-fact-sheet#q1.

[196] A. A. Ehrhardt, H. F. Meyer-Bahlburg, L. R. Rosen, J. F. Feldman, N. P. Veridiano, I. Zimmerman, and B. S. McEwen, "Sexual Orientation after Pre-natal Exposure to Exogenous Androgen," *Archives of Sexual Behavior* 14.1 (February 1985): 57 – 77.

sexual orientation found no clear-cut differences can be demonstrated to date between DES-unexposed and DES-exposed women in gender-related behavior, although the physical and psychological impact of the problems associated with exposure to DES are well documented. The article concluded by saying "there are a vast number of biological and psychosocial factors that are interacting to explain specific behavioral traits. To date, no clear-cut differences have been reported between unexposed and DES-exposed women in gender-related behavior."[197] A 2003 study of men and women found prenatal DES exposure in men was unrelated to the likelihood of ever having been married, age at first intercourse, number of sexual partners, and having had a same-sex sexual partner in adulthood. The same study found only minor differences exposed and unexposed DES women and concluded their findings provide little support for the hypothesis that prenatal exposure to DES influences the psychosexual characteristics of adult men and women.[198]

Scott Kerlin is a social scientist at the University of British Columbia and he himself was exposed to DES in utero. In an online study of 500 men in a children-of-DES support group, he reported that 150 of the men identified themselves as having any of a variety of gender-related disorders. While Kerlin does not claim DES caused this disorders, he thinks the data is significant and should be considered when evaluating the influence of prenatal hormones.[199]

The higher incidence of homosexuality among CAH women indicates prenatal hormone exposure is a contributing variable to their sexual identity, but the data does not demonstrate prenatal hormone exposure causes the orientation. Likewise, children exposed to DES in utero do not provide substantial evidence for the prenatal hormone theory regarding

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[197] Retha R. Newbold, "Gender-Related Behavior In Women Exposed Prenatally to Diethylstilbestrol," *Environmental Health Perspectives* 101.3 (August 1993): 213.

[198] L. Titus-Ernstoff, K. Perez, E. E. Hatch, R. Troisi, J.R. Palmer, P. Hartge, M. Hyer, R. Kaufman, E. Adam, W. Strohsnitter, K. Noller, K.E. Pickett, R. Hoover, "Psychosexual Characteristics Of Men and Women Exposed Prenatally to Diethylstilbestrol," *Epidemiology* 14.2 (March 2003): 155 – 160.

[199] Ernie Hood, "Are EDCs [Endocrine Disrupting Chemicals] Blurring Issues of Gender?," *Environmental Health Perspectives* 113.10 (October 2005): 676.

homosexuality or transgenderism. Some researchers acknowledge he effect of pre-natal hormones on a predisposition towards homosexuality is weak in both males and females, with Louis J. Gooren going so far as to say, "So we are far away from any comprehensive understanding of hormonal imprinting on gender identity formation."[200] Competing opinions abound regarding the pre- or postnatal effect of hormones on gender identity.

Fraternal Birth Order Effect

The "Fraternal Birth Order Effect" refers to the claim that males with multiple older brothers are more likely to be homosexual. To state it differently, homosexual males tend to born later in the families than their heterosexual brothers. This idea is most closely associated with an article published by Ray Blanchard and Anthony Bogaert in 1996 in *The American Journal of Psychiatry*, where the authors claimed each additional older brother increases the odds of homosexuality by 33%.[201] Blanchard and his colleagues suggest the connection between birth order and homosexuality may reflect a maternal immune reaction. The argument is that in some cases the mother's immune system may react to H-Y antigens in a male fetus. When this happens, H-Y antibodies from the mother may cross the placental barrier and enter the brain of the baby boy. Blanchard explains, "When that happens, these antibodies partly prevent the fetal brain from developing in the male-typical pattern, so that the individual will later be attracted to men rather than women."[202] In a later study led by Blanchard,

[200] Louis J. Gooren, "The Biology of Human Psychosexual Differentiation," *Hormone Behavior* 50.4 (2006): 589 – 601. In a more recent article in *The New England Journal of Medicine*, Gooren says the cause of gender-identity disorder are unknown, but he does seem to grant some weight to the idea that male-to-female transsexuals have a more female pattern of sexual differentiation in the brain. He plainly rejects the ideas that gender identity disorders are rooted in hormonal abnormalities, chromosomal patterns (genetics), or in particular family dynamics. Gooren would disagree with the moral stance I take in regards to homosexuality or transsexuality. *See* Louis J. Gooren, "Care of Transsexual Persons," *The New England Journal of Medicine* 364.13 (March 31, 2011): 1251 – 1257.

[201] Ray Blanchard and Anthony F. Bogaert, "Homosexuality in Men and Number of Older Brothers," *The American Journal of Psychiatry* 153:1 (January 1996): 27 – 31.

[202] Ray Blanchard, "Fraternal Birth Order and the Maternal Immune Hypothesis of Male Homosexuality," *Hormones and Behavior* 40: (2001): 109 – 110.

researchers claimed the birth order effect only applies if the younger brother is right-handed.[203]

As with some of the studies mentioned earlier, Blanchard and Bogaert's research is plagued by sample problems. For example, they clearly state that homosexuals were recruited for their research at homosexual community organizations and at the 1994 Toronto Lesbian and Gay Pride Day parade.[204] Jones and Kwee suggest a negative effect on the sample because later born gay men were perhaps more apt to be "out and proud."[205] At the same time, Kwee and Jones add, "Despite various methodological problems with the fraternal birth order research, we concede that the evidence as a whole points to some sort of relationship between the number of older brothers and homosexuality."[206]

Blanchard and Bogaert's research has been challenged in two other ways. First, their maternal immunosensitization hypothesis is just that – a hypothesis and has not been proven. Second, at least three studies have claimed that a fraternal birth order effect is at work to some degree in certain sexual offenders.[207] If this correlation is confirmed, certainly no one would then suggest the sexual offenders are, through no fault of their own, pre-disposed to act in such a way and thus should be excused from moral censure. But perhaps the strongest critique of the fraternal birth order affect is the fact that half or more of all homosexual men have zero older brothers, a fact that Blanchard himself admits and comments, "The

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[203] Ray Blanchard, J.M. Cantor, Anthony F. Bogaert, S.M. Breedlove, and Lee Ellis, "Interaction of Fraternal Birth Order and Handedness in the Development of Male Homosexuality," *Hormones and Behavior* 49.3 (2006): 405 – 414.

[204] Ray Blanchard and Anthony F. Bogaert, "Homosexuality in Men and Number of Older Brothers," 28.

[205] Jones and Kwee, "Scientific Research, Homosexuality, and the Church's Moral Debate: An Update," 309.

[206] Ibid., 310.

[207] Martin L. Lalumière, Grant T. Harris, Vernon L. Quinsey, and Marnie E. Rice, "Sexual Deviance and Number of Older Brothers Among Sexual Offenders," *Sexual Abuse: A Journal of Research and Treatment* 10.1 (1998):5 – 15; K. Côté, C.M. Earls, and Martin L. Lalumière, "Birth order, Birth Interval, and Deviant Sexual Preferences Among Sex Offenders," *Sexual Abuse: Journal of Research and Treatment* 14 (2002): 67 – 81; S.I. MacCulloch, N.S. Gray, H.K. Phillips, J. Taylor, and M.J. MacCulloch, "Birth Order in Sex-Offending and Aggressive Offending Men," *Archives of Sexual Behavior* 33.5 (October 2004): 467 – 74.

maternal immune hypothesis was never intended to account for the sexual orientation of all homosexual men." [208]

Various and Assorted Assertions About Homosexuality

Several other biological or genetic traits among homosexuals are often mentioned. There are frequent claims that handedness – being left handed versus right handed – correlates with homosexuality, the assertion being that homosexual men have a higher incidence of being left-handed. Some say that right-handed men with older brothers and left-handed men without older brothers have a higher chance of being homosexual.[209] Another common claim relates to the ratio of length between the ring finger and the index finger with an increased propensity for homosexuality. In males, the ring finger is usually longer than the index finger. In females, the ring finger and the index finger are typically about the same length. One group of researchers reported that lesbians have finger lengths more similar to men than to women and suggested this was due to prenatal exposure to androgens.[210] In 1998, University of Texas, Austin Dennis McFadden and Edward G. Pasanen claimed the click-evoked otoacoustic emissions [CEOAE] are stronger in women than in men, but the CEOAEs of homosexual and bisexual females were found to be intermediate to those of heterosexual females and heterosexual males. McFadden and Pasanen suggested the reason for their findings might be that the auditory systems of homosexual and bisexual females, and the brain structures responsible for their sexual orientation, have been partially masculinized by prenatal exposure to high levels of androgens.[211] In 2005,

[208] Ray Blanchard, "Controversies in Sexual Medicine – Male Homosexuality: Nature or Culture?" *Journal of Sexual Medicine* 7.10 (October 2010): 3248.

[209] Ray Blanchard, James M. Cantor, Anthony F. Bogaert, S. Marc Breedlove, and Lee Ellis, "Interaction of fraternal birth order and handedness in the development of male Homosexuality," *Hormones and Behavior* 49 (2006): 405 – 414.

[210] Bernd Kraemer, Thomas Noll, Aba Delsignore, Gabriella Milos, Ulrich Schnyder, Urs Hepp, "Finger Length Ratio (2D:4D) and Dimensions of Sexual Orientation," *Neuropsychobiology* 53.4 (2006): 210 – 214.

[211] Dennis McFadden and Edward G. Pasanen, "Comparison of the auditory systems of heterosexuals and homosexuals: Click-evoked otoacoustic emissions," *Proceedings of the National Academy of Sciences* 95.5 (March 3, 1998): 2709 – 2713. For a review critical of McFadden and Pasanen's methodology see Bonnie P. Spanier and Jessica D. Horowitz, "Looking for a Difference: Methodology is in the Eye of the Beholder," *Gender and the Science*

researchers associated with the Karolinska Institute in Stockholm claimed homosexual men reacted to male pheromones in a manner similar to heterosexual women.[212] Another study even attempted to find a connection between scalp hair rotation patterns and male homosexuality.[213] Other claims abound and more will certainly emerge in the near future.

VIII. Is Homosexuality Immutable?

Is homosexuality an immutable trait? In *Obergefell v. Hodges,* Justice Kennedy said, "Only in more recent years have psychiatrists and others recognized that sexual orientation is both a normal expression of human sexuality and immutable."[214] A careful study of data regarding homosexuality and sexual orientation change efforts shows the complete change in orientation is extremely rare. Movement on a continuum of change happens more frequently than pro-LGBTQ advocates care to admit, but not as often as Christians might hope.

IX. Conclusion

In 2004, United Artists released *Saved!*, a film which lampooned Evangelical Christians as hypocritical, shallow, and judgmental. The film was directed by Brian Dannelly, who attended a conservative Christian high school as a teenager and now identifies as a homosexual. Michael Stipe of the rock group R.E.M., who has self-identified as "queer," also co-produced the movie. The film itself centers on the fictional American Eagle Christian High School with characters Dannelly says are drawn from his own experience in a religious school. The movie is replete with secular stereotypes of Evangelical Christians: The school's principle, "Pastor

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of Difference: Cultural Politics of Contemporary Science and Medicine, Jill A. Fisher, ed. (New Brunswick, NJ: Rutgers University Press, 2011), 43 – 66. Spanier and Horowitz affirm homosexual rights.

[212] Ivanka Savic, H. Berglund, P. Lindstrom, "Brain Response to Putative Pheromones in Homosexual Men," *Proceedings of the National Academy of Science* 102 (2005): 7356 – 7361.

[213] A.J.S. Klar, "Excess of Counterclockwise Scalp Hair – Whorl Rotation in Homosexual Men," *Journal of Genetics* 170 (2004): 2027 – 2030.

[214] *Obergefell v. Hodges* 576 U.S. 8 (2015).

Skip," is having an extra-marital affair; the most outspoken Christian girl in the movie is hateful and mean, and, of course, homophobia is ever present.

The movie's plot revolves around a high school senior named Mary who is in love with her Christian boyfriend, Dean. Then, Dean tells Mary he is homosexual. After bumping her head underwater, Mary is convinced she has a vision in which Jesus tells her to sleep with Dean in order to cure his homosexuality. Ultimately, Dean's parents send him to a Christian ministry for "de-gayification" while Mary becomes pregnant with Dean's baby. Like many other high school movies, *Saved!* reaches its climactic point at the prom in a scene where Dean arrives with his boyfriend, proud of his homosexual identity. Pastor Skip is furious and insists homosexuality is wrong and a moral "black and white" issue. To this, Pastor Skip's son responds that there is no black and white, only gray. What is perhaps most frustrating and intriguing about *Saved!* is that the characters with the greatest religious devotion are the most self-centered while the least religious characters display compassion, empathy, and concern for others.

The view of Christians and homosexuality in *Saved!* predominates in our culture. "Born this way" arguments are presented as intelligent, cogent, compassionate, and compelling. The traditional Christian view that homosexual behavior is sin is viewed as antiquated, unkind, rooted in ignorance, and hopelessly inconsistent with the best science while Christians themselves are portrayed as mean-spirited and cruel. Furthermore, we are told that in sexual ethics, there are no remaining areas where we can speak in moral absolutes – everything is only "gray" with no distinct difference between right and wrong. "Born this way" arguments are central to this new ethic, insisting homosexual behavior is a natural variation in human behavior to be celebrated. But, are homosexuals correct when they assert they are "born this way"? Have the clear boundaries of "black and white" morality dissolved into an ethical gray? Does science require us to suspend moral judgment regarding homosexuality?

Several scientific studies were initiated in recent decades to substantiate the pro-homosexual argument that homosexuals are not just different in their sexual behavior, but they are constitutionally different from heterosexuals. Pro-homosexual advocates hope these studies will remove the moral stigma associated with homosexuality by proving it is not really a "choice," but an expression of their innate nature. Their goal is to convince others that if homosexuals are "born this way," then they should not receive moral censure for their sexual lifestyle. Furthermore, homosexuality should be viewed as an innate characteristic as immutable as one's race. The claim is then made that if it is wrong to discriminate against someone because of race, it is equally wrong to discriminate against someone because he or she is homosexual.

How do we respond to "born this way" arguments? Now that we have reviewed most of the major findings regarding homosexuality, we will summarize a Christian response. We have seen that while there are some genetic or biological factors that correlate with a higher incidence of same-sex attraction and homosexual behavior, as of yet there is no proof of genetic or biological causation for homosexuality. A Christian response will engage data focusing on several key areas including the oft cited "gay gene," prenatal hormones, and sexual orientation change efforts. The overriding principle that emerges for a correct understanding of the data is to remember that *correlation does not equal causation*. Interpreting the data correctly also depends on a robust understanding of the Christian doctrines of sin and temptation.

No Gay Gene

Contrary to popular opinion, scientific research has not found a "gay gene." No genetic marker has yet been found that conclusively links homosexuality with any specific genetic sequence. Despite the common myth that a gay gene has been found, the claim of a genetic marker for homosexuality at Xq28 has been refuted. Some twin studies do indicate correlation for a genetic component to some forms of homosexuality, but even in these studies, the correlation is weak and is neither

necessary nor sufficient for homosexuality.[215] Earlier twin studies which claimed a much stronger correspondence for homosexuality among twins have not been replicated.[216] Twin research indicates a genetic aspect may be at work at a low level of importance. The fraternal birth order effect seems to show a weak correlation between some forms of male homosexuality and maternal inheritance. Explanations of this phenomenon are speculative at present.

"Born this way" arguments regarding genetics must also admit the most obvious fact: To date, no genetic markers whatsoever have been associated with female homosexuality. If one is going to use genetic research to date to argue homosexuality is an innate characteristic, then one must admit any data that exists – and that data is weak – applies only to males and not to females. Yet, the very fact that current research has found *no areas of significance* which indicate a genetic link for female homosexuality surely points out the inherent weakness in the overall "gay gene" argument. Claims that homosexuality is caused by a "gay gene" are simplistic and imprecise.

Christians must be aware of the manner in which activists misrepresent data concerning genetic linkage to homosexuality. In their summary of research on twin data and homosexuality, pro-homosexual authors Wilson and Rahman begin by saying, "Clearly, genetic factors are involved in the origins of sexual orientation."[217] Christians can concur at this point, since no one denies a genetic influence on any number of behaviors. But Wilson and Rahman then immediately follow this statement with the completely false assertion, "Now that we know that 'gay genes' really do exist, how do we go about finding them?"[218] This second statement is a non sequitor – it is an unwarranted conclusion based on equivocation. Wilson and Rahman wrongly move from discussing "influence" to claiming causation ("gay genes exist"). Twin studies do not prove a "gay gene" exists.

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[215] Several times in my conclusion, I will mention that no factor has been found that is necessary or sufficient for homosexuality. My thinking here is influenced by Stanton Jones and Mark A. Yarhouse, *Homosexuality: The Use of Scientific Research in the Church's Moral Debate* (Downers Grove, IL: InterVarsity Press, 2000), 81ff.

[216] Frustratingly, popular literature on homosexuality repeats the findings of earlier twin studies without citing the later research. For example, Cheryl L. Weill cites the twin research of Bailey from 1991 and 1993, but does not mention the later findings from the Australian twin study which contradict Bailey's earlier research even though Weill is clearly aware the findings exist. See Cheryl L. Weill, *Nature's Choice: What Science Reveals About the Biological Origins of Sexual Orientation* (New York: Routledge, 2009):65 – 68.

[217] Glenn Wilson and Qazi Rahman, *Born Gay: The Psychobiology of Sex Orientation* (London: Peter Owen Publishers, 2005, 2008), 49.

[218] Ibid., 50.

To assume sexual orientation is a Mendelian trait[219] such as hair color or eye color is incautious and misguided. As a behavioral phenotype, homosexuality is the result of a complex interaction between the genotype, environment, and choices made by a particular person. A person's genotype is one factor among many influencing the type of person we become. Reducing homosexuality to a Mendelian trait assumes humans are genetic automatons, hard-wired to act according to a predetermined set of coded instructions. No evidence indicates homosexuality is a trait equivalent to hair color or skin pigmentation and assuming so confuses the essential distinction between physical traits and behavior. A lifestyle with as many broad expressions as homosexuality is certainly the result of multiple causes.

We should not overlook the worldview conflicts that are inherent in the debate about homosexuality and genetics. As noted, pro-homosexual researchers will sometimes point out that homosexual behavior is observed in animals.[220] Thus, from a Naturalistic Darwinian worldview, homosexuality in humans then is neither good nor evil but simply a naturally occurring variation of sexual behavior.[221] But this is a form of the naturalistic fallacy, drawing the conclusion homosexuality ought to be affirmed because it is "natural." In this way a strong overtone of genetic determinism drones on in the background of many pro-homosexual arguments. If one agrees that Darwinian naturalism is the correct meta-narrative and that humans are nothing more than chemicals that have learned to self-reflect, then we are truly the sum of our DNA and doomed to whatever behavioral destiny is in the millions of base pairs inside the double helix of our genetic structure, and therefore not responsible for our deeds. But what if we are more than that? What if the Bible is correct when it says each person has a soul that transcends our genetic and biological composition? In that case, we really are morally accountable decision-makers with an ability to move beyond the limits of genetic or biological predisposition.

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[219] What I call a Mendelian trait seems to be the same idea expressed by the Feinbergs when they discuss the difference between an inherited trait (Mendelian) versus heritability. *See* John Feinberg and Paul Feinberg, *Ethics for a Brave New World*, 2nd ed,(Westchester, IL: Crossway, 2010), 366 – 367.

[220] The most well-known presentation of this argument is found in Bruce Bagemihl, *Biological Exuberance: Animal Homosexuality and Natural Diversity* (New York: St. Martin's Press, 1999).

[221] Some have even argued that homosexuality can be explained as a favorable evolutionary trait related to high female fecundity within certain groups. See Andrea Camperio Ciani, Paolo Cermelli, and Giovanni Zanzotto, "Sexually Antagonistic Selection in Human Male Homosexuality," *PloS ONE* 3.6 (June 2008): e2282. Barry Kuhle and Sarah Radtke have argued female homosexuality is an adaptive form of sexual fluidity in women, claiming the ability to have same-sex intimate relationships among primitive women may have allowed for a shared parenting load. Barry X. Kuhle and Sarah Radtke, "Born Both Ways: Alloparenting Hypothesis for Sexual Fluidity in Women," *Evolutionary Psychology* 11.2 (April 2013): 304 – 323.

Brain Research Does Not Provide Incontrovertible Evidence

There is no conclusive evidence that homosexuals have a substantially different brain structure than heterosexuals. Claims that homosexuals have a brain structure that differs from their gender (males have "female" brains or females have "male" brains) find their origin in over-stated claims about the differences in male and female brains.[222] Two findings in particular seem to have found possible differences between heterosexual and homosexual brain structures. First is LeVay's claim that INAH 3 is smaller in homosexual males than in heterosexual males. Yet even this claim is based on very limited research and Byne, et al have stressed that the difference is not in the number of neurons, but in the density of the neurons of the INAH 3 in homosexuals. Again, this finding shows a weak correlation and the difference in INAH 3 is neither necessary nor sufficient for homosexuality. Second, Savic and Lindström's findings about patterns of brain asymmetry in homosexuals are intriguing, but await replication by other researchers. What Christians must remember is there are people who self-identify as homosexual who do not share the INAH 3 pattern identified by LeVay or the brain asymmetry patterns identified by Savid and Lindström. Thus, neither a particular INAH 3 size nor a specific pattern of brain asymmetry is either necessary or sufficient to cause homosexuality.

Prenatal Hormones

Recent "born this way" arguments about homosexuality are focusing more and more on the role of prenatal hormones in affecting sexual orientation. The basic claim is that homosexual females were exposed to inordinate amounts of androgens at crucial points in prenatal development, thus "masculinizing" their brain. Likewise, it is claimed that homosexual males were exposed to either excessive amounts of female hormones or insufficient male hormones at crucial points in prenatal development and, thus, their brains failed to masculinize properly resulting in same-sex attraction.[223] Quite often, research in which animals were intentionally exposed to divergent patterns of sex hormones during gestation is cited in favor of the prenatal hormone theory. Some of these animals which

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[222] One work says, "In human brains, dimorphisms have so far proven to be small, subtle, few, and of unknown function." Going on to say, "Perhaps the most reliable conclusion we can draw about sexual dimorphisms in human brain structure is that there are so few of them." Mark Bear, Barry W. Connors, Michael A. Paradiso, *Neuroscience: Exploring the Brain*, 3rd ed. (Baltimore: Lippincott, Williams, and Wilkins, 2007), 546, 548.

[223] Frankowski comments, "There is some evidence that prenatal androgen exposure influences development of sexual orientation, but postnatal sex steroid concentrations do not vary with sexual orientation." Barbara L. Frankowski, "Clinical Report: Guidance for the Clinician in Rendering Pediatric Care – Sexual Orientation and Adolescents," *Pediatrics* 113.6 (June 2004): 1828.

were so treated demonstrate behavior more like the opposite sex in adulthood. Thus, the claim is made that something similar happens in humans resulting in homosexual behavior.

Ethical guidelines prohibit the type of research on humans and prenatal hormones such as have been seen in animals. However, two naturally occurring cases of divergent patterns of prenatal hormone exposure are often cited in born this way arguments: Congenital Adrenal Hyperplasia (CAH) and Androgen Insensitivity Syndrome (AIS). Yet, neither of these naturally occurring cases provides proof of the "born this way" argument. Most females with CAH identify as heterosexual females as adults. Likewise, XY children with the most extreme forms of AIS give every physical appearance (except for lack of secondary hair) of being female. The vast majority identify as heterosexual females as adults, a sexual identity that fits the way their bodies look. In short, neither CAH nor AIS prove the "born this way" argument in relation to prenatal hormones. Hormones obviously affect the development of children in the womb, but it is still unclear exactly how prenatal or postnatal hormones affect one's later gender-identity. Some avenues of future research may connect the emerging science of epigenetics with particular patterns of prenatal hormone exposure.

Correlation Doesn't Equal Causation

A survey of modern scientific research demonstrates there are some factors that *correlate* with a higher incidence of homosexuality among some populations. However, there are no biological or genetic factors that have been shown to *cause* homosexuality. In moral debate, this vital distinction between correlation and causation is usually lost. Correlation refers to the degree that two different variables are related. When two sets of data are strongly linked together (example: smoking and lung cancer), there is a strong correlation. When two sets of data are not strongly linked together, there is a weak correlation.[224] The research we have surveyed has tried to find out how one variable, homosexuality, is dependent on other variables such as brain structure or genetics. We conclude that no such strong correlation can be established between homosexuality and other genetic or

[224] Researchers typically use the terms no relationship, positive relationship, and negative (inverse) relationship. I've used the terms "strong" and "weak" for simplicity and convenience.

biological variables. Furthermore, these weak correlations may be due to some other confounding variable as yet not studied.

But correlation does not equal causation. To say that one variable *causes* another variable is logically immodest and equally difficult to substantiate. In causation, one event or variable necessarily precedes another event or variable. Causation is a relationship of *necessity* among events or variables such that whenever X happens, event Y cannot fail to follow. In that case, X is said to *cause* Y.[225] If the first event does not occur, the second event does not follow. No relationship like this exists between any biological or genetic factors and homosexuality. Furthermore, in a cause – effect relationship, the "effect" is unlikely to have occurred without the previous cause. Again, no relationship like this exists between any biological or genetic factors and homosexuality. When most people think of a genetic link to any form of human behavior, they tend to think of genes like a "light switch": the same-sex attraction is either on or off. Human behavior, especially sexual behavior, is far more complex than that.

One of the more frustrating aspects of public debate about homosexuality is the manner in which the distinction between correlation and causation is blurred, ignored, or knowingly misrepresented. For example, when reporting on Dean Hamer's research, the July 26, 1993 cover of *Time Magazine* was emblazoned with the words: "Born Gay: Science Finds a Genetic Link." In fact, Hamer's research at best showed a genetic component to homosexuality, but the *Time* headline pronounced "Born Gay," a statement implying that all argument about the cause of homosexuality should cease. Furthermore, there was no headline on *Time* when Hamer's research was not replicated.[226]

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[225] Donald Palmer, *Looking At Philosophy: The Unbearable Heaviness of Philosophy Made Lighter,* 7th ed. (New York: McGraw-Hill Education, 2020), 416. I am only citing Palmer as a helpful definition of causation.

[226] In a similar way, Swaab and Hofman's findings about the SCN were widely reported as proof of a biological cause for homosexuality. But most reports failed to mention that Swaab and Hofman themselves said, "The relationship between a SCN and homosexuality is, of course, not necessarily a causal one." D.F. Swaab and Michael A. Hofman, "An Enlarged Suprachiasmatic Nucleus in Homosexual Men," *Brain Research* 537 (1990): 146.

Other factors show a weak correlation to homosexuality and none of them are sufficient or necessary for homosexuality. Claims that "10% of all people are gay" are based on Kinsey's flawed data and even misrepresent what Kinsey claimed. Perhaps 2 – 3% of the population is either exclusively homosexual or bisexual, and exclusive homosexuality is more common among men than women.

Christians Don't Deny Genetic or Biological Aspects to Homosexuality

Are there genetic or biological components to homosexuality? Absolutely. Every choice we make while living on Earth is affected by biology and genetics. Most temptations have a strong biological component: We are tempted to engage in forbidden pleasure because it initially feels good. We seek pleasure and shun pain, sometimes to destructive ends. The problem is that our very bodies have been affected by the Fall, and even our genetic code is not what it was intended to be. In New Testament terms, we *battle the flesh*. When examined from this New Testament perspective, we begin to see the flawed logic of a "born this way" mentality. Yes, we are born this way – we are born sinners with a rebellious desire to do what God says not to do, but a "natural desire" to participate in an act does not necessarily mean the act in question is approved by God, something Paul goes to great lengths to explain in Romans 1 – 6. Christians should not be surprised to find some genetic or biological component to homosexuality. Creation, including the human body, is burdened by the weight of the Fall and awaits the second coming of Christ.

An example of the way in which genetics predispose individuals to certain destructive behavioral patterns can be seen in alcoholism. There is evidence of a genetic component for susceptibility for risk of alcoholism.[227] However, a genetic predisposition to alcoholism does not require the specific behavior of consuming alcohol to excess. In fact, awareness of a genetic predisposition would call for increased vigilance in this area of

[227] Fred Beauvais, "American Indians and Alcohol," *Alcohol Health and Research World* 22.4 (1998): 253 – 259.

one's life. Likewise, if one desires to be a fully devoted follower of Jesus Christ, awareness of one's predispositions in different areas of life requires special focus, even if we are discussing sexual temptations.

What we really are discussing is human sinfulness. One of the tragedies of sin is that while most people know how to begin a particular sin or sinful habit, very rarely do we realize the third and fourth order consequences of sin. One of the most painful results of sin is that it is habit-forming. As was noted earlier, the brain can construct neural pathways and these pathways become reinforced and stronger each time we engage in various sins. In this way, we begin to live out the consequences of Jesus' warning, "Everyone who commits sin is a slave of sin" (John 8:34).

Sympathy for Those Who Are Tempted

While there is no proof of genetic causation of homosexuality, there is enough of a genetic or biological component that we should show sympathy for those who struggle with this temptation. When some homosexuals claim that they have always struggled with same-sex attraction, even from their youth, I believe they are telling the truth. I suspect that in the future research will demonstrate that homosexuality is a highly complex combination of many factors, including biology, genetics, family of origin, social environment, and human choice. Furthermore, male homosexuality and female homosexuality seem to be significantly different in both their occurrence and etiology with men and women apparently arriving at a homosexual orientation via different paths. People are not lying when they describe this as a strong attraction. While not compromising the clear Biblical message, we should demonstrate compassionate pastoral care, just as we would to someone struggling with other temptations. Yet, we must reassert continually that a *predisposition* to a certain temptation does not mean one is *predetermined* to participate in a particular behavior.

If a member or attender of our church admits they have experienced or do currently experience same-sex attraction, we should not automatically assume they are a militant gay activist. While many Christians now distance themselves from the term "culture war," I do not and I certainly believe there are activists who want to remove Christians from the public marketplace of ideas. But the person at church who confesses such a struggle is probably not one of these people. Much like the movie *Saved!*, the culture tells church attenders who struggle with same-sex attraction to expect to be ostracized and abandoned. Without affirming homosexual behavior, a genuine Christian response walks along with a brother or sister to help them follow God's ways, especially when the culture tells them God's ways are actually wrong and should be rejected.

A Godly response to same-sex temptation may look different for each individual, falling broadly into three categories. Some single Christians may choose to live a life of Godly celibacy. Virtuous singleness is certainly an option for Christians. Others may in fact find love and affection with someone of the opposite sex, be faithful in marriage, but still have same-sex temptations from time to time without surrendering to them. Others may yet find love in a heterosexual marriage and move beyond feelings of same-sex temptation. Each of these options is consistent with Christian sexual ethics. But what we must not compromise is that sex is designed by God to be experienced in heterosexual and monogamous marriage (Genesis 2:24 – 25).

<u>Gay Christians?</u>

Many evangelicals were taken completely off guard in 2008 when popular Christian song writer and recording artist Ray Boltz announced he had divorced his wife in order to embrace homosexuality. The author of favorite songs such as "Thank You," "Take Up Your Cross," and "The Anchor Holds," Boltz declared his homosexuality in a September 12, 2008 article in *The Washington Blade*, a homosexual newspaper. Bolz now claims

to affiliate with the Metropolitan Community Church, a denomination which self-identifies as a refuge for "Gay Christians." Much as Lady Gaga appeals to God as the cause of any number of sexual preferences in her song *Born This Way*, the "Gay Christian" movement appeals to a vague form of God's love to substantiate their claim that God created them as homosexuals.

Scientific arguments for an innate propensity towards same-sex attraction are leveraged by those seeking to defend a pro-homosexual Biblical hermeneutic. Some libertine interpreters will argue that science has proven homosexuality is genetic or biological and, thus, is a temptation for which people are not responsible. Therefore, the plain Scriptural teaching that homosexual behavior is sin is incorrect and passages teaching as much must either be discarded or reinterpreted in a different way, a way favorable to acceptance of homosexuals and gay marriage. A major flaw in this line of reasoning is that it assumes science has proven homosexuality is a predetermined and immutable characteristic. Science has not proven this, but research does indicate a positive correlation between some limited factors and a somewhat higher incidence of same-sex attraction. The scientific evidence does not require us to abandon the historical, Biblical stance of the church and adopt a new hermeneutic normalizing all forms of licentious behavior.

<u>Children Become Casualties</u>

"Born this way" arguments are closely tied to sexualizing young children prematurely. One example of an adult imposing his own view of sexuality on children is found in Simon LeVay's 2011 book, *Gay, Straight, and the Reason Why*. LeVay addresses several arguments that seem to contradict his theory that homosexuality is innate. Among the data, he reviews some findings which indicate gay men and lesbians are far more likely than heterosexuals to have had sexual contact with an older person of their own sex during childhood. In other words, homosexuals have a statistically higher incidence of being molested by a homosexual when they

were a child. If such data is accurate, it could mean that being sexually molested as a child affects one's future sexual orientation. Conversely, this data would weaken LeVay's claim that one is "born" homosexual. In a paragraph devoid of sympathy for abused children, LeVay addresses the claim that homosexual molestation may influence sexual orientation and says:

> For this to be true, however, we would have to assume that children or adolescents were sexually passive targets for molestation by their elders. In reality, it is likely that many of them, especially the adolescents, already felt sexually attracted to same-sex partners. If so, they may have initiated the contacts or responded willingly to the older person's advances. Even if not, the older person may have picked up on the cues that were indicative of the child's future sexual orientation and selected the child on that basis.[228]

LeVay blames young children for being molested. The children may actually have "been attracted to same-sex partners." The children possibly "initiated" the sexual contact with an adult. Even if an adult initiated the contact, the children "responded willingly." This calloused and cruel view of homosexual child molestation demonstrates a man accustomed to same-sex attraction forcing his own warped worldview on innocent boys and girls. Essentially he says, "They really wanted the adult to have sex with them!"

LeVay's opinion is not an isolated one among homosexual authors. Wilson and Rahman make a similar claim in their 2005 book *Born Gay*. After summarizing some research on homosexual sex-play among children, they claim boys who had homosexual play with other boys knew about their purported homosexual orientation prior to these childhood encounters. Wilson and Rahman then say this "strongly suggests that childhood sex play or willing sexual activity with unrelated older males is

[228] Simon LeVay, *Gay, Straight, and The Reason Why: The Science of Sexual Orientation* (New York: Oxford University Press, 2011), 35.

not a cause, but rather a consequence, of inborn early homosexual feelings."[229] Much like LeVay, these authors suggest some children abused by pedophiles were not actually seduced, but desired the contact because they were "born this way."

In fact, the age of sexual debut, the context in which it occurred, and the age and sex of the person with whom the sexual contact occurred all have a strong organizing effect on one's later sexual identity.[230]

We live in a culture imposing adult sexuality on young children. This is exactly the vision of Alfred Kinsey. Recall his inability to identify the pain of children being molested by adults. He believed children should be exposed to sexual variations and practices in order to overcome inhibitions to sexual experimentation. Kinsey's vision has arrived and American children suffer. In fact, parents who oppose the sexualization of children are called narrow-minded while the descendants of the Sexual Revolution assert their own enlightened sexual views. Meanwhile, boys and girls are forced to learn about adult matters at younger and younger ages and innocence is lost.

Worldview War

The worldview war at the heart of scientific research and homosexuality is more intense than most evangelicals realize. Modern psychiatry asserts that sexual orientation cannot be changed. Thus, when we call people to repent of their sin, many mental health professionals believe that homosexuality should be excluded from this discipline. Most Christian ministers may be unaware of how strongly this idea of an "immutable" sexual orientation is embedded within modern mental health

[229] Glenn Wilson and Qazi Rahman, *Born Gay: The Psychobiology of Sex Orientation* (London: Peter Owen Publishers, 2005, 2008), 36.

[230] Because even homosexual activists acknowledge the influence sexual abuse can have on sexual identity, I am quite perplexed to read Frankowski say, "Although there continues to be controversy and uncertainty as to the genesis of the variety of human sexual orientations, there is no scientific evidence that abnormal parenting, sexual abuse, or other adverse life events influence sexual orientation." Frankowski, "Sexual Orientation and Adolescents," 1828.

doctrine. When someone comes to a pastor and admits homosexual sin, a faithful pastor will encourage confession, repentance, and prayers to God for forgiveness. Furthermore, a pastor will encourage the development of spiritual disciplines such as prayer, scripture memory, fasting, and accountability partners in order to find victory over sin. In contrast, many (most?) mental health professionals would consider this type of spiritual counsel counter-productive to the mental health of someone involved in homosexuality. Instead, this type of pastoral care is considered harmful to the well-being of an individual and in fact they believe this will push the person deeper into conflicted feelings about their sexuality. Modern psychiatry argues the healthiest thing a person can do is accept their own sexual orientation, embrace it in a healthy manner, and celebrate who they are.[231]

The worldview collisions associated with homosexuality are clearly seen in the personal examples of many of the researchers attempting to find genetic or biological causes to homosexuality. As was noted, Kinsey was sexually adventurous. Dean Hamer is in a same-sex marriage to Joe Wilson. The two of them produced *Out in Silence*, a film with the stated purpose of changing the way rural communities and small towns view homosexuality. Simon LeVay is an open homosexual and avid activist for gay rights. Richard Pillard was the first openly gay psychiatrist in the United States. Mustanski is the director of the Impact GLBT Health and Development program at Northwestern University. Michael Bailey generated controversy in 2011 when he let two sexual "exhibitionists" perform sex acts in front of a college class.[232] The idea that these are coldly analytical scientists with no biases is ludicrous: These are agenda-driven activists with a significant emotional investment into the cause of sexually libertine morals and gay rights.

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[231] With this strong animus against the Christian position, it is difficult to believe the current Department of Defense policy -- allowing military chaplains to preach within their faith convictions about homosexuality -- will be retained for any length of time. To be blunt: In the armed forces, chaplains who tell people to repent of sexual sin will be considered part of the problem and not the solution. More likely, the current policy is a half-step towards prohibiting military chaplains from publicly or privately suggesting homosexuality is sinful.

[232] "NU Cancels Human Sexuality Class," CBS 2 Chicago, May 9, 2011, accessed January 31, 2013, http://chicago.cbslocal.com/2011/05/09/nu-cancels-human-sexuality-class.

Summary

There are genetic and biological aspects to same-sex attraction and homosexual orientation, with some evidence pointing to a correlation between certain variables and same-sex attraction. This should not surprise us. The Christian worldview asserts that we are both body and soul. Because this is so, we are not doomed by a genetic predisposition towards any number of sinful behaviors. The Christian worldview also asserts that the entire creation has been damaged by a historical Fall wherein sin entered the world and distorted everything, including our biological and genetic predispositions. Homosexuality is rooted in multiple factors and future research will probably address different "homosexualities" as opposed to one form of "homosexuality." A strong inclination towards a particular behavior does not prove that all behavior is necessarily good or that we should suspend moral judgment about the behavior.

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