What is Homosexuality?

A Survey of the Scholarly Literature

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Both before publishing this paper and since, I have been unable to find significant discussion of the scientific evidence concerning the nature of same-sex attraction (SSA), outside the scientific journals themselves. Notably, none of the dissenting opinions in the decision of the Supreme Court of the United States in *Obergefell v. Hodges*, which legalized same-sex marriage throughout the United States, made any appeal to the scientific literature on SSA. It's as if the claims that SSA is inborn and immutable had been established as scientific fact – even though the science is strongly to the contrary.

Meanwhile, the Church's response to the successes of the gay rights movement, particularly post-*Obergefell*, has been primarily to quote scriptural prohibitions against homosexual behavior, without actually disputing claims that SSA is inborn and immutable. This, I believe, is a dangerous oversight. These are *fact-claims*: either homosexuality is inborn and immutable, or it is not. Quoting scripture while neglecting the science of SSA leaves the fact-claims unchallenged, implicitly conceding them. Given the clear scriptural prohibitions against homosexual behavior, this either removes homosexuals from God's grace, bringing His compassion and justice into question, or what is probably worse, it makes the scriptures seem irrelevant.

Such a monumental and damaging concession would be unnecessary if it should appear that there is no scientific basis for gay advocates' fact-claims. Evidence, therefore, gained through empirical investigation, is an appropriate response to such claims – not theology or exegesis alone.

My review of over 150 scientific articles, published in the leading scholarly journals, in my opinion clearly justifies the following conclusions:

1. Biological factors probably contribute causally about 20%-30% to the incidence of SSA. Genetics, however, is probably not involved. Instead, the biological factors are probably in the nature of epigenetic programming errors. This suggests that SSA is in the nature of a disorder. Other evidence supports this conclusion, including the association of SSA with mental disorders and other social dysfunctionalities.

2. Experiential factors, particularly childhood sex abuse and parental influences, probably causally contribute at least another 10% to the incidence of SSA.

3. The rest of the incidence of SSA may be attributable to individual free choice, as is suggested by the fact that homosexuals do change their sexual preferences and even their sexual orientations, spontaneously or through counseling, in large numbers.

Of course these conclusions do not justify unkindness toward homosexuals. God loves them, and the Church is called to serve them. If SSA is a disorder, however, then the unkindness would be in causing them to believe otherwise.

But the science of SSA does not merely constitute a valuable basis for pastoral appeal to those entangled in the homosexual lifestyle. It also has the potential for vindicating the biblical view to a wider community which, uncomprehending of the church's stance, is already reluctant to defend core principles of religious liberty against LGBT overreach. Leveraging the science, then, may both promote the Gospel and protect the Church and her educational and other eleemosynary institutions against adverse legislative and judicial action.

One: Introduction

The Church is Being Pressed to Change

For at least 45 years the church[1] has found it necessary to respond to the demands, both political and ecclesiastical, of gays and lesbians. Now, the Supreme Court of the United States will soon issue its decision about the constitutionality of bans against same-sex marriage. Christian bakers, photographers, and florists who decline to serve at same-sex weddings are facing ruinous fines. The church is being warned that by her opposition to gay marriage, she is alienating younger Americans who disproportionately support it. It is even said that as and when the churches' governing bodies become increasingly controlled by younger people, they will one-by-one capitulate to the cultural "tsunami" which is now washing across America.

All of this implicates me personally in many ways – as a human being, as a citizen, as a father, as a lawyer, and as a Christian. The last 45 years happens to coincide with nearly all of my adult life – I was a college sophomore at the time of the Stonewall riots of 1969. I was in law school in 1971 when the Oregon Legislative Assembly repealed the prohibition against sodomy for consenting adults. I became personally involved in the debate in 1996 as a member of the New Community Meeting, a conference of gays and Christians who reached agreement on a statement of principles for respectful political dialogue over the issue of gay rights. Later, many members of the same conference, including myself, met to foster communication among the University of Oregon and the gay and church communities concerning the evangelical Promise Keepers' use of Autzen Stadium for an evangelistic gathering.

In that course of time I formed an opinion about homosexuality, but until I began this survey of the scholarly literature on that topic, my opinion was based on a plethora of facts and opinions I'd read or heard from a thousand different sources which I could no longer identify, and I would not have been able to defend my views with any authority. Indeed, never having studied the matter carefully, there was always the possibility that I was mistaken about some important aspect. Is there a genetic component to same-sex attraction (SSA)? Can homosexuals change?

So as I began this survey, I wanted to know: To what extent, if any, does the scholarly research into SSA enable us to know what homosexuality is? Is there any tension between the science and the biblical prohibitions against homosexual activity, and if so, how much?

There is a fundamental mismatch between the arguments which are being made as to why the church should, and why she should not, soften her view concerning SSA. The claim that SSA is inborn, and the claim that homosexuals cannot change – indeed, the claim that

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[1] When I employ the phrase, "the church," I am usually referring to the universal church, the people who now or who have ever belonged to the Lord Jesus Christ.

encouraging them to attempt to change is cruel, since it inevitably leads to a deeper sense of failure – are fact claims: either SSA is inborn, or it is not, and either homosexuals can change, or they cannot. The Hebrew and Christian scriptures have an indirect bearing on such fact-claims, since it is unlikely that God, who made us all, would condemn behaviors in which the individual does not freely choose to engage; but modern science affords ways to address such questions more directly, namely, empirical investigation. Neglecting the science of SSA leaves the fact-claims unchallenged, implicitly conceding that SSA is indeed inborn and immutable – which, given the clear scriptural prohibition, effectively removes homosexuals from God's grace, bringing His compassion and justice into question. Such a monumental and damaging concession would be unnecessary if it should appear that there is no scientific basis for these fact-claims. Evidence, therefore, gained through empirical investigation, is an appropriate response to such claims – not theology or exegesis alone.

Two Books

The Bible teaches that God is the Creator of the universe, and that his deity and power are plainly revealed in what he has made. In his letter to the Romans, the Apostle Paul said that "the invisible things of him from the creation of the world are clearly seen, being understood by the things that are made, even his eternal power and Godhead; so that they are without excuse." Romans 1:20. That is, God has revealed himself not just in scripture, but also in nature. We may have much to learn about him, then, by studying nature. Just as importantly, what we learn about God from nature may inform our understanding of scripture, and our understanding of scripture may inform our investigation of nature. This is the "Two Books" model for understanding the relationship between faith and reason. According to this model, since God is the author of both Books, they cannot contradict each other. Therefore, if we find that the book of scripture and the book of nature appear to be incompatible, we may assume that it is because we have not understood one or the other, or both, and we must therefore question them both more carefully.

If this is true, then it is not enough to rely on one of the Books to the neglect of the other. If God has revealed Himself in nature, then we must study nature in order to discover that revelation. We call this Science; and if we wish to understand homosexuality, we do not merely consult the books of Deuteronomy and Romans: we also consult the scientific literature on the subject.

The controversy concerning SSA and the church is a perfect illustration of this. Since the Old and New Testaments of the Bible characterize homosexual acts as sinful, Christians and Jews ought to expect science to confirm that view. We would then regard science as a potential resource for defending the biblical view and for relieving the wider community's incomprehension of our opposition to homosexuality.

The Danger is Great, but so is the Opportunity

In this context there is a great need for a response from the church which is both intelligent and welcoming. The opportunity for healing, should the church rise to this challenge, is as great as the danger of even greater alienation, should she respond in fear or in condemnation. This is true of relations between the church and society, and it is also true of our personal relationships. One of my strongest motivations for conducting this survey has been to understand SSA well enough to be able to explain to my family, friends, and acquaintances what I believe and why, in the wistful hope that they will not think less of the Lord Jesus because of me. What is needed is an honest and open conversation based on the truth about SSA. The church should be the first to model that kind of citizenship.

To become more intelligent in our response to the claims of gay rights advocates, it is necessary to inform ourselves of the facts concerning homosexuality, to the extent to which those facts are accessible to us. Indeed, we are responsible to the wider community to do so, since they are looking to the church for an answer. To become more welcoming to homosexuals, we should regard the present political climate not as a threat but as a great opportunity to foster more open dialogue leading to an increase in trust and good will. In this time the world is watching the Supreme Court of the United States, certainly; but it is also watching the churches, the seminaries, and the bible colleges of the United States. This is our moment to live out the self-giving, redeeming love of God.

A Preview

My study has made it clear that while I was correct in my previous belief that homosexuality is not well understood, much more research has been done on the question than I had realized. The literature consistently indicates that homosexuality results from many causal factors, among which are one or more biological factors – genetic, or epigenetic, or hormonal, or a combination of these. Also, as a class, homosexuals have distinctive brain differences – although no study has determined whether this is cause or effect of homosexuality. Additional evidence that a predisposition toward homosexuality may be partly inborn is found in the distinctive trait patterns which homosexuals exhibit. Such findings preclude simplistic notions of homosexuality as a moral failing and nothing else.

Despite all of this, however, experiential and individual factors still seem to account for most – probably about 70% to 80% – of the incidence of SSA. Moreover, the association of homosexuality with physical and mental illness and social dysfunction renders it problematic to assume that homosexuality is good for the individual or for society.

When I refer to "experiential" factors, I am referring to the influence of the post-natal environment: the affect of abuse, neglect, parental and peer group influences, and the like, upon

the incidence of homosexuality. Some of these factors are relatively well-studied in the literature, and have been quantified to some extent.

By "individual" factors I am referring to the free choices which people make concerning their sexuality. As best I can see, these factors have been completely ignored in the literature. This presumably reflects the naturalistic bias of most researchers: there is heredity, and there is environment: there is nothing else, because human free will is an illusion. It must be emphasized that science has not demonstrated this to be the case. We will return to this question at the end of the paper.

A current statement from the American Psychological Association encourages me to think that my study may be found useful. It also illustrates why we cannot uncritically accept what others, even the experts, say about this very difficult subject. The APA writes:

> . . . [A]lthough much research has examined the possible genetic, hormonal, developmental, social, and cultural influences on sexual orientation, no findings have emerged that permit scientists to conclude that sexual orientation is determined by any particular factor or factors. . . .
>
> . . . [L]esbian, gay, and bisexual orientations are not disorders. Research has found no inherent association between any of these sexual orientations and psychopathology. Both heterosexual behavior and homosexual behavior are normal aspects of human sexuality." (https://www.apa.org/topics/lgbt/orientation.pdf).

Here we see that the professional organization most deeply involved in developing a scientific understanding of homosexuality states that although it is not understood what causes SSA, it nevertheless may confidently be stated that it is a normal expression of human sexuality. It seems a bit surprising that anything can be declared normal when its cause is not understood.

We shall see that homosexuality is better understood than the APA suggests.

The Usual Caveats

Several qualifications need to be made. I do not claim to have reviewed all the evidence relevant to the question of the nature of SSA. I do not claim to be as qualified as many others to interpret the evidence. On the other hand, I am not willing to permit others to do my thinking for me on this question. Gays and lesbians have a stake in gaining the approval of society, and of the church in particular, for the way they express their sexuality; therefore I cannot accept their claims at face value. At the same time, many of the arguments of leading evangelicals so far have been based on the biblical texts, often to the neglect of the scientific evidence concerning SSA.

And so I set myself to determine to my own satisfaction, if possible, what SSA is, based on the best scientific evidence which might be available. Having done so, to the very modest limits of my ability within the time available to me, I share my findings with anyone who cares about the mission of the church, the well-being of the several million of our fellow citizens who are homosexual, or the health of our political community.

Two: Demographics

Some wild claims have been made concerning the incidence of homosexuality in the general population, but the literature provides consistently modest numbers: the percentage of the population self-identifying as either exclusively or predominantly homosexual or bisexual has been found to be between 1.4% and 2.9%. In interpreting such results, it is important to bear in mind that they are based entirely on self-reported sexual identity. Also, the various studies often measure very different things: some measure behavior, others measure same-sex attraction, and still others measure sexual identity. Some lump homosexuality and bisexuality together, while others assess them separately, making comparisons difficult.

A 2001 study of 13,000 New Zealand adults (age 16+) found that 0.8% were homosexual, 0.6% bisexual, 0.3% "something else," and 0.1% were "not sure." 3.2% reported some same-sex experience, and 1.9% reported a same-sex relationship. (Wells, 2001.)

Another study, based on a sample of 967 participants (469 males, 498 females) in the Christchurch (NZ) Health and Development Study, found 2.8% to be predominantly or exclusively homosexual, but another 9.6% were predominantly heterosexual but had some same-sex inclinations or experience. (Fergusson, 2005.)

In 2007 Savin-Williams discussed earlier studies which had found that exclusive same-sex behavior in the United States, the United Kingdom, and France rarely characterized more than 1% of the adult population (Sell, Wells, & Wypij, 1995); in a representative survey of U.S. adults, 8% reported at least some degree of same-sex attraction, 7% had at least one same-sex behavior since puberty, and 2% identified as gay/lesbian/bisexual (Laumann, Gagnon, Michael, & Michaels, 1994); and about twice as many had engaged in same-sex behavior as actually identified as gay/bisexual (Black, Gates, Sanders, & Taylor, 2000).

Surveys from 2002 to 2006 in Massachusetts found a prevalence of 1.9% for homosexual identity and 1.0% for bisexual identity compared with New Zealand estimates of 0.8% and 0.6%, respectively (Wells, 2011).

Varnell found that there are about twice as many gay men as lesbians (Varnell, 1999). Pattitucci found that bisexuality in males was "essentially absent," but that in women bisexuality is "equal in stability to both heterosexual and lesbian orientations, suggesting a trimodal character for sexual orientation in women." (Pattitucci, 1998.)

Table 1 is a summary of the foregoing findings.

Table 1. Summary of Demographic Findings

Exclusively homosexual:		0.8-1.9%
	Wells 2001	0.8 (NZ)
	Savin-Williams	1.0
	Wells 2011	1.9
Exclusively or predominantly homosexual:		2.8%
	Ferguson	2.8 (NZ)
Bisexual:		0.6-1.0%
	Wells 2001	0.6 (NZ)
	Wells 2011	1.0
Exclusively or predominantly homosexual or bisexual:		1.4 - 2.9%
	Wells 2001	1.4
	Savin-Williams	2.0
	Wells 2011	2.9

If there are twice as many gays as lesbians, then the range of 1.0% to 1.9% (excluding New Zealand) for exclusive homosexuals would have to be averages for the two sexes, and the rate for males would have to be twice the rate for females. If the combined rate is 1%, then the rate for females would be 0.67% and the rate for males would be 1.33%. If the combined rate is 1.9%, then the rate for females would be 1.27% and for males, 2.53%. This does not include bisexuals, all or nearly all of whom are women.

Three: Brain differences

Researchers hoping to discover the roots of homosexuality have compared several brain structures of homosexuals with those of heterosexuals in attempts to identify consistent differences. Structures of interest have been the hypothalamus, the anterior commissure (AC), the corpus callosum (CC), the amygdalae, and brain hemispheric symmetry/asymmetry. Based on results so far, there is evidence that the hypothalamus, the amygdalae, and brain asymmetry have some association with homosexuality, and that the AC and the CC do not.

The Amygdalae and Brain Hemisphere Asymmetry

In a 2008 MRI and PET scan study of 90 individuals, Savic concluded that heterosexual men and homosexual women showed right hemispheric asymmetry and greater connectivity from the right amygdala, while homosexual men and heterosexual women showed hemispheric symmetry and greater connectivity from the left amygdala, suggesting a linkage between sexual orientation and these neurobiological entities. (Savic, 2008.) As the discussion in the section following suggests, such structural differences may be implicated in some of the trait differences in homosexuals.

The Hypothalamus

The hypothalamus is an almond-sized brain structure located just above the brain stem which, according to the National Institutes of Health, "produces hormones that control body temperature, hunger, mood, the release of hormones from many glands, especially the pituitary gland, sex drive, sleep, thirst, [and] heart rate." http://www.nlm.nih.gov/medlineplus/ency/article/002380.htm. In 1991 LeVay measured the volumes of four cell groups in this region (interstitial nuclei of the anterior hypothalamus (INAH) 1, 2, 3, and 4) in postmortem tissue from three subject groups: women, men who were presumed to be heterosexual, and homosexual men. LeVay concluded that INAH 3 was more than twice as large in heterosexual men than in either women or homosexual men. LeVay mentioned as a caveat that "Brain tissue from individuals known to be homosexual has only become available as a result of the AIDS epidemic. . . . [U]ntil tissue from homosexual men dying of other causes becomes available, the possibility that the small size of INAH 3 in these men reflects a disease effect that is peculiar to homosexual AIDS patients cannot be rigorously excluded."

But in 2001 Byne found that the reduction in the volume of INAH 3 results from increased density of the neurons, not from a decrease in the number of neurons, and stated that there is no known connection between an increase in density and sexual orientation. Indeed, there is a mechanism known to occur in animals in which such an increase in density occurs post-natally. "At present . . . we can neither ascribe any function to INAH3, nor can we interpret the

functional significance of its sexual dimorphism. If INAH3 is a site related to the functional circuitry of sexual orientation, then the current data suggest that measures other than simple nuclear volume are needed to discern the relationship. Based on the results of the present study as well as those of LeVay (1991), sexual orientation cannot be reliably predicted on the basis of INAH3 volume alone."

In 2005, however, Savic conducted positron emission tomography (PET) scans of heterosexual men and women and of homosexual men while they sniffed putative male and female pheromones. The homosexual men's and the heterosexual women's hypothalamuses were activated by male pheromones and not female pheromones, while the heterosexual men's hypothalamuses were activated by the female pheromones and not the male pheromones. (Similar results were obtained in a 2006 study of putative human pheromones and lesbian women. (Bergland, 2006.)) Said Savic:

> The difference between HoM and HeM could reflect a variant differentiation of the anterior hypothalamus in HoM, leading to an altered response pattern. Alternatively, it could reflect an acquired sensitization to AND [the male hormone] stimuli in the hypothalamus or its centrifugal networks, due to repeated sexual exposure to men. A third possibility is that HeW and HoM associated AND with sex, whereas HeM made a similar association with EST [the female hormone]. These tentative mechanisms are not mutually exclusive, nor can they be discriminated on the basis of the present PET data.

Thus again, there is evidence of an association between SSA and the hypothalumus, but its significance is uncertain.

The Anterior Commissure and the Corpus Callosum

The anterior commissure (AC) is a bundle of nerve fibers, and the corpus callosum (CC) is a much larger bundle, both connecting the two cerebral hemispheres. A study by Allen and Gorski examined the AC, finding that females and homosexual males exhibited a larger size than heterosexual males, and data collected before 1910 from cadavers seemed to show that males have larger CCs than women. However, later studies using larger sample sizes found no such differences. Bishop, 1997; Lasco, 2002. Bishop stated:

> A meta-analysis of 49 studies published since 1980, most of which used magnetic resonance imaging (MRI), reveals no significant sex difference in the size or shape of the splenium of the corpus callosum. . . . The widespread belief that women have a larger splenium than men and consequently think differently is untenable.

And Lasco, et al. reported that they had "examined the cross-sectional area of the AC in postmortem material from 120 individuals, and found no variation in the size of the AC with age, HIV status, sex, or sexual orientation."

Reverse causation

Complicating the issue of brain differences between homosexuals and heterosexuals is the problem that sexual experiences themselves can affect brain structure. In 1997 Breedlove found that rats allowed to copulate by being caged with receptive females developed brain structures different from those which were not allowed to copulate by being caged with non-receptive females. "Copulatory experience can therefore alter the size of neurons. . . . Whether the sensory experience or motor activity of copulation induced these morphological changes, interpretations of correlations between human behaviour and neural morphology must acknowledge that the two are reciprocally related. It is possible that differences in sexual behaviour cause, rather than are caused by, differences in brain structure."

Four: Trait Differences

Homosexuals exhibit abilities and other traits more commonly associated with the opposite sex, and many of these differences have been studied and quantified. It is difficult to account for many of those trait differences without positing some sort of organic etiology. As Rahman observed in 2003:

> . . . [H]omosexual males perform poorly on mental rotations and better on verbal fluency tasks as compared to heterosexual males. . . . Gay men are also less accurate on visuomotor targeting tasks (typically male advantage) compared to heterosexual men, and no more accurate than heterosexual women. . . . The profile may be present in childhood, as gender-nonconforming boys show diminished spatial ability compared to controls. . . . Interestingly, we have recently discovered that homosexual men perform in female-typical directions on object location memory, a spatial task which usually shows a modest effect size in favour of females. . . . This suggests that gender-atypical shifts cut across the de-coupling in some spatial functions, e.g. mental rotation (male-favouring) versus location memory (female-favouring).
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> . . . [T]he data fit well with the notion of differential interhemispheric "cross talk," mediated by the anterior commissure (AC). The larger AC of homosexual men and heterosexual women may contribute to greater inter-hemispheric transfer, and thus bilateral representation, of language functions, specifically verbal fluency. . . This is consistent with the findings of greater verbal fluency, and lower visuo-spatial ability, in homosexual men and heterosexual women.
>
> Greater inter-callosal transfer could also occur through the larger isthmus of gay men. . . . A larger isthmus would allow greater interconnection of right and left speech regions. This may determine the pattern of varying linguistic function and increased prevalence of non-right handedness in homosexual men.

I was puzzled by Rahman's reference to homosexual men's larger anterior commissures and corpus callosa, in light of Bishop and Lasco's then-recent findings (no variations in size in the AC or the CC, page 8, above). I searched for subsequent literature on the question and what I found is that as recently as 2012 commentators were still citing the earlier studies refuted by Bishop and Lasco, with no mention of Bishop and Lasco's work. Whether this represents neglect, or obfuscation, or something else, I don't feel qualified to say; but lacking a basis for discounting Bishop's and Lasco's findings, I find Rahman's theory about the etiology of gay-straight trait differences unpersuasive. Obviously, that doesn't mean there cannot be other brain differences – one might be more likely to suspect the involvement of brain hemispheric symmetry/asymmetry and sex-crossed amygdalae connections, and/or other undiscovered brain differences. But these trait differences seem quite real – indeed, some of them are so widely recognized as to be folklore.

There is evidence of an association between childhood gender nonconformity and homosexuality. "That is, gay men tend to recall having been feminine boys, and lesbians, masculine girls." (Bailey, 2000.) Bem (1996) found that 63% of adult homosexual subjects recalled that they had not enjoyed sex-typical activities as children, compared to 10% of heterosexuals, and that 48% of homosexuals recalled that they had enjoyed sex-atypical activities as children, compared to 11% of heterosexuals.

There is a weakness in such retrospective studies, in that they are reliant on the accuracy of recollections, which may be affected by subsequent experience, politics, and group solidarity. Some prospective studies were conducted earlier with that in mind. Green, 1987, and Zuger, 1984, in longitudinal studies of their own patients (*i.e.*, not representative populations) found that 75% and 92% of two samples of extreme cross-gendered boys were non-heterosexual in adulthood. Both of those studies involved patients who happened to be involved in a clinical practice – that is, young people whose behavior was sufficiently problematic for them to seek treatment, or for their parents to seek treatment on their behalf. I was unable to find more recent studies, or any involving more representative samples.

Five: Prenatal Factors

Two factors have been identified as potentially affecting sexual orientation during gestation: the exposure of the fetus to male hormones, and the fraternal birth order effect.

Androgen

The occurrence of several very rare maladies attributable to male hormone anomalies has attracted many researchers' attention to the role which the prenatal hormonal environment may play in the etiology of homosexuality. Androgen insensitivity syndrome (AIS), for instance, which affects 2 to 5 in 100,000 (0.002% to 0.005%, or two one-thousandths to five one-thousandths of one percent), results, in its extreme form, in a genetic male with female anatomy and a sexual preference for males. (Rahman, 2003; http://ghr.nlm.nih.gov/condition/androgen-insensitivity-syndrome.) Congenital adrenal hyperplasia (CAH) syndrome, according to Ellis (1987), is a "genetically caused sexual inversion . . .in genetic females. . . . [In CAH], a genetically controlled enzyme deficiency causes testosterone production in male-range quantities by the fetal adrenal glands instead of cortisol (Money & Dalery, 1976). . . ." According to Burr (1993), studies showed that 37% of CAH females identified themselves as lesbian or bisexual. About 1 in 15,000 have CAH, or .007% (http://www.nlm.nih.gov/medlineplus/ency/article/000411.htm). If 3% of women are lesbian, then CAH may account for 0.231% of lesbians, or about 1 in 2,300.

There is evidence (see next section) of a "fraternal birth order effect" – that the more older brothers a male has, the more likely he is to be homosexual. (Williams, 2000.) (Zietsch's 2012 twin study, the largest to date, was negative regarding the fraternal birth order effect.) Williams found that the more older brothers a male has, the smaller (more masculine) will be the ratio of the length of his index finger ("2d") to the length of his fourth finger ("4d"). The inference is then made that male SSA results from an *excess* of male hormone. McFadden (2002) also proposes an excess of prenatal androgens for male homosexuals because certain features of their auditory systems are "hypermasculinized."

These results are of course confusing: it seems unlikely that excesses in prenatal androgen can cause both heterosexuality and homosexuality.

Obtaining data about prenatal hormone environments is difficult. It would be unethical to administer hormones to pregnant women experimentally to determine the effect on their offspring's sexual orientation. But circumstances have created some limited opportunities to assess the influence of gestational hormones. The study of certain developmental disorders and the prescription of hormones to pregnant women have yielded some information, and there have been studies which measured hormones in amniotic fluid or maternal blood and which have related those measures to the later behavior of their offspring.

In the 1940s, 1950s, and 1960s, a synthetic estrogen known as diethylstilbestrol (DES) was prescribed to 1 million to 5 million pregnant women in the U.S. in the mistaken belief that it would help to prevent miscarriage. Hines (2011) assessed for SSA 97 women exposed *in utero* to DES, together with a control group, and found that 6% of the controls and 24% of the DES-exposed women exhibited other than exclusive or near-exclusive heterosexuality.

An earlier, much larger study, however (Titus-Ernstoff, 2003, 3,946 women exposed prenatally to DES and 1,740 women not exposed to DES), reached the opposite conclusion.

And as Hines notes, "The possibility that exposure to ovarian hormones before birth influences sexual orientation in males also has been investigated. These studies have produced largely negative results. . . ."

Hines states that adult homosexuals and heterosexuals do not manifest identifiable hormonal differences; but she proposed that it might be possible to make a connection between prenatal hormonal environments and adult SSA indirectly, by first making a connection between prenatal hormonal environments and sex-atypical play in children, and then making another connection between sex-atypical play in children and adult SSA. In her 2001 study, Hines cites an earlier study of her own which had shown that normal variation in maternal testosterone during pregnancy accounted for 2% of the variance in the gender role behavior of pre-school girls, but not boys, together with two studies (Green 1987, Zuger, 1984, both mentioned above, page 11) showing that 75% and 92% of two samples of extreme cross-gendered boys were non-heterosexual in adulthood; and she concluded:

> This article [*i.e.,* Hines, 2002] addressed the question of whether gonadal steroid exposure during prenatal development is one of the factors, in at least one of the pathways, that lead to variability in sexual orientation outcomes. Based on the compelling evidence that prenatal testosterone exposure influences children's sex-typical play behavior, on the well-established links between childhood play interests and adult sexual orientation, and on the evidence showing altered sexual orientation in women exposed to high levels of androgens prenatally, because of CAH, the answer appears to be "yes."

This conclusion would seem to be more than a little overstated. Perhaps the weakest link in Hines' argument might be the supposition that there is "compelling evidence that prenatal testosterone exposure influences children's sex-typical play behavior." A 2% shift among girls only does not seem impressive or "compelling." Also, the findings that certain very rare hormonal disorders are a factor in a minuscule portion of the homosexual population is of no clear significance at all in discerning the causes of the phenomenon of homosexuality more broadly speaking. There is no reason to assume that the mechanisms leading to AIS or CAH are responsible for SSA generally.

To her credit, Hines herself mentions several other reasons to question the role of hormones in SSA:

It also is noteworthy that, despite the evidence that many women exposed to high levels of androgens prenatally because of CAH are not heterosexual, most, even many of those with the most severe form of the disorder, are exclusively or almost exclusively heterosexual. Similarly, although many homosexual men and women recall cross-gendered childhood interests, this is not universal. In addition, most people who are not heterosexual have no evidence of prenatal hormone abnormality. It is possible that these individuals have hormonal differences in circumscribed brain regions, or abnormalities downstream from testosterone itself, e.g., in PGE2, but such possibilities are highly speculative. In addition, factors other than the early hormone environment are likely to be important for the development of sexual orientation. There is at present very little information, however, as to exactly what these factors might be.

Fraternal Birth Order Effect

Several studies seem to show that the incidence of homosexuality in men is increased according to the number of older brothers that they have. The most popular explanation is that "a maternal immune response provoked by male-specific foetal products shifts male-typical neurodevelopment in a sex-atypical direction. The immune system 'memory' keeps a tally of preceding male foetuses, and modulates its response accordingly, usually a stronger one." (Rahman, 2003.) Rahman reports that "It has been estimated that each older brother increases the odds of being a homosexual man by 33–48%," citing Blanchard and Bogaert (1996b) and Blanchard and Klassen (1997)(which I have not reviewed).

Blanchard (2004) states that "Calculations assuming a causal relation between older brothers and sexual orientation have estimated the proportion of homosexual men who owe their sexual orientation to fraternal birth order at 15% in one study and 29% in another." And Blanchard also found that the effect is more pronounced in right-handed than in left-handed homosexuals. (Blanchard, 2008.) Camperio-Ciani (2004) estimated that the number of older brothers accounts for 6.7% of the variance in sexual orientation.

Zeitsch, 2012, however, in the largest twin study to date, found no such effect. "The number of older brothers had little effect on . . . sexual orientation. . . ." And he cites another study (Bogaert, 2010) which also obtained negative results.

Six: Genetic Factors

Population studies in recent years suggest that there could be one or more genetic factors associated with increased incidence of homosexuality. There is evidence that male siblings of homosexuals are six to ten times more likely to themselves be homosexual than the siblings of heterosexuals (Pattitucci, 1998). Another study found elevated levels of homosexuality in the distant relatives of homosexuals. (Hamer, 1993.) A number of studies have found a prevalence of homosexuality among homosexuals' maternal relatives (Pattitucci, 1998.) Camperio, 2004, estimated that about 14% of the variance in sexual orientation is accounted for by the maternal effect. And Rahman, 2003, concluded that "Homosexuals overall have a 39% greater likelihood of being non-right handed; gay men 34% and lesbians 91%."

Population studies involving large numbers of both identical and fraternal twins have been conducted since 1999, but they have been largely inconclusive. (Bailey et al. 1999; Bailey, 2000; Rice, et al. 1999b; Schwartz et al. 2010; Langstrom 2010.) Langstrom, 2010, was the largest twin study of same-sex sexual behavior up until that time. Data were taken from the 2005-2006 survey of all twins in Sweden. 3,826 twin pairs were involved in the study. The researchers compared the incidence of homosexual behaviors in identical twins of homosexuals with the incidence of homosexual behaviors in fraternal twins of homosexuals. They found that heredity accounted for 39% of the incidence of lifetime same-sex partners and environmental factors accounted for 61%. In women, the numbers were 18-19% and 81-82%. Siblings of homosexual identical male twins were more than twice as likely to themselves be homosexual than the siblings of homosexual fraternal male twins. In females, the ratio was only 1.3/1 to 1.5/1. The researchers concluded: "Our results support the notion that same-sex behavior arises not only from heritable but also from individual specific environmental sources." (Langstrom, 2010.)

The Langstrom study, however, despite its effort to obtain the largest possible sample, was hampered by the relatively low incidence of homosexuality:

> This study . . . was inevitably limited by the fact that same-sex behavior was relatively rare. With the unexpectedly low familial effects, the twin method has restricted power to detect statistically significant influences. For example, with 4% prevalence and a sample size of 2,000 twin pairs, there was 25% power to detect a heritability of 20% at the 5% level. . . . To achieve 80% power to detect significant genetic effects . . . one would need to increase the sample size 5-fold. . . . Consequently, though familial effects certainly are important for same-sex behavior, the exact magnitude of genetic and environmental contributions to these effects should be interpreted cautiously.

The difficulty becomes obvious when one considers the fact that only 7 out of 71 identical twin pairs were both SSA, and only 3 of 53 fraternal twin pairs were both SSA. Thus if there had been only 2 less concordant identical twin pairs and two more concordant fraternal twin pairs, it

would have appeared that having an identical twin with SSA would make one *less* likely to be SSA.

In 2012, Zietsch conducted an even larger twin study involving 9884 identical and fraternal Australian twins. He concluded that genetic factors accounted for 31% of the variance in sexual orientation. Langstrom, 2010, and Zietsch, 2012, seem to be the best measure of the extent to which biological factors affect SSA – although Langstrom critiqued Zietsch in advance when he stated that a five-fold increase in sample size would be desirable. Zietsch's sample was only 50% larger than Langstrom's. Zietsch's results should therefore also be viewed with caution.

A 30% contribution to a genetic condition is considered "weak." This can be illustrated by the fact that peer-reviewed studies have found genetic contributions of 50% to such behaviors as divorce (McGue, et al, 1992), depression (Walters, 1992), altruism (Rushton, 1984), religiosity (Bouchard, 1990), fundamentalism (Waller, 1990), psychological inpatient care (Allgulander, 1991), fear of the unknown (Stevenson, 1992), perhaps alcoholism (Horgan, 1993), and very interestingly, homophobia (Verweij, 2008). Bouchard, 1990, was a longitudinal study of 100 pairs of twins separated in infancy and reared apart. The author concluded that

> For almost every behavioral trait so far investigated, from reaction time to religiosity, an important fraction of the variation among people turns out to be associated with genetic variation. This fact need no longer be subject to debate. . . .

One remarkable thing about all such population studies is that none of them measure genetic factors directly. The incidence of homosexuality among those who are in certain familial relationships with homosexuals is compared with its incidence among those who are not. Since we know that families have a common genetic heritage, it is natural to assume that such differences result from genetic variation, but it is only an assumption.

Recent advances in genomic technologies are beginning to enable researchers to measure genetic differences directly through molecular studies. Statistical surveys indicating sex-linked genetic factors, such as the prevalence of homosexuality within gays' maternal relatives, have led researchers to focus on the X-chromosome. As Pattitucci explains, males have one X- and one Y-chromosome, while females have two X-chromosomes– this is what makes them male or female. This means that a woman may have a gene for a given trait on one of her X-chromosomes but not exhibit the trait; while a male who carries such a gene on his X-chromosome would always exhibit the trait. This is a well-known phenomenon with respect to various sex-linked diseases such as hemophilia. Hence, if there are one or more sex-linked genes which increase the likelihood of SSA, we would expect the rate of manifestation of that trait to be higher in males than in females. We would also expect the rate of manifestation of homosexuality among male relatives in the maternal lineage to be higher than in the paternal lineage of a family, since a male inherits his single X chromosome exclusively from his mother and never from his father.

Several genome-wide molecular scans have been conducted, producing conflicting results. In May 2015, however, the largest study by far (409 independent pairs of homosexual brothers) was published, tending to confirm the existence of genes on the X-chromosome linked to homosexuality in men, and also on one other chromosome. (Sanders, 2015.) The particular genes were not identified, only the chromosomes. The authors mention several genes on each chromosome which other studies have shown to be associated with sexual behaviors in mice or humans, suggesting the possibility that they could be the genes implicated in the linkages identified in this study. The authors do not attempt to estimate the strength of these associations.

The very notion that genetics plays a role in the formation of homosexuality is placed in doubt, however, by the largest genetic study of SSA to date, conducted by the on-line geneological service 23andme. The study involved 23,000 of 23andme's members whose sexual profiles were compared with their DNA. The result showed a complete absence of any genetic factor in SSA. The 23andme study can be found at http://blog.23andme.com/wp-content/uploads/2012/11/Drabant-Poster-v7.pdf.

What is going on here? Have we not already seen that SSA runs in families in many ways? It predominates in the maternal line; male siblings of homosexuals are six times more likely to be homosexual than male siblings of heterosexuals; and identical twins are 20% more likely than fraternal twins to be homosexual if the other member of the twin pair is homosexual. And yet identical twins have the same genes. Why are they concordant for homosexuality only 20% of the time? Recent discoveries in the infant science of epigenetics suggest another explanation, which we shall now consider.

Seven: Epigenetics

Epigenetics is the study of mechanisms which have the effect of turning genes on or off, resulting in variability in the expression of traits despite an unchanging genome. For example, many genes have methyl receptors. If a methyl receptor is filled with methyl molecules, the gene is "turned off" and is not expressed. According to Kaminsky (2009), "In the last decade, evidence has been accumulating that epigenetic modifications of DNA and histones can have *a primary role* in phenotypic outcomes. . . ." (Emphasis added.) Epigenetic changes can occur in childhood and even adulthood; but the vast majority occur prenatally. Saffery, 2011.

A 2012 study published in The Quarterly Review of Biology, 87(4), 343-368, "Homosexuality as a consequence of epigenetically canalized sexual development," proposes a non-genetic theory for SSA. The authors are William Rice, an evolutionary geneticist at the University of California, Santa Barbara; Sergey Gavrilets, a mathematician at the University of Tennessee; and Urban Friberg, an evolutionary biologist at the University of Uppsala. The article was reviewed January 2013 in the online magazine, *The Scientist*, and that review can be found at http://www.the-scientist.com/?articles.view/articleNo/33773/title/Can-Epigenetics-Explain-Homosexuality-/.

As previously noted, the twin studies discussed above assumed that they had identified genetic factors underlying SSA; but Rice, *et al*. point out that all of those studies employed a methodology incapable of distinguishing between genetics and epigenetics. All that was known about the subjects' genetic profiles is that they were twins of one type or the other (identical or fraternal). Their sexual profiles were established by question-and-answer surveys. Thus, the conclusion that sexual differences between identical twins and fraternal twins reflect a genetic factor for SSA was only an assumption – an assumption which is placed in serious doubt by the 23andme study and by the very recent discovery of a set of nongenetic, prenatal factors potentially affecting SSA, namely, epigenetics.

The fascinating theory of Rice, et al. rests on the following findings:

1. It is now well established that epigenetic modifications of a parent's genome sometimes carry over across generations and influence the phenotypes of their offspring. Mouse studies have shown this to be the case with respect to epigenetic modifications of genes expressed in male mice that feminize their brains and behavior.

2. Male human fetuses exposed to low levels of the male hormone testosterone are usually not feminized as a result, and female fetuses exposed to high levels of testosterone are usually not masculinized as a result, which suggests that some separate mechanism causes male fetuses to be more sensitive to testosterone and female fetuses to be less sensitive.

3. During normal development, epigenetic marks are erased from sperm and egg cells so that they can be epigenetically reprogrammed at the embryonic stage. However, sometimes some epigenetic marks are not removed, and as a result are passed on to the next generation.

Rice's theory, as the review in *The Scientist* puts it, is that "If epi-marks that direct sexual development are not erased correctly, a mother could pass down epi-marks that direct female development to her son, resulting in an attraction to men, and vice versa for a father and his daughters. . . ."

Rice, et al. conclude that "[E]pigenetics is . . . a probable agent contributing to homosexuality."

Kaminsky's 2009 study, mentioned above, was not concerned with SSA, but it also demonstrates the importance of epigenetics generally. He showed that epigenetics can make a profound difference in the expression of genes, even as between identical twins. Each member of a fraternal (*dizogotic*, or "DZ") twin pair has a separate placenta. They are *dichorionic*. Only about one-third of identical (*monozygotic*, or "MZ") twins, however, are dichorionic. The rest are *monochorionic*, having a single shared placenta for each twin pair. (Minnesota Center for Twin and Family Research, University of Minnesota, https://mctfr.psych.umn.edu/twinstudy/twin%20FAQ.html.) According to Kaminsky, researchers believe that if the fertilized egg splits into two individuals during the first four days after conception, the two MZ twins will be dichorionic; otherwise, they will be monochorionic.

Monochorionic MZ twins are at a disadvantage: according to Kaminsky, it appears that twins who share a placenta often share it unequally, with the result that they become very different from each other epigenetically. In fact, the degree of epigenetic difference between monochorionic MZ twins is *roughly equal* to the degree of genetic difference between DZ twins!

It should be emphasized here that the epigenetic effects of monochorionicity do not explain SSA. The phenomenon is described here only to illustrate the importance of epigenetics generally.

The study by Rice, *et al*. was discussed in a 2014 article by Ngun, *et al*., who wrote,

> We find the first two pillars of Rice, et al.'s model to be sound. We believe it is very likely that sex-specific epigenetic marks are (at least partly) responsible for sexually dimorphic traits including sexual orientation. . . . [T]he evidence concerning epigenetics and human sexual orientation that we have presented strongly suggests a link between the two.

Ngun's only criticism of the Rice study is that it assumed that both sexes are affected by epigenetics in the same manner. They question that assumption because there are several

differences in the expression of SSA in males as compared with females.

In its review of the Rice study, *The Scientist* noted that some people don't see a need to understand the epigenetics of SSA. I think that is understandable. Many people are satisfied to think that there is a biological basis for SSA, because that enables them to consider it to be "a part of natural human variation." But even supposing SSA to be part of natural human variation: if it were also established that the biological basis for that variation is erroneous epigenetic programming, then it would be necessary to recognize SSA for the disorder which it increasingly appears to be.

Eight: Taking Stock

So far we have looked at brain differences, trait comparisons, prenatal factors, genetic factors, and epigenetic factors. None of these factors, nor all of them together, account for all the data. I believe we have seen enough, however, to conclude tentatively that in all probability, somewhere in the interplay of all of these factors lies a biological underpinning for at least a partial causal theory of homosexuality. Before we proceed to a consideration of the possibility that other, non-biological factors also have significant explanatory power, let us assess what we have already learned.

We have considered the hypothalamus, the anterior commissure and the corpus callosum, the amygdalae and brain hemispheric asymmetry – all the various brain structures which have been thought to be candidates for clues about SSA – and have seen that the positive indications of possible connections to SSA are very limited. The only brain structures which the research consistently shows to be correlated with homosexuality are the reduced brain hemispheric asymmetry among male homosexuals, the increased asymmetry among female homosexuals, and the sex-reversed amygdalae connections in homosexuals of both sexes; and we do not know whether these differences represent a causal link with homosexuality, or if they do, which is the cause and which is the effect.

But there is an intriguing possibility in what would seem to be a likely relationship between brain structure differences and trait differences in homosexuals. Homosexual men are more like heterosexual women in their spatial perceptual and verbal abilities, homosexual women are more like heterosexual men in these respects, and the same differences have also been observed in cross-gendered children. These trait differences are of such a kind that they would not be expected to occur in the absence of corresponding cross-gendered brain traits as well. But does the correspondence indicate causation? If it does, which is the cause, and which the effect? Also, if brain differences are not caused by experience, then the question would be what does cause them, since there are no data showing whether or not there is any correlation between genes and sex-atypical brain structures.

The evidence of a pronounced tendency on the part of extreme cross-gender children to become homosexual suggests that if neuroanatomy is in play, then it probably is in play *before* the brain can be shaped by sexual experience. An alternate explanation might be that children become cross-gendered as a result of earlier sex abuse. (We will look at that possibility shortly.) But we have no evidence that cross-gendered children are brain-symmetry or amygdalae atypical – Savic's 2008 study involved adult subjects – and we do not know the frequency of sex abuse among cross-gendered children. These considerations suggest avenues for further research.

The likelihood of a connection between brain differences and SSA is strengthened by the studies in heredity, genetics, and epigenetics which we have considered. The molecular studies tend to confirm a role for genetics, and the family pedigree surveys suggest the existence of one

or more biological precursors. The 23andme study's finding of no genetic factor, together with Rice's and Kaminsky's findings on the importance of epigenetics, may reflect a reduced role for genetics, without impairing the case for the importance of biological factors more generally.

Despite the fact that no direct connection between genetic or epigenetic factors and brain structure has ever been made, it seems reasonably likely that if there are genetic or epigenetic differences in all or some homosexuals, they produce physical differences of some kind. Perhaps they produce atypicalities in brain symmetry or in left- or right-handed amygdala dominance. Although this may be the best theory we have to date, it remains conjecture, since some unknown factor might contribute to SSA, and it might be SSA and/or its consequent sexual activity which cause the asymmetry and the amydgalae reversal.

Another, weaker positive result from brain studies is Savic's finding that the hypothalmuses of homosexuals are more reactive to same-sex putative sex hormones. Savic himself observed that this could be an acquired trait due to repeated same-sex sexual exposure; but here is another candidate for an anatomical structure mediating between genetic factors and SSA.

We have also considered two putative prenatal factors, namely, the role of gestational hormones, and the fraternal birth order effect. At best, the research on the role of hormones is merely suggestive. Intuitively the idea that gestational hormones could affect brain development seems attractive; but while hormonal anomalies have been shown to produce sexual atypicalities in very small populations subjected to extreme levels of sex hormones, so far the research does not support hormonal anomalies as a significant factor in SSA generally . And while most research seems to show that there is such a thing as the fraternal birth order effect and that it may account for about 7% or even for as much as 29% of male homosexuality, there is no evidence of what causes it, and the largest twin study to date (Zietsch, 2012) produced negative findings in this respect.

Of course, if the fraternal birth order effect exists at all, that would be important in itself, since it would show that for some men, at least, there is a factor which they did not choose which predisposes them toward SSA. On the other hand, it must of course be remembered that even if there is a fraternal birth order effect, having an older brother would still not by itself account for SSA in anyone. There is no number of older brothers which will result in the next male child *always* being homosexual: having older male siblings would only affect the probability that the next male child will be homosexual.

To what extent do all of these results explain SSA? I think it is impossible to say with any precision. If we take Camperio's (2004) 14% maternal line effect and add it to Pattitucci's (1998) 13% male sibling effect, we are very close to Zietsch's (2012) 30% for the strength of biological factors overall. But the maternal effect and the male sibling effect might be expected to overlap. Clearly the male sibling effect and the fraternal birth order effect would overlap.

What would it mean if the explanation has more to do with epigenetics then with genetics? And what about the 70%?

Nine: Experiential Factors

Commonly heard are the claims by or on behalf of homosexuals that homosexuality is entirely or at least primarily inborn. "I was born this way" and "This is who I am" are the oft-heard refrains. The evidence of genetic or epigenetic precursors for homosexuality is sufficient to preclude the casual dismissal of such claims. In Bem's 1996 survey, 63% of homosexuals, both men and women, recalled that they had not enjoyed sex-typical activities as children. Although some questioned the reliability of such retrospective studies, they have some weight; and clearly some children do not enjoy sex-typical activities – although I was unable to find a report of an attempt to determine how many such children there are.

Nevertheless at least 37% of both male and female homosexuals did enjoy sex-typical activities. The identical twins of homosexuals are more likely to be heterosexual than homosexual (Pattitucci, 1998). The fraternal birth order effect, if it exists at all, does not account for up to 93% of male homosexuality. The male sibling effect does not account for up to 94%. The maternal line effect does not account for 86%. Of course all of these numbers are themselves subject to revision as the result of further research.

Accordingly, many researchers have emphasized that such findings reflect a role for experience in the formation of SSA. For instance, Camperio, 2004, in reporting his estimate that about 14% of the variance in sexual orientation is accounted for by the maternal effect and 6.7% by the number of older brothers, emphasized that "over 79% of the variance in male sexual orientation in our sample remains unaccounted for by the factors of an excess of maternal homosexual kin and number of older brothers. This is consistent with theoretical and empirical studies, which show that individual experiences are a powerful determinant of human sexual behaviour and self-identity. . . ." There is an extensive literature supporting this view. The most consistent finding in this regard is that sex abuse of children increases the incidence of SSA. A few studies suggest that the presence of nonbiological parents in the household may figure in SSA, and a few suggest that physical abuse may also contribute.

Childhood Sexual Abuse

Many studies have documented a connection between childhood sexual abuse and homosexuality. In a 2011 study of 13,000 New Zealand adults (age 16+), Wells found that 15% of homosexuals had experienced sexual abuse during childhood, as compared with 5% of heterosexuals. Purcell, et al. (2004) had obtained nearly identical results. In a study of 63,028 women participating in the Nurses' Health Study, Austin, et al. (2008) documented a higher prevalence of physical abuse in lesbian (30%) and bisexual (24%) young women, as compared with 19% of heterosexual women, and also of sexual abuse (19%, 20%, and 9%, respectively). Zeitsch, 2012, concluded that "childhood sexual abuse and risky family environment were significant predictors of both sexual orientation and depression, [accounting for] 8.5% and 7.7%

of the covariance between sexual orientation and depression respectively." Roberts, 2013, "estimated 9% of same-sex attraction, 21% of any lifetime same-sex sexual partnering, and 23% of homosexual or bisexual identity was due to childhood sexual abuse. . . ."

In a 30-year prospective study of physically and sexually abused children and controls, Wilson and Widon (2010), too, showed that men (but not women) with documented childhood sexual abuse (but not physical abuse) were 6.75 times more likely to later report having ever had same-sex partners. Wilson, et al., cited earlier findings that "Men who have sex with men report rates of childhood sexual abuse that are approximately three times higher than that of the general male population (Purcell, Malow, Dolezal, & Carballo-Die´guez, 2004)."

Wilson mentions possible causal mechanisms. Maybe sex abuse causes homosexuality. Maybe males who are abused begin to suspect they are gay (since most abusers are male). Or maybe the victims were already gay and that made them more vulnerable to abuse or more attractive as victims; although "One study examining this possibility found that GLB youths who described themselves as gender-nonconforming in childhood . . . were more likely to report verbal or physical, but not sexual, abuse (D'Augelli, Grossman, & Starks, 2006)," which would seem to contradict the theory that the SSA preceded the abuse.

I suspect it is at least partly the modeling of homosexual behavior to boys which suggests to their minds that it might be acceptable or even normal behavior. The introduction to their minds, by whatever means, of the possibility of same-sex activity would tend to increase the incidence of repetition on their parts. This could happen as a result of abuse; it could also happen as a result of being raised by homosexuals, a factor which will be discussed shortly; and it could also happen as a result of the modeling of homosexual behavior by society at large.

Also of interest is Roberts' 2013 study employing "instrumental variable analysis" in an effort to establish a causal link between childhood abuse and SSA. Instead of measuring abuse, the researchers measured social factors known to be associated with increased rates of abuse and which could not plausibly be thought to have been caused by the subjects' sexual orientation, namely: presence of a stepparent before age 5, poverty, parental alcohol abuse, and parental mental illness. Instrumental variable analysis has been shown elsewhere to demonstrate causation where it exists. (Angrist, 1996; Greenland, 2000.) Roberts found that "Childhood sexual abuse and non-sexual maltreatment were approximately linearly predictive of report of same-sex attraction, partners, and identity. . . . All three dimensions of sexual orientation were more strongly associated with sexual abuse than with non-sexual maltreatment. The association of sexual abuse with all three dimensions of same-sex sexual orientation was stronger among men than women. We estimated 9% of same-sex attraction, 21% of any lifetime same-sex sexual partnering, and 23% of homosexual or bisexual identity was due to childhood sexual abuse. . . ."

Bailey, 2014, criticized the Roberts study. "Both neuroticism (Zietsch, Verweij, Bailey, Wright, & Martin, 2011) and depression (Zietsch et al., 2012) have been found to correlate at the genetic level with adult sexual orientation," they said. "Therefore, parents with these genes may

be more likely to divorce (resulting in stepparent presence), live in poverty, abuse alcohol, be diagnosed with mental illness, and to have children who are maltreated." Roberts responded in 2014 by saying that ". . . Bailey and Bailey's hypothesis implies that gay men and lesbians carry genes – passed down from their parents – that increase their risk of mental illness, alcohol use, poverty, and instability in long-term relationships. To our knowledge, there is no genetic research that supports this possibility. We therefore examined the association of our instruments with same-sex sexuality among persons who did not experience childhood abuse. . . . Among persons reporting no abuse, the prevalence of same-sex attraction, partners, and identity was, in general, the same or lower in those who experienced poverty, parent alcohol problem, a stepparent or parental mental illness compared with those who did not. Although not conclusive, these data suggest that there is no effect of these non-normative experiences on sexuality except when child abuse occurs."

Homosexual Parents

Paul Cameron published a study in 2006 that claimed that the children of homosexual parents expressed a homosexual orientation much more frequently than the general population. Although claims of bias were made against the study, another study by Walter Schuum in 2010 confirmed Cameron's results by statistically examining the results of 10 other studies of a combined 262 children raised by homosexual parents. Schuum stated:

> Data from ethnographic sources and from previous studies on gay and lesbian parenting were re-examined and found to support the hypothesis that social and parental influences may influence the expression of nonheterosexual identities and/or behaviour. . . . Percentages of children of gay and lesbian parents who adopted non-heterosexual identities ranged between 16% and 57%, with odds ratios of 1.7 to 12.1, depending on the mix of child and parent genders. Daughters of lesbian mothers were most likely (33% to 57%; odds ratios from 4.5 to 12.1) to report non-heterosexual identities.

Since homosexuals makeup less than 5% of the population, this study is strong evidence that parenting can and probably does influence sexual orientation. One thing we do not know, of course, is the proportion of homosexuals who have been or are being raised by homosexuals. Whatever that number is, it is presumably rising with increasing rates of adoption, surrogacy, *in vitro* fertilization by homosexuals, and the general rise in social approval of such family arrangements.

Ten: Mental Health of Homosexuals

Homosexuals' higher incidence of depression and suicide have become a prominent part of the debate about gay rights. Gay advocates have offered such circumstances as evidence of the harmful effects of social prejudice against homosexuality. The truth would seem to be slightly more complicated than that.

Zietsch, 2012, who produced evidence of both the higher rate of neuroticism and depression in homosexuals and of the existence of common genetic factors underlying both those mental disorders and SSA (see below), cautioned that his results "should not be interpreted so as to pathologize non-heterosexuality. . . . Research aiming to understand the link between sexual orientation and psychiatric disorder should not be stymied by groups that seek to misuse the findings to support an anti-gay agenda."

I doubt Zietsch would have expressed this concern were it not for the fact that the research has already been politicized. How would the opponents of gay marriage, for instance, be motivated to obstruct scientific research if they were to suspect there is such a link? Wouldn't they want even more research? As Socrates said, we must follow the evidence wherever it leads.

That homosexuals are indeed at higher risk for mental disorders does not appear to be in question. Ferguson (2005) tested for incidence of "major depression; anxiety disorders including generalized anxiety disorder, panic disorder, agoraphobia, social phobia and specific phobia; alcohol dependence; cannabis and other illicit drug dependence," and found that homosexuals are 1.5 - 12 times more likely to suffer mental disorders, depending on where they are along the non-heterosexual spectrum, the exclusively homosexual being the most at risk.

> Males classified as predominantly homosexual had an overall rate of problems that was over five times the rate for exclusively heterosexual males (3.00 v . 0.56 problems respectively): in comparison the rate of problems amongst predominantly homosexual females was only 2.3 times that of exclusively heterosexual females (1.40 v . 0.62 problems respectively).

Langstrom (2010) concurred, stating that "Same-sex behavior remains a substantial correlate of sexually transmitted infections in men and is also associated with increased risk of physical and psychiatric morbidity among both men and women in the general population."

In 2005 Ferguson voiced the then-prevailing view that elevated rates of mental illness in homosexuals reflect social hostility:

> . . . These findings are consistent with the view that the greater stigma attached to male homosexual behaviour may make male homosexuals more vulnerable to psychiatric disorder and distress.

> There are several possible pathways by which associations between sexual orientation and mental health may arise. First, it may be suggested that this association reflects the effects of social prejudices, homophobic attitudes, victimization and harassment in increasing the vulnerability of bisexual, gay and lesbian young people to mental health problems (e.g. D'Augelli, 1996; McDaniel et al. 2001). Alternative explanations include: (1) the possibility that the association is artefactual, as a result of measurement errors or other research design problems; (2) the possibility of reverse causality in which young people prone to psychiatric disorders are more prone to experience same-sex attraction; or (3) the possibility that lifestyle choices made by young people of nonheterosexual orientation place them at greater risks of adverse life events, stresses and similar factors that may increase risks of mental health problems.

Of course there is another possible explanation. In 2011 Zietsch, et al. found that genetic factors are involved.[2] In a study of 4797 identical and fraternal Australian twins, the research team found that:

> . . . scores on both Neuroticism and Psychoticism were significantly elevated in nonheterosexuals compared with heterosexuals, indicating greater vulnerability to neurotic and psychotic disorders, respectively. Secondly, analyses with our genetically informative sample revealed significant genetic correlations between sexual orientation and both Neuroticism and Psychoticism, but corresponding environmental correlations were not significant. *This suggests that some of the genetic variation underlying sexual orientation also affects levels of Neuroticism and Psychoticism."* [Emphasis added.]

In his 2012 followup study involving 9884 twins, Zietsch was able to quantify the genetic factors:

> Non-heterosexual males and females had higher rates of lifetime depression than their heterosexual counterparts. Genetic factors accounted for 31% and 44% of variation in sexual orientation and depression respectively. Bivariate analysis revealed that genetic factors accounted for a majority (60%) of the correlation between sexual orientation and depression.

Also of interest is Zietsch's finding that:

> . . . childhood experiences of sexual abuse and risky family environment accounted for 8.5% and 7.7% of the covariance between sexual orientation and depression respectively. . . .

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[2]We must not forget that, as shown above, twin studies such as Zietsche's do not distinguish between genetics and epigenetics.

Zietsch mentioned that “Bailey (1999) suggested that elevated rates of nonrighthandness that have been observed in both nonheterosexuals (Lippa, 2003) and the mentally ill (DeLisi et al., 2002; Elias, Saucier, & Guylee, 2001; Hicks & Pellegrini, 1978) may reflect a biological factor that manifests in unusual laterality, sexual orientation, and psychiatric problems.”

Eleven: The Third Factor: Human Freedom

While the literature clearly supports the view that there are biological determinants which contribute to the incidence of homosexuality, it nevertheless remains the case that much, and probably most, of the incidence of homosexuality cannot be explained in such terms. This view is supported by studies which show that organic factors account for only part of homosexuality; by studies which show that experiential factors are also very important; and by the circumstance that even taken together, organic and experiential factors do not account for most of the incidence of homosexuality.

If one were to conclude that organic factors probably account for about 20-30% of homosexuality and experiential factors probably account for another 10-23% (and it must be emphasized that the science is really quite preliminary), that would mean that something else accounts for 47-70% of SSA.

Zietsch (2011) states that "Unknown non-genetic [and non-epigenetic] factors not shared between twin pairs, along with measurement error, account for over 50% of variance in sexual orientation. These factors could include prenatal effects, idiosyncratic experiences, unequal parental treatment, interactions with siblings, or influences outside the family (e.g., teachers and peers)." It should not go unnoticed that the only factors Zietsch mentions are heredity and environment. For some, this will immediately call to mind one of the central dogmas of scientific and philosophical naturalism, so-called because of its presupposition that nature is all that exists. In its stronger, atheistic form, naturalism posits that physical reality is all that exists. In its weaker form, naturalism becomes methodological naturalism, which is the idea that although the material universe may or may not be all that exists, science must limit itself to naturalistic explanations – that is, explanations which appeal to mechanical and impersonal causes. The effect is that inferences to personal causation and hence, moral agency, are precluded.

Thus it is that William Provine of Cornell University, a leading historian of science, was able to say:

> Modern science directly implies that the world is organized strictly in accordance with mechanistic principles. There are no purposive principles whatsoever in nature. There are no gods and no designing forces that are rationally detectable. . . .
>
> Second, modern science directly implies that there are no inherent moral or ethical laws, no absolute guiding principles for human society.
>
> Third, human beings are marvelously complex machines. The individual human becomes an ethical person by means of two primary mechanisms: heredity and environmental influences. That is all there is.

> Fourth, we must conclude that when we die, we die and that is the end of us. . . .
>
> Finally, free will as it is traditionally conceived – the freedom to make uncoerced and unpredictable choices among alternative possible courses of action – simply does not exist. . . . There is no way that the evolutionary process as currently conceived can produce a being that is truly free to make choices. [William Provine, guest editorial, *The Scientist*, September 5, 1988.]

According to this view, not only is there no God: there are no persons of any kind. That is to say that you, kind reader, do not exist. Your decisions may carry the illusion of autonomy, but in fact they are determined by the motions of the atoms in your brain, since "that is all there is."

It must, of course, be emphasized that naturalism is not science, and it is not even based on science. No one has discovered naturalism in a test tube or on a distant planet. There are no peer-reviewed studies explaining human consciousness.

The biblical view of man is that we have value and dignity by virtue of who and what we are – namely, creatures made in the image of God. But what does this mean?

The Bible teaches that God is a personal God. He is a conscious entity with thoughts, emotions, purposes, volitions. He is capable of having relationships with other persons, and of acting to preserve and promote authentic value in other persons. We, then, having been created in that image, are also authentically personal beings. We are capable of love.

If naturalistic philosophy were true, we would expect to discover that homosexual behavior – indeed, all behavior – is 100% the result of heredity and environment. We would expect a paucity of evidence that SSA is freely chosen. If biblical anthropology is true, though, we would expect a wealth of such evidence. What we have is a wealth of such evidence.

Change.

Can homosexuals become heterosexuals? Can they change their sexual behavior? Can some of them change their behavior, while others cannot? These questions are charged with emotion and with no small amount of political fervor. But what are the facts?

In 2007 Savin-Williams and Ream reviewed the literature on this question. Their findings:

- Among the 14% of Dutch adult males who reported ever having had physical attraction to other males, about half noted that these feelings disappeared later in life (Sandfort, 1997).

- Comparing sexual attraction in a New Zealand birth cohort of 1,000 at age 21 and again at age 26, Dickson et al. (2003) found that:

  > Only 38% of exclusive same-sex attracted females stayed in this group with the rest moving into "occasional" same-sex attraction (38%) or exclusive opposite-sex attraction (25%).
  >
  > One half of female and one third of male 21-year olds with occasional same-sex attraction only had opposite-sex attraction as 26-year olds.
  >
  > The proportion of males who reported at least occasional same-sex attraction increased 50% (from 4% to 6%) and 78% among females (from 9% to 16%).
  >
  > Nearly all heterosexual males (98%) kept their opposite-sex attraction; 12% of heterosexual females experienced at least occasional same-sex attraction. Migration was in both directions – from heterosexuality to homosexuality and vice versa.

- Savin-Williams and Ream describe a retrospective study of adults by Kinnish, et al., 2005, involving 762 participants (420 men and 342 women) between the ages of 36 and 60. Of the men, 163 (39%) currently identified as heterosexual, 76 (18%) as bisexual, and 181 (43%) as gay. Of the women, 119 (35%) currently identified as heterosexual, 65 (19%) as bisexual, and 158 (46%) as lesbian:

  > Change scores were derived from participants' ratings of their sexuality for every 5-year period beginning with ages 16-20 years. Although most (97%) heterosexuals maintained their heterosexual identity, nonheterosexuals frequently changed their identity label over the life course: 39% of gay males, 65% of lesbians, 66% of male bisexuals, and 77% of female bisexuals. The dimensional assessments of fantasy, attraction, and behavior reflected similar trends. Although roughly 90% of heterosexually identified individuals had none or one point changes during their lifetime, the majority of gay (52%), lesbian (80%), and bisexual (90%) identified individuals had multiple changes on the dimensional variables. For nonheterosexuals, sexual behavior changed more often than romantic attraction and for all sexual identity categories except bisexuals, women changed more than men.

Diamond's 2003 5-year study of 80 lesbians found that over a quarter of these women relinquished their lesbian/bisexual identities during this period: half reclaimed heterosexual identities and half gave up all identity labels. In her 2000 survey of young minority women (16-23 years of age), half of the participants changed their sexual identities more than once during the two-year survey period. (Diamond, 2000.)

Jones (2011) conducted a longitudinal study of 72 men and 26 women over a 6-7 year period for a group of individuals seeking sexual orientation change via a diverse cluster of religious ministries under the Christian umbrella organization, Exodus. There were dramatic changes in both directions – both toward and away from a homosexual orientation. 23% attained heterosexual reorientation and another 30% attained chastity without reorientation. Another 16% continued 6 and 7 years later to pursue change.

Was the effort harmful to those whose homosexual orientation was confirmed?

> The attempt to change sexual orientation did not appear to be harmful on average for these participants. The only statistically significant trends that emerged for the GSI (global) and PSDI (distress intensity) variables indicated improving psychological symptoms T1 to T6 [that is, from the beginning of the study to the end]. Despite these findings, we cannot conclude that particular individuals in this study were not harmed by their attempt to change. Specific individuals may claim to have experienced harm from the attempt to change, and those claims may be legitimate, but although it may be that the attempt to change orientation caused harm by its very nature, it may also be that the harm was caused by particular intervention methods that were inept, harsh, punitive, or otherwise ill-conceived, and not from the attempt to change itself. Our findings mitigate against any absolute claim that attempted change is likely to be harmful in and of itself.

The research described above does not show that every homosexual can change his or her sexual behavior, or especially his or her SSA; but it shows that some can and do change their SSA, whether spontaneously or through counseling, and many more become much more heterosexual in their choice of sex partners. But I have seen no scholarly research providing any evidence that homosexuals, as a group, cannot change.

Of course Exodus International was recently dissolved, with an apology from its chief executive Alan Chambers. In a statement, Chambers apologized for the organization, which, he said, "bore responsibility for some of the anger and hurt" experienced by homosexuals. He said nothing about what his organization had done to cause the anger and hurt, however, and elsewhere an Exodus board member specifically declined to disavow the methods which Exodus had employed. (CNN, July 8, 2013, http://www.cnn.com/2013/06/20/us/exodus-international-shutdown/.)

In his statement, Chambers stated that he and his wife feel they were "marginalized" and unsupported by the church, and his words suggest they had become exhausted by the conflict over homosexuality:

> For years we have been marginalized by society for our belief that God's creative intent for sexual expression is one man married to one woman for one lifetime. Now in our efforts to simply and wholly love and serve people, we have been marginalized by the mainstream Christian Church who once hailed our story as miraculous. We are now an embarrassment to many and I'm not always exactly sure why. Ironically, it is now the LGBT community who respect our complex story and are increasingly accepting us for who we are: unlikely friends. [http://alanchambers.org/unlikely-love/#more-692]

And he stated that from now on, the Gospel is enough, and "I will promote nothing else."

Thus we have seen that there is substantial evidence that homosexuals who want to change, sometimes do so – usually on their own, but sometimes with the help of others. But is this information readily available to the public? Here is what the American Psychological Association has to say about change therapy:

> All major national mental health organizations have officially expressed concerns about therapies promoted to modify sexual orientation. To date, there has been no scientifically adequate research to show that therapy aimed at changing sexual orientation (sometimes called reparative or conversion therapy) is safe or effective. Furthermore, it seems likely that the promotion of change therapies reinforces stereotypes and contributes to a negative climate for lesbian, gay, and bisexual persons. This appears to be especially likely for lesbian, gay, and bisexual individuals who grow up in more conservative religious settings.
>
> Helpful responses of a therapist treating an individual who is troubled about her or his samesex attractions include helping that person actively cope with social prejudices against homosexuality, successfully resolve issues associated with and resulting from internal conflicts, and actively lead a happy and satisfying life. Mental health professional organizations call on their members to respect a person's (client's) right to self determination; be sensitive to the client's race, culture, ethnicity, age, gender, gender identity, sexual orientation, religion, socioeconomic status, language, and disability status when working with that client; and eliminate biases based on these factors. [https://www.apa.org/topics/lgbt/orientation.pdf.]

In the recent decision of the US Court of Appeals for the Third Circuit in King v. Governor of New Jersey (3rd Cir., 2014)(http://www2.ca3.uscourts.gov/opinarch/134429p.pdf), upholding a legislative ban against sexual orientation change efforts ("SOCE counseling)," the Court explained its decision in the following way:

> To be sure, the APA Report suggests that the bulk of empirical evidence regarding the efficacy or harmfulness of SOCE counseling currently falls short of the demanding standards imposed by the scientific community. See J.A. 327 (noting the "limited amount of methodologically sound research" on SOCE counseling); id. at 367 (noting that "[t]he few early research investigations that were conducted with scientific rigor raise concerns about the safety of SOCE" but refusing "to make a definitive [page 54] statement about whether recent SOCE is safe or harmful and for whom" due to a lack of "scientifically rigorous studies" of these practices).
>
> Yet a state legislature is not constitutionally required to wait for conclusive scientific evidence before acting to protect its citizens from serious threats of harm. . . .

There are excellent reasons to believe that biblical theism is true, with the Big Bang, the Fine-Tuning of the Universe, and the information content of biological systems as prime examples. But for those who deny the importance of these things, the question I would ask them is, What is the alternative to biblical theism? Is it the ontological oblivion to which naturalism would consign us? Or is it the denial of the reality of all love, truth, beauty, justice, and wisdom? From where does the obligation to treat others with equity come? Historically speaking, it has come from the Bible. Where do we stand to demand equity if we are merely highly-organized collections of cheap chemicals? I tell you, you are entitled to equity only because you are a child of God.

Therefore we are highly amenable to an interpretation of the scientific evidence which leaves room for human freedom. Indeed, the Two Books model, to which we appealed at the outset, compels us to embrace that interpretation unless and until it becomes completely untenable, and we are nowhere near that point yet.

The findings concerning the influence of homosexual parents upon the sexual preferences of their children is very pertinent in this respect. The resulting increase in the incidence of homosexuality far surpasses the documented influence of all of the genetic and/or epigenetic factors discussed. It has been my observation in my own life and in the lives of those close to me, that to us as children, our parents, whether intentionally or not, introduce us to the world, and what we see in our parents then constitutes for us what is true, what is right, and what is possible, until as young adults we learn otherwise. In short, homosexual parents model for their young the legitimacy, the open possibility, of choosing a mate from among their own sex. For the children of heterosexuals, it is not a choice, not because we are specifically taught, but because it never occurred to us to ask. But for the children of homosexuals, it is an active possibility. When the child chooses between that and other possibilities, is that not a free choice?

Zietsch, 2011, states that “Unknown non-genetic factors . . . account for over 50% of variance in sexual orientation. These factors could include . . . idiosyncratic experiences, unequal parental treatment, interactions with siblings, or influences outside the family (e.g., teachers and peers).” If so, would such factors, all taken together, attenuate personal

responsibility in the least? Are we not responsible for our own decisions? If we are not responsible for our own decisions, are we personal beings at all?

Twelve: Conclusions

Let us bring together the most salient aspects of the foregoing discussion. What have we learned from the evidence that makes a practical difference in our assessment of SSA?

Three things stand out. The first is that there are probably one or more genetic or epigenetic precursors which may account for 20-30% of SSA.

The second outstanding finding is that about 70-80% of SSA is probably not attributable to biological factors. About 10% may be attributable to childhood sex abuse. The remaining 60-70% is something else. Some part, and perhaps all or nearly all, of that 60-70% is the free choice of the individual. (Parental modeling substantially increases the incidence of SSA in children raised by homosexuals, but I have no information as to the number of such households or hence, the amount of the resulting increase in the incidence of SSA.)

Finally, the results of numerous studies showing that many homosexuals do change their behavior and even their same-sex attraction, are also very important.

Also of interest is the evidence concerning the particular nature of the genetic factors contributing to SSA. First – assuming a role for genetics as distinguished from epigenetics – at least some of the genetic factors are sex-linked – that is, they appear to be X-chromosome genes which are passed from mothers to their children. This does not automatically mean that SSA is like undesirable sex-linked traits such as hemophilia. Many sex-linked traits are beneficial, indeed crucial, to the normal functioning of the individual. But there are many that are not, and the analogy to sex-linked disorders is therefore plausible. Without contrary evidence, this precludes any assumption that SSA is normal simply because it occurs.

And second, there is evidence that some of the genetic and/or epigenetic factors contributing to SSA are the same factors which also contribute to mental disease in homosexuals. It could even be argued that this circumstance strengthens the analogy to undesirable sex-linked traits. Finally, the possibility that the role for epigenetics results from incomplete erasure of the epigenome in the germ cell stage also suggests the possibility that SSA is in the nature of a disorder.

Arguably, the inconclusive nature of other findings is of secondary importance. Whether hormones are involved, whether brain hemispheric symmetry is implicated – these have to do not with the nature or cause of SSA but merely with the pathway for its expression. The population studies indicate that a pathway exists, and our ignorance about what that pathway is probably doesn't much affect our assessment of SSA itself.

Thirteen: The Church's Response

What shall we make of the apparent fact that there are genetic/epigenetic and experiential precursors to homosexuality? I believe it would be possible to argue that the information summarized above supports the following conclusions:

1. It seems likely that SSA is to some extent rooted in circumstances outside the control or choosing of those affected by it. Both genetic or epigenetic factors and experience probably play a role.

2. That does not by itself provide any support for the view that SSA is normal or good, any more than other conditions which have been shown to have a genetic component, such as sickle-cell anemia, hemophilia, or alcoholism, are normal or good. Indeed, there does not appear to be any scientific basis for affirming that homosexuality is normal or good. Rather, it is more likely that the genetic and/or epigenetic precursors are anomalous, and that the experiential factors involved are in the nature of psychological injuries.

3. The roles which genetics/epigenetics and experience play in SSA are limited, leaving ample room for human freedom and responsibility.

4. The foregoing conclusions do not compel the abandonment of biblical teaching about the moral character of same-sex sexuality. Indeed, they support that teaching over against the unsupported claims that SSA is normal and good but that no one chooses it.

5. Nothing about SSA as such impairs a person's value or dignity as a human being, created in the image of a personal God.

6. The church offers the crucified and risen Christ as the solution for all human woe. Her Gospel is true. Gays, lesbians, bisexuals, transsexuals, just like everyone else, must be granted the privilege of hearing their declaration of freedom, and the blessedness of knowing the Son of God and the fellowship of his people.

There is no dichotomy between science and religion. Religions are true or false and can be tested empirically. There is abundant scientific evidence for biblical cosmology (the Big Bang, the Fine-Tuning of the Universe, the information content of DNA, etc.), and there is compelling historical evidence for the Resurrection of Jesus[3]; hence it is no surprise that the Book of Nature and the Book of Scripture affirm the same view of human sexuality.

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[3]See, for instance, Michael R. Licona. The Resurrection of Jesus: A New Historiographical Approach (Nottingham, England: IVP Academic 2010).

While the apparent fact that there are genetic or epigenetic and environmental precursors to SSA does not falsify the biblical teachings about homosexuality, it must encourage the church to be gentle and patient with those who wish`to change, and humble in the face of our common human frailty. At the same time, the fact, also apparent, that biological factors cannot account for the majority of SSA, will help us to counter the claim that the condition is unchangeable, and to inspire hope in those who wish to change.

The entire elite of American society – the gay community, the media, institutions of higher learning, the scholarly community, and the government – may desperately wish the church to drop her teaching about homosexuality, but for her to do so would be a betrayal of that very society, whom the church has been called to serve, specifically by announcing the wonderful news of God's forgiveness in Jesus Christ. As R. Albert Mohler, president of the Southern Baptist Theological Seminary, has very recently written in regard to this question, if the biblical authors got it wrong about human sexuality and what it means to be human, then what else did they get wrong about the human condition? If the clear teachings of scripture concerning human sexuality can be dismissed when we find them inconvenient, how can we regard any other biblical text as authoritative? If the Bible is not authoritative, how can we know who God is or that we can be reconciled to him?

> . . . [W]ithout any authoritative revelation of what sin is . . . we cannot know why we need a savior, or why Jesus Christ died. . . . Could the stakes be any higher than that? This controversy is not merely about sex, it is about salvation. [Mohler, 2014.[4]]

If the biblical teaching about the human condition is true (and it is), then it is loving and compassionate to teach accordingly, and cruel to teach the contrary. If anyone asks whether the church seriously suggests that homosexuals should become celibate, or chaste, I believe there can be no better response than what Jesus said:

> For whosoever will save his life shall lose it: and whosoever will lose his life for my sake shall find it.
>
> For what is a man profited, if he shall gain the whole world, and lose his own soul? or what shall a man give in exchange for his soul? (Matthew 16:25-27.)
>
> Then answered Peter and said unto him, Behold, we have forsaken all, and followed thee; what shall we have therefore?
>
> And Jesus said unto them, Verily I say unto you, That ye which have followed me, in the regeneration when the Son of man shall sit in the throne of his glory, ye also shall sit upon twelve thrones, judging the twelve tribes of Israel.

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[4]For a defense of the traditional interpretation of the biblical texts concerning homosexuality, this is an excellent book.

> And every one that hath forsaken houses, or brethren, or sisters, or father, or mother, or wife, or children, or lands, for my name's sake, shall receive an hundredfold, and shall inherit everlasting life. (Matthew 19:27-29.)

And Paul:

> I count all things but loss for the excellency of the knowledge of Christ Jesus my Lord: for whom I have suffered the loss of all things, and do count them but dung, that I may win Christ,
>
> And be found in him, not having mine own righteousness, which is of the law, but that which is through the faith of Christ, the righteousness which is of God by faith:
>
> That I may know him, and the power of his resurrection, and the fellowship of his sufferings, being made conformable unto his death;
>
> If by any means I might attain unto the resurrection of the dead. (Philippians 3:8-11.)

We are talking about God, the Son of God, the Creator of the universe, who loved us, gave himself for us, calls us into fellowship, and who by his Spirit gives us power to live free. We are to be with him forever, in a community of the just and the true. This is not an invention: it is what the living God has actually done, for which there are many excellent proofs. To my gay and lesbian friends, I say that we are all alike in this respect: that we are all called to forsake everything for him. And when we understand who he is, doing so is our greatest joy.

Bibliography

Allgulander C., et al. (1991), Psychopathology and treatment of 30, 344 twins in Sweden II Heritability estimates of psychiatric diagnosis and treatment in 12884 twin pairs. Acta Psychiatrica Scandinavica 83(1):12-15.

Angrist, et al. (1996), Identification of causal effects using instrumental variables. *Journal of the American Statistical Association*, 91, 444–455.

Austin, et al. (2008), Disparities in child abuse victimization in lesbian, bisexual, and heterosexual women in the Nurses' Health Study II. *J Womens Health* (Larchmt) 17(4):597-606.

Bailey, J. M., & Zucker, K. J. (1995), Childhood sex-typed behavior and sexual orientation: A conceptual analysis and quantitative review. *Developmental Psychology*, 31, 43-55.

Bailey, J.M., et al. (2000), Genetic and Environmental Influences on Sexual Orientation and Its Correlates in an Australian Twin Sample. *Journal of Personality and Social Psychology* Vol. 78, No. 3, 524-536.

Bailey, D. H., et al. (2014), Genetic Confounds in the Study of Sexual Orientation: Comment on Roberts, Glymour, and Koenen. *Archives of Sexual Behavior* DOI 10.1007/s10508-014-0269-3.

Bem, D. J. (1996), Exotic becomes erotic: A developmental theory of sexual orientation. *Psychological Review*, 103, 320-335.

Berglund, H., et al. (2006), Brain response to putative pheromones in lesbian women. *Proc Natl Acad Sci USA* 103:8269 - 8274.

Bishop, K. M., et al. (1997), Sex Differences in the Human Corpus Callosum: Myth or Reality? *Neuroscience & Biobehavioral Reviews* 21: 581-601.

Blanchard, R. (2004), Quantitative and theoretical analyses of the relation between older brothers and homosexuality in men. *Journal of Theoretical Biology* 230, 173–187.

Blanchard, R., et al. (2008), The Sex Ratio of Older Siblings in Non-Right-Handed Homosexual Men. *Arch Sex Behav* 37:970–976.

Blanchard, R. (2008). Sex ratio of older siblings in heterosexual and homosexual, right-handed and non-right-handed men, *Archives of Sexual Behavior*, 37, 977–981.

Bouchard, T.J., et al. (1990), Sources of human psychological differences: the Minnesota study of twins reared apart. Science 250:223-8.

Breedlove, M.S. (1997), Sex on the brain. *Nature* 389: 801.

Burr, C. (1993), "Homosexuality and Biology." *The Atlantic Monthly*, March 1993, pp. 47-65.

Byne W., et al. (2001), The interstitial nuclei of the human anterior hypothalamus: an investigation of variation with sex, sexual orientation, and HIV status. *Horm. Behav*. 40:86-92.

Cameron, P. (2006), Children of homosexuals and transsexuals more apt to be homosexual. *J. Biosoc. Sci*. 38:413-418.

Camperio-Ciani, et al. (2004), Evidence for maternally inherited factors favouring male homosexuality and promoting female fecundity, *Proceedings of the Royal Society of London B*, 271, 2217–2221.

Camperio-Ciani, A., et al. (2012), Fecundity of paternal and maternal non-parental female relatives of homosexual and heterosexual men, *PLoS One*, 7, e51088.

Churchill, W. (1967), Homosexual behavior among males: a cross-cultural and cross-species investigation. (Englewood Cliffs, NJ: Prentice-Hall.)

Corliss, H. L., et al. (2009), Sexual risk in "mostly heterosexual" young women: Influence of social support and caregiver mental health. *Journal of Women's Health*, 18, 2005– 2010.

Diamond, L. M. (2000), Sexual identity, attractions, and behavior among young sexual-minority women over a 2-year period. *Dev. Psychol*. 36: 241-50.

Diamond, L. M. (2003), Was it a phase? Young women's relinquishment of lesbian/bisexual identities over a 5-year period. *J. Pers. Soc. Psychol*. 84: 352-64.

Dickson, N., et al. (2003), Same-sex attraction in a birth cohort: Prevalence and persistence in early adulthood. *Social Science & Medicine*, 56, 1607?1615.

E. M. Drabant, et al. (2012), 23andMe Genome Wide Association Study of Sexual Orientation in a Large, Web-based Cohort. Abstract presented at the American Society of Human Genetics annual meeting in San Francisco.

Ellis, L., et al. (1987), Neurohormonal functioning and sexual orientation: a theory of homosexuality–heterosexuality.
Psychological Bulletin, 101, 233–258.

David M. Fergusson, et al. (2005), Sexual orientation and mental health in a birth cohort of young adults, *Psychological Medicine*, 35, 971–981.

Gilbert, Kathleen, Former Lesbian: I Craved Emotional Balance of Hetero Relationship by Kathleen Gilbert (page no longer available).

Glatze, M. (2011), How a 'gay rights' leader became straight. WorldNetDaily (http://www.wnd.com/2007/07/42385/).

Green R. (1987), The "Sissy Boy Syndrome" and the Development of Homosexuality. Yale University Press; New Haven: 1987.

Greenland, S. (2000), An Introduction to Instrumental Variables for Epidemiologists, *International Journal of Epidemiology*, 29:722-729.

Hines, M. (2011), Prenatal endocrine influences on sexual orientation and on sexually differentiated childhood behavior, *Front Neuroendocrinol*. 32(2): 170-182. (http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3296090/)

Horgan, J. (1993), Eugenics revisited. Scientific American 268 (June):92-100.

Jones, S. L., et al. (2011), A longitudinal study of attempted religiously-mediated sexual orientation change. *Journal of Sex and Marital Therapy* 37: 404-427.

Kaminsky, et al., DNA methylation profiles in monozygotic and dizygotic twins. Nature Genetics 41(2), 240-245, February 2009.

Kinnish, K. K., Strassberg, D. S., & Turner, C. W. (2005). Sex differences in the flexibility of sexual orientation: A multidimensional retrospective assessment. Archives of Sexual Behavior, 34, 173 - 183.

Kirk, K., et al. (2000), Etiology of male sexual orientation in an Australian twin sample, *Psychology, Evolution & Gender* 2: 301?311.

Kirk, K., et al. (2000), Measurement models for sexual orientation in a community twin sample. *Behavior. Genetics* 30: 345?356.

Langstrom, N., et al. (2010), Genetic and Environmental Effects on Same-sex Sexual Behavior: A Population Study of Twins in Sweden, *Archives of Sexual Behavior,* 39:75-80.

Lasco, M. S., et al. (2002), A lack of dimorphism of sex or sexual orientation in the human anterior commissure. *Brain Research* 936: 95-98.

Laurent, et al. (2012), Non-random mate choice in humans: insights from a genome scan. *Molecular Ecology* 21:587-596.

LeVay, S.(1991), A difference in hypothalamic structure between heterosexual and homosexual men. *Science*, 253, 1034–1037.

Martin, et al. (2004), The cognitive, behavioral, and personality profiles of a male monozygotic triplet set discordant for sexual orientation. *Arch. Sex. Behav*. 33:497-514.

McConaghy, N., et al. (2006), Fraternal birth order and ratio of heterosexual/homosexual feelings in women and men. *J. Homosex*. 51:161-74.

McFadden, D. (2002), Masculinization effects in the auditory system. *Arch. Sex. Behav*. 31:99-111.

McGue M, Lykken, D.T. (1992), Genetic influence on risk of divorce. Psychological Science 3(6):368-373.

Mohler, R. Albert, Jr., ed. (2014), God and the Gay Christian? A Response to Matthew Vines (Louisville: SBTS Press.

Ngun, Tuck C. and Vilain, Eric, "The Biological Basis of Human Sexual Orientation: Is There a Role for Epigenetics?" Epigenetic Shaping of Sociosexual Interactions: From Plants to Humans, https://books.google.com/books?hl=en&lr=&id=p2ZzAwAAQBAJ&oi=fnd&pg=PA167&ots=4RtvcSxIcX&sig=uWoP5pAaT73m67-0jpO4s0S6cKY#v=onepage&q&f=false, pp 167-184.

Pattatucci, A.M.L. (1998), Molecular investigations into complex behaviour: lessons from sexual orientation studies. *Human Biology*, 70, 367–386.

Rahman, Q., et al. (2003), Born gay? The psychobiology of human sexual orientation. *Personality and Individual Differences*, 34, 1337–1382.

Ramagopalan, S. V., et al. (2010), A genome-wide scan of male sexual orientation. *J. Hum. Genet*. 55: 131?132.

Regnerus, M. (2012), How different are the adult children of parents who have same-sex relationships? Findings from the New Family Structures Study, *Social Science Research*, vol. 41, Issue 4 (July 2012), pp. 752-770. (http://www.sciencedirect.com/science/article/pii/S0049089X12000610)

Rice, G., et al. (1999), Male Homosexuality: Absence of Linkage to Microsatellite Markers at Xq28. *Science* 284: 665-667.

Rind, B. (2013), Homosexual Orientation – From Nature, Not Abuse: A Critique of Roberts, Glymour, and Koenen (2013), *Archives of Sexual Behavior*, 42: 1653-1664.

Roberts, A. L., et al. (2013), Does maltreatment in childhood affect sexual orientation in adulthood? *Archives of Sexual Behavior*, 42, 161–171.

Roberts, A. L., et al. (2014), Considering Alternative Explanations for the Associations Among Childhood Adversity, Childhood Abuse, and Adult Sexual Orientation: Reply to Bailey and Bailey (2013) and Rind (2013), *Archives of Sexual Behavior*, 43:191-196.

Rushton, J.P., et al. (1984), Altruism and genetics. Acta Geneticae Medicae et Gemellologiae 33:265-271.

Saffery, R., *et al*., Cohort Profile: The Peri/post-natal Epigenetic Twins Study, *International Journal of Epidemiology*, http://ije.oxfordjournals.org/content/early/2011/10/02/ije.dyr140.full.

Sanders, A. R., *et al*. (2012), Genome-wide linkage scan of male sexual orientation, *Psychological Medicine*, 45, 1379–1388.

Sanders, et al., Genome-wide scan demonstrates significant linkage for male sexual orientation. *Psychol Med*. 2015 May; 45(7):1379-88.

Santtila, P., et al. (2008), Potential for homosexual response is prevalent and genetic. *Biological Psychology* 77: 102?105.

Savic, I., et al., (2005), Brain response to putative pheromones in homosexual men. *Proceedings of the National Academy of Sciences*, 102, 7356–7361.

Savic, I., et al. (2008), PET and MRI show differences in cerebral asymmetry and functional connectivity between homo- and heterosexual subjects. *Proceedings of the National Academy of Sciences*, 105, 9403–9408.

Savin-Williams, R. C., et al. (2007), Prevalence and Stability of Sexual Orientation Components During Adolescence and Young Adulthood. *Arch Sex Behav* 36:385-394.

Scott, C. E. (2015), "Carl's Rock Songbook No. 106, John Lennon, "Imagine No Religion." http://www.nationalreview.com/postmodern-conservative/418703/carls-rock-songbook-no-106-john-lennon-imagine-no-religion-carl-eric, 5.21.15.

Schuum, W. R. (2010), Children of homosexuals more apt to be homosexuals? A reply to Morrison and to Cameron based on an examination of multiple sources of data. *J. Biosoc. Sci*. 42:721-742.

Stevenson J., et al. (1992), Fears and fearfulness in children and adolescents: a genetic analysis of twin data. Journal of Child Psychology and Psychiatry 33(6):977-985.

Titus-Ernstoff, et al. (2003), Psychosexual characteristics of men and women exposed prenatally to diethylstilbestrol. *Epidemiology* 14(2):155-60.

Varnell, P. (1999), "More Gays than Lesbians." (IGF Culture Watch, 1999), http://igfculturewatch.com/1999/11/30/more-gays-than-lesbians/)

Verweij, K.J., et al. (2008), Genetic and environmental influences on individual differences in attitudes toward homosexuality: an Australian twin study. Behavior Genetics 38(3):257-65.

Waller, N.G., et al. (1990), Genetic and environmental influences on religious interests, attitudes and values: a study of twins reared apart and together. Psychological Science 1:138-142.
Wells, E., et al. (2011), Multiple Aspects of Sexual Orientation: Prevalence and Sociodemographic Correlates in a New Zealand National Survey. *J. Archives of Sexual Behavior*, February 2011, Volume 40, Issue 1, pp 155-168.

Walters, E.E., et al. (1992), Bulimia nervosa and major depression. A study of common genetic and environmental factors. Psychological Medicine 22:617-622.

Whoops! Lesbian 'Person of the Year' in Gay Press Goes Straight With Baby by Tim Graham (http://newsbusters.org/blogs/tim-graham/2009/07/19/whoops-lesbian-person-year-gay-press-goes-straight-baby).

Williams, T. J., et al. (2000), Finger-length ratios and sexual orientation. *Nature* 404: 455-456.

Wilson, H. W., et al. (2010), Does Physical Abuse, Sexual Abuse, or Neglect in Childhood Increase the Likelihood of Same-sex Sexual Relationships and Cohabitation? A Prospective 30-year Follow-up, *Archives of Sexual Behavior*, 39, 63-74.

Zietsch, B. P., et al. (2011), Sexual orientation and psychiatric vulnerability: A twin study of neuroticism and psychoticism. *Archives of Sexual Behavior*, 40, 133–142.

Zietsch, B.P., et al. (2012), Do shared etiological factors contribute to the relationship between sexual orientation and depression? *Psychological Medicine*, 42, 521–532.

Zuger, B. (1984), Early Effeminate Behavior in Boys:
Outcome and Significance for Homosexuality. *The Journal of Nervous and Mental Disease*, 172(2): 90-97.