Book Review of Simon LeVay's

Gay, Straight and the Reason Why

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Summary: This book reports on a very good survey of the possible biological/prenatal/neurohormonal origins of SSA (same-sex attraction) and a small but inadequate survey on OSA (opposite-sex attraction). While it contains a large number of new insights and useful interpretations, some of the points need significant modification in view of recent literature. The book presents a rather confused picture of whether social/psychological factors have any importance, and its discussion of them is far too superficial. It opts for most SSA being caused by prenatal hormones, a conclusion not consistent with the much replicated results of twin studies, which show all prenatal factors are only a minor influence. That twin studies conclusion is a maximum estimate not likely to change. Prenatal origin for sexual orientation is now not the majority view of researchers, who assign a prime role to postnatal factors. Contrary to the position suggested in the book, the brain is not strongly gendered at birth. One section suggests that limited change in sexual orientation is possible. The book discusses the increasing doubts about the "elder brother hypothesis" (i.e., the more elder brothers the greater the possibility of SSA), but overvalues the biological explanations. Overall the book is complex and the confusing results from many experimental programs trying to explain SSA by biological factors suggest there are too many layers of hypothesis.

Book Review of Simon LeVay's *Gay, Straight and the Reason Why*

Gay, Straight and the Reason Why by Simon LeVay (O.U.P. 2011) is about sexual orientation, both gay and straight. It asserts the origin of both is the same: prenatal and hormonal. While both sexual orientations are discussed, the book concentrates on same-sex attraction (homosexuality); the explicit discussion of opposite-sex attraction (heterosexuality) is quite thin.

Author and neuroanatomist Simon LeVay is best known for his 1991 claim that the INAH3 nucleus size in the hypothalamus of brains of homosexual men is significantly more like that of women (LeVay, 1991). He has also written several books dealing with the science about SSA.

Published by Oxford University Press, the book appears to be scientifically careful. It cannot discuss papers dated after its 2010 publication, however, so please bear that in mind for the other literature cited in this review.

Gay, Straight and the Reason Why demonstrates clearly what a prereviewer on the back cover calls LeVay's "mastery of the literature." It provides an excellent discussion of the biological literature. While I do not agree with many of the biological arguments because of results described in later papers, I have never come across a text with so many stimulating new ideas—so despite my disagreement, I felt the book was well worth the purchase price. Contrary to a back cover reviewer's comment about its "balance," however, I found it surprisingly unbalanced; it is weak on the social side and misses many explanations that would occur instantly not only to clinicians but to general social scientists.

There are 295 pages of text, but a glossary, chapter references, and bibliography extend the book to 412 pages; like many academic books, the actual text ends well before expected. Of the actual text, only 16 pages are a superficial survey of social factors. Since LeVay concentrates on the science, so will I.

The Prenatal Neurohormonal Theory

LeVay's basic goal seems to be to present evidence for the neurohormonal, prenatal theory of homosexuality (and, of course, heterosexuality).

I should immediately say that because the theme of the book is sexual attraction, the overall framework of sex and reproduction is assumed but neglected. Yet that framework is ultimately needed. Most novels present life as mainly about sex, with little about the bringing up of children. Real life is the opposite!

The neurohormonal theory dates back at least fifty years (Phoenix, Goy, Gerall, & Young, 1959), but LeVay's book can be regarded as a good update. LeVay strongly endorses:

- "the idea that the origins of sexual orientation are to be sought in the interaction between sex hormones and the developing brain" (p. xi).
- "a chain of causal events that leads from genes to sexual differentiated mental or behavioural traits" (p. 50).
- "we should not expect to identify a specific cause behind every individual's sexual orientation beyond a general attribution to the prenatal hormonal mechanisms that I've been discussing" (p. 283).
- "I'm inclined to place most of the developmental control in the hands of prenatal hormones" (p. 279).

(Incidentally, how tentative that last comment is!)

The basic theory has been that the brain is sexually organized prenatally and intensely—mainly by hormones, particularly testosterone. The idea is that the created neuronal structure is similar to computer hardware, and that nothing can change its organization until puberty. The theory states that a child is born with a highly gendered brain but that the preferred gender is mainly invisible. The theory further states that sexual orientation is not

fully developed until puberty, when the sexual orientation of the brain is activated by high hormone levels, somewhat like throwing a switch. Proponents of the theory maintain that homosexuality is a prenatal disturbance in this process of sexual orientation development.

In the past, proponents have not been clear about the status of newborns because they should show no gender differentiation at all; it should be latent until activated at puberty. However, claimed gender differences at birth have been published quite often and have tended to be taken as some evidence that strongly gendered brain structure exists from earliest days. Some postnatal influence is seldom completely rejected in the book, which throws up the possibility that some sexual orientation is learned. LeVay reflects this ambivalence. On the one hand, he assigns a majority of influence to prenatal hormones. On the other, he allows for other (presumably minor) influences, unlike many other authors.

"The Reason Why" part of the title really refers to a single general unified theory of biological origins for sexual orientation, one that he thinks predominates over all others. However:

- "Is this preference 'organized' by sex hormones during development? The answer is yes—to a degree" (p. 56). (This quote is rather remarkable for its tentative air.)
- A diagram (p. 64) is labelled as a "prenatal hormonal theory," but it includes the possibility of (presumably minor) idiosyncratic influences and environmental influences that affect sexual orientation.

The belief that the prenatal hormonal theory is predominant implies at least a semiquantitative assessment of the strength of its influence, but no such assessment is found in the book. Perhaps LeVay might ultimately be reluctantly open to the idea that various psychological and social influences combined could be even stronger than genetic influences.

Innately Gendered Brain?

LeVay's original 1991 statement that homosexual male brains were more feminine than usual was not confirmed in a subsequent study by Byne (Byne et al., 2001); knowing that readers may be familiar with that issue, LeVay devotes a small section to it. He comments that although Byne did not find statistical significance to confirm that notion, he did find a trend, so Byne's study is not really a refutation. LeVay points out that parallel findings were found in sheep; however, such studies are notoriously irreproducible, as illustrated in the work he describes by Swaab and others on a hypothalamus region called INAH1, very near the INAH3 region he himself studied. A claimed sex dimorphism in size could not be confirmed by three laboratories, but it seems Swaab still believes it is sexually dimorphic. Because of known plasticity of the brain, I believe it is almost certain that any reproducible results in altered brain structure or neural networks will turn out to be the result of sexual/mental activity connected with sexual behaviour.

Levay is aware of and endorses the fifteen-year-old argument that "brain organisation cannot be genetically specified in precise detail" (p. 60) because 22,000 genes cannot specify 100 billion neural connections—there are far, far too many. LeVay does not explore the concept that most brain connections are formed in reaction to the environmental experiences undergone by the young child, so social influences should predominate.

LeVay believes that the adult brain is highly gendered. I agree that there are numerous male–female differences for adult subjects, but the perennial question is whether these are inborn or developed under the strong influence of maternal/paternal interaction, sexual experiences, and other factors. The idea that the new brain is highly gendered has almost no support; it certainly is not strongly anatomically dimorphic, but LeVay cites one of the rare established differences: Girls pay more attention to faces, and boys pay more attention to things. Even in this statistical difference there is a lot of overlap between the genders. The newborn brain is far less sexually dimorphic than the genitals by any criterion.

A salutary tale about gender differences is the historic idea that female newborns are much more sensitive to touch than are newborn males. Some authors cited studies from the last fifty years that showed complete sexual dimorphism, with results from the two genders not even overlapping. The most careful study—because it was blind—showed there was no difference at all (Jacklin, Snow, & Maccoby, 1981), and touch is now rarely cited as an example of gender differences. But this shows the strong human urge to demonstrate sexual dimorphism and the degree of self-deception that can result.

The following paragraphs show that sexual dimorphism in the brain, even at the biochemical level, is far less than commonly believed.

Recent Brain Research

LeVay and other authors state that most influences on sexual development are only prenatal and only hormonally driven. It's important to understand that such ideas are not currently accepted: "Our current knowledge of sex-based neurobiology has outgrown this simplistic model. Multiple lines of research have contributed to this conclusion" (Reinius, 2011, p. 15). LeVay either does not mention this literature or could not comment because it was more recent than his manuscript. But an extraordinary amount of work has been published, much drawing on genome studies of various animal tissues.

The perspective that is emerging is extremely interesting but its detail is beyond the scope of this book review; a review paper is in preparation. Following is a very condensed summary of experimental work done on various animals (mice and rats, unless otherwise noted):

1. Perhaps 50% of fetal brain biochemical sexual dimorphism is independent of sex hormones (Dewing, Shi, Horvath, & Vilain, 2003; Lee et al., 2009 [chickens]; Sreenivasan et al., 2008 [zebrafish]; Reinius, 2001 [humans]). The authors describe the idea of obligatory association of sexual dimorphism with prenatal hormones as a "dogma."

2. Sexual dimorphism in the brain and subsequent sexual behavior just after birth is sensitive to prenatal stress (Mychasiuk, Gibb, & Kolb, 2012)—called here an "epigenetic" (environmental) effect.

3. Extraordinarily, the brain, hypothalamus, and gonads are far less sexualized tissue when compared with liver and adipose tissue (Yang et al., 2006; Gregg, Zhang, Butler, Haig, & Dulac, 2010; Hadziselimovic, Hadziselimovic, Demougin, & Oakeley, 2011 [humans]; van Nas et al., 2009).

4. Most development of the brain is postnatal. There are growth and pruning cycles that are strongly influenced by the environment, including hormonal cycles that cause sexually dimorphic pruning (Kauffman, 2009; Martin et al., 2010; Hisasue, Seney, Immerman, & Forger, 2010; Semaan & Kauffman, 2010; Hines, 2011 [humans]). Some of the authors say that the *majority* (my emphasis) of known sex differences are induced by the sex steroid milieu during early postnatal development (as contrasted with prenatal influence only). This applies to laboratory animals, with social conditions held very constant and, I assert, presumably to humans.

5. The human brain changes biochemically in a sexually dimorphic way at the moment of birth and again at age twenty (in other words, at adulthood) but surprisingly not much at puberty (Colantuoni et al., 2011; Kang et al., 2011). One interpretation of this may be that we have overestimated the changes associated with sexual maturation.

6. In my view, recent observation of some transgender individuals who rapidly alternate their perceived gender (Case & Ramachandran, 2012) makes it unlikely that this perception is innate or tied strongly to neuronal pathways .

While the literature is complicated and will demand several changes in our concepts, it is shifting more and more in the direction that prenatal influences are less important than postnatal influences. This means that the view LeVay prefers—that sexual orientation is rather rigidly fixed before birth—is becoming less and less mainstream. He does not seem closed to fresh evidence, however, and some of the statements in his book seem couched to cover some of the recent possibilities.

Possible Social/Psychological Theories

LeVay spends a chapter explaining why social influences are inadequate and a biological theory ought to be investigated instead. In that chapter he mentions only three influences: psychoanalytical ideas (such as those of Freud), learning (the first sex act is very important), and gender learning.

Contemporaries Socarides and Nicolosi are quoted in this chapter, but their views are examined rather superficially. Nicolosi's recent work is not mentioned, nor is there much exploration of other views, including the considerable literature on effects of social factors on gender development. While he admits that the factor exists, LeVay attacks the idea of the father being important in the development of SSA among men. Again he seems to imagine that prenatal factors are strong and other factors are weak. I believe that *all* the factors are weak, but some postnatal factors are very important to some individuals.

One of the postnatal factors that can be important for some is sexual abuse. LeVay doesn't think that sexual abuse has much of a link to adult SSA, and there are indeed a few studies that have failed to show a link. Unfortunately, LeVay's citations are quite inadequate in this area, which could be due mainly to the time frame of publishing the book. Important studies showing such links include Arreola, Neilands, and Diaz (2009); Austin et al. (2008); Cutajar et al. (2010); Rosario, Schrimshaw, and Hunter (2009); Rothman, Exner, and Baughman (2011); Tomeo, Templer, Anderson, and Kotler (2001);

Wilson and Widom (2010); and Zietsch et al. (2012). I doubt any sex researcher today would seriously query the link, although its strength is open to debate.

In support of LeVay's idea that sexual orientation is not learned, he quotes the famous case of David Reimer, who was born XY but who was brought up as a girl because a medical error resulted in him losing his penis when he was very young. Contrary to upbringing, he ultimately settled for a male identity. Unlike most authors, LeVay goes on to cite the less-known case where a boy who had lost his penis was brought up as a girl and developed a stable feminine gender identity but is bisexual and currently in a relationship with a woman. From these two cases—along with cloacal exstrophy, or intersex, cases—that "nature" usually trumps "nurture" (p. 40). This is a poor summary of the literature.

In one far larger study in which penises were lost very early, 69% elected to remain female; in another study, 75% elected female identity (Meyer-Bahlburg, 2005). But telling patients they were fundamentally XY did make a difference, and some preferred to shift to male—a culturally favored gender. If anything, the bulk evidence here is that nurture trumps nature. Though the Reimer case received massive media attention, it is simply not typical.

To support the idea that learning has a negligible effect on sexual orientation, LeVay writes: "Children raised by gay parents don't differ in sexual orientation . . . from those raised by straight parents" (p. 40). But I find the critiques of that argument convincing (Schumm, 2008; Schumm, 2010; Schumm, 2011). Those children *do* differ, and there is some effect on their sexual orientation. Parental influence is not overwhelming but neither are prenatal influences.

Twin Studies

LeVay considers twin studies as evidence that there is at least some genetic influence on SSA, but even the sources he cites show that prenatal influences are not predominant.

The results of the eight major twin studies (many unreferenced in the book) varied. Even those that used twin registers or good, large random samples and that are the

least biased so far had different results. Some showed no genetic or prenatal influence on SSA, while some showed significant genetic/prenatal influence—but a mean of the first seven studies (Whitehead, 2011) gave a weak to modest 23% for males and 37% for females. (These should not be confused with the pairwise concordances mentioned below.) LeVay quotes a 30 to 50% figure relying on fewer studies, but genetic influence is still not predominant in that estimate—and for reasons given elsewhere in extensive detail (Whitehead, 2011), all the twin studies' genetic estimates are almost certainly much too high for adults and are zero for adolescents. LeVay fails to include or discuss any of the numerous papers in the literature that suggest this. A weak to modest influence is much more likely, and a majority influence of prenatal factors is probably impossible.

These studies sum up all the influences known and yet to be found, so there is no way we can say that prenatal common factors are predominant, or will ever be found to be. The error on the prenatal influence estimate is still relatively high. In contrast, the nonshared environmental factors gave 63% for both males and females and the error was relatively small. This leaves room for little else. These idiosyncratic factors predominate. (Heterosexual origin cannot be studied this way because of mathematical difficulties.)

LeVay does mention that in twin studies of SSA, the nonshared environment factor is significant (see p. 167). While this statement is correct, it is a little misleading. As shown above, not only is it consistently significant (and in many studies the genetic component is not), but it is consistently dominant. However, because there is so much interest in the genetic influence, this fact tends to get generally ignored in the wider literature.

On page 282, LeVay cites roughly even odds that if one member of a monozygotic twin pair is gay, the monozygotic cotwin will also be gay. At best this is misleading. LeVay may be citing the probandwise concordance for monzygotic twins, and this is the type of technically defined nonintuitive concordance essential in further calculations of genetic influence and other factors. But as an illustration for nonspecialists, it is much more helpful to give the pairwise figures. In the present case, taking Bailey et al. (2000)

as an example, if one male identical twin has SSA, only 11% of his cotwins will have the same attraction. For a female, the figure is 14%.

The landmark paper using traditional classic twin study methodology (Bailey, Dunne, & Martin, 2000)—and the one usually cited—did not find any genetic influence on SSA. Instead of that paper, LeVay cited another paper (Kirk, Bailey, Dunne, & Martin, 2000) that used an untried, unreplicated, and rather arbitrary measure of sexual orientation and was about trying different measurement models. It gave a result as high as 60% genetic influence for one of the models. It is worrisome that results are so model-dependent. At best, this selective literature evaluation demanded explanation by LeVay in a footnote. (For further, more detailed discussion, I propose consulting Whitehead, 2011.)

Diversity

LeVay thinks the diversity of gay people has not been studied enough. I agree, but one very recent result on diversity in sexual orientation is a little thought-provoking. That study (Savin-Williams, Joyner, & Rieger, 2012) used five categories—exclusively heterosexual, mostly heterosexual, bisexual, mostly homosexual, and exclusively homosexual—to classify the large sample of young adults from the ongoing ADD Health Study in the United States. As shown in Figure 1, the authors found the percentage of males who classified themselves as "mostly heterosexual" to be about 3% of those who classified themselves as "exclusively heterosexual." However, those who classified themselves as "mostly homosexual" were 55% of those who classified themselves "exclusively homosexual," which is much larger than the 3% for the heterosexual comparison. Although the authors do not examine the issue in detail, the diversity of orientation is many times stronger for the homosexuals compared with the heterosexuals. The same general results are found for the women. One could argue that the same factor is creating greater diversity in the homosexual groups and less in the heterosexual groups—that is, a drive to heterosexuality!

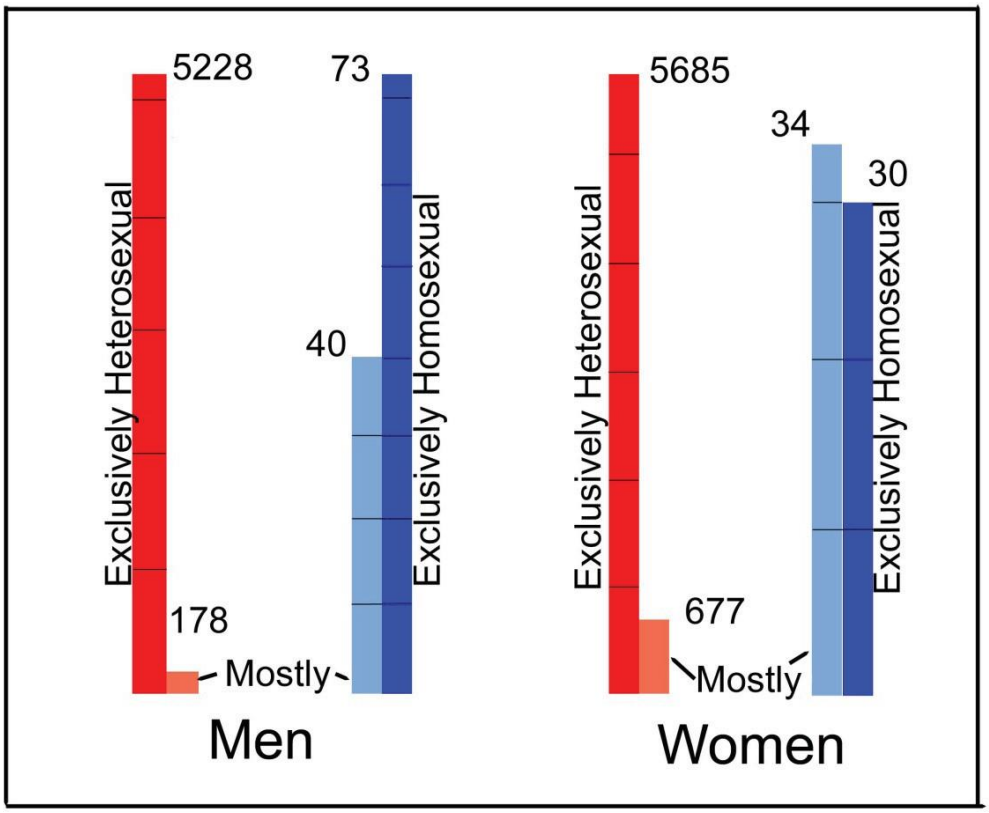

Figure 1. Relative concentration of subjects in the "Exclusive Heterosexual" class. Bisexual class not graphed. Numbers of subjects in a category are shown, and note that each group of two bars has its own independent numerical scale.

LeVay mentions that Bailey's research team could not verify the existence of bisexuals using phallometric techniques. However, a more recent study by Bailey using the same technique came to a different conclusion (Rosenthal, Sylva, Safron, & Bailey, 2011). People with responses to two different erotic stimuli—heterosexual and homosexual—did seem to exist. This was further confirmed recently by a different technique that involved measuring the length of time subjects paid attention to pictures of each gender (Ebsworth & Lalumiere, 2012). If bisexual people exist, there could be slow movement through the bisexual category to exclusive heterosexuality. If bisexual people do not exist, then movement toward heterosexuality would be impossible.

Mental Rotation Tests

Mental rotation tests—a rather pure test of brain function—are often used as an indicator of relative masculinity/femininity. LeVay argues that male-type mental rotation, in which boys score higher than girls, is seen as early as three months, which he thinks is too early for socialization effects. He therefore thinks mental rotation must be innate and related to prenatal testosterone exposure. I assert that such an effect is possibly influenced by the known early *post*natal testosterone surge.

More recently, this has been directly tested by analyzing amniotic fluid for testosterone, then checking mental rotation ability well after birth (Auyeung et al., 2011). There was no correlation. On the other hand, results of an embedded figures test (which measures attention to detail) in both boys and girls well into childhood *was* predicted by fetal testosterone. One might have predicted the girls would do better, but the opposite was true. This is a complicated issue and does not support the simple hypothesis of a prenatal hormone influence predictably organizing the gender of the brain.

LeVay also discusses the way that girls with congenital adrenal hyperplasia (CAH) who experience extreme levels of prenatal androgens are shifted in many of their behaviours toward the masculine side (p. 79). Considering the extreme levels of androgen involved, the real surprise for me is how small the effects actually are. He is disconcerted that these girls only show typical feminine values for the mental rotation test rather than more masculine values. However, they are better at the targeting visual test, which would be more typical of boys. He also mentions that the mental rotation test on CAH boys actually showed worse performance than on unexposed boys, an unexpected result. He introduces yet another level of hypothesis to explain this, saying that perhaps they are not exposed to androgens to the degree supposed. However, it is known that the adrenals are active in fetal development from weeks 10 to 13, before the development of the male genitalia and testosterone surge at weeks 14 to 20, so there should be adequate androgen exposure.

Do Elder Brothers Make You Gay?

The elder brother hypothesis derives from a long series of papers—particularly by Blanchard and Bogaert in Canada—showing that the greater number of elder brothers in a family, the more likely it is that a later-born male will be SSA. LeVay is not completely uncritical of these and cites a number of studies by other groups that are now failing to find elder brother influence. Another more recent study was done by Zietsch and colleagues (Zietsch et al., 2012). One of the best studies mentioned by LeVay (Frisch & Hviid, 2006) involving a very large Danish sample (2 million people!) did not find an elder brother influence but, contrary to the hypothesis, found an elder sister effect, which was also noted in a few other studies.

LeVay correctly points out that elder brother data depend on family size. The most important source for this emphasis is a paper by one research group that found an elder brother effect, only to have it vanish when family size was factored in (Langevin, Langevin, & Curnoe, 2007; see also the subsequent discussion with Blanchard in the same journal). LeVay does not cite this debate, and it is far more important than most people think. It may mean much of the literature on the elder brother hypothesis needs to be reevaluated, and this is now impossible because maximum family sizes are usually not available from historic census data.

However, LeVay concludes that if there is an elder brother effect, it "does not work by broadly feminizing the brain development of late born sons" (p. 269). I suspect further work will support that.

I personally think a simple psychological explanation of the supposed elder brother effect is rejection or rebelliousness toward the overbearing behavior of a set of elder brothers. Either one joins them, imitates them in the best way possible, and heads for the opposite-sex attraction (OSA) world, or the younger brother rejects their masculine modelling and starts on a path toward SSA.

The elder brother hypothesis is usually explained (including by LeVay) in terms

of the maternal immune hypothesis, in which a male fetus creates an immunological reaction by the mother, who does not have Y-chromosome-specified proteins. The hypothesis is that the mother's immune system could attack the brains of subsequent male fetuses, impairing the masculinization of their brains and causing them to develop SSA. This condition is also supposed to cause lower birth weight.

I have pointed out elsewhere (Whitehead, 2007), as have others (Zietsch et al., 2012), that the discordance of identical twins for SSA makes the scenario of immunological attack very unlikely. However, LeVay mentions an important Danish paper (Nielsen, Mortenson, Schor, Christiansen, & Andersen, 2008) that at least shows that the maternal immunological attack on male fetuses is real, but confirms doubts in my mind about whether this is relevant to sexuality. The study, based on about 350,000 births from the Danish medical records, found lesser birth weight for later males, consistent with the hypothesis.

There were some conflicts with a similar previous Norwegian study of 180,000 births (Magnus, Berg, & Bjerkedal, 1985) that concluded flatly that the maternal immune hypothesis was untenable (Whitehead, 2007). However, the much more recent Danish study is larger, more thorough, and better controlled, and the effect on birth weight is likely to be real. While it may be real, the birth weight effect is quite small and the conflict between the studies shows the classic difficulty of looking for a very small effect in the presence of many other factors that may easily overwhelm it.

Both studies found that birth weights of *girls* as well as boys were affected by the previous birth of a brother. This is contrary to the hypothesis, which argues that only subsequent *boys* should be affected; the Danish researchers think this is due to a lessening of specificity of immunology with time (another layer of hypothesis). It must be a large decrease of immunological specificity indeed if the mother's immune system can now attack either sex, but such a decrease calls into serious question whether the effect is still strong and specific enough to attack male brains and cause homosexuality.

The Danish group (Nielsen et al., 2010) posited further evidence of an "anti-male" antibody: when recurrent miscarriages occurred after a first successful birth of a boy, the sex of a successfully born later child was 83% likely to be female. The Danish researchers have followed this group of mothers for a long time—some of them since 1986—and an obvious test should determine whether there is increased prevalence of SSA in any second-born males in this group.

From a Danish study (Frisch & Hviid, 2006), LeVay fails to point out the many social correlates with SSA or "homosexual marriage"—older mothers, divorced parents, absent same-sex parents, and being the only or youngest child. Each younger sibling decreased the chance of homosexual marriage in the older sibling by 9.2% for men and 13% for women, which cannot be a prenatal effect. Frisch and Hviid concluded that whatever other factors might be involved, parental interactions were important. The same study also presents various effects on heterosexual orientation as measured by heterosexual marriage. Similar to genetic effect influence, most social effect influences were modest in size.

Blanchard/Bogaert introduced an extra layer of hypothesis in which left/right-handedness is a modifying factor as to whether a subsequent son is SSA. LeVay is dubious; so am I. Blanchard has very recently published yet another independent layer of hypothesis (this one also including lesbians) to try to explain SSA in firstborns (Blanchard, 2012). I'm even more dubious about this hypothesis and await some replication.

Bodily Differences

LeVay's book has a very good section reviewing the physical body differences in straight/SSA people, and it is now established that there are some statistical differences. Male gays are slightly shorter and lighter than other males, and lesbians have longer arm-to-body ratios than the average for women. Again this is statistical only, but could in principle be some evidence for a biological origin of sexual orientation. However, for me it raises the question of how far self-image actually arises from the bodily properties. Does a male smaller than

average find himself not well suited to sports, think himself less masculine, suffer ridicule, and tend to SSA? Does a woman with more masculine finger ratios and longer arms think of herself as less feminine? Such questions need to be answered. And how much of a factor is a conventional gender-appropriate bodily shape in pushing us toward heterosexuality?

Genes

LeVay mentions the studies trying to find genetic linkages and scans of the whole genome to find SSA-related genes. I believe these are unlikely to succeed. The perspective of the last ten years is that linkage studies to genes for any trait are notoriously, indeed embarrassingly, difficult, with numerous incorrect publications that are discredited when a whole genome scan is done—at which point entirely new and unsuspected candidate genes are revealed. The rule of thumb is now that many or very many genes are involved, each with very small contributions. There is a genetic contribution for many things, and I expect that this will ultimately be shown for SSA, but the task of establishing it is logistically immense, possibly demanding analyses on 100,000 people as it did for genetic studies on schizophrenia. It may show that some specific genes are involved, but to put a numerical figure on the extent of the combined influence, we will still need twin studies—and we already know from existing twin studies that the influence is minor.

I suspect many genes each have a small influence, but this makes it hard to explain why family studies give such erratic results for the appearance of SSA. A trait dependent on many genes will vary slowly with the generations, as *g* ("IQ") does. Many genes, each with small effects, should produce overall a bell-shaped distribution of sexual orientation, which is the opposite of what we find—sexual orientation has a J-shaped distribution. Aware of this difficulty, LeVay inserts yet another layer of hypothesis to explain why sexual orientation is channelled into two streams. According to LeVay, this demands prenatal organization of the brain into a kind of masculine and feminine channel, or at least awareness of the two genders. I find this proposition that something like Jungian archetypes are in the brain interesting and

novel but speculative; I also find it contradicted by the quite slow development of awareness of gender in young children rather than full gender recognition from birth.

Culture

LeVay hypothesizes there is a basic stratum of homosexuality in culture having a biological origin. But he agrees homosexuality is expressed in rather wildly varying ways in different cultures and eras. He does not mention that such diversity should usually mean that genetic influence is rather weak. Genetically caused homosexuality would be tightly circumscribed in expression and change little over centuries. He does acknowledge that modern patterns of SSA relationships between adults were historically rare. But if the predominant historic pattern has been pederastic, would this mean any worldwide genetic pattern causing SSA was also most "naturally" pederastic?

LeVay states, "My conclusion . . . [is] that sexual orientation is indeed a fairly stable aspect of human nature, and that straight, gay, and bisexual people have existed across many, perhaps all cultures" (p. xii). This is misleading because it implies SSA is stable. One can indeed say the statement about stability is true of heterosexuality, but it is far less true of homosexuality. This is shown to greatest extent in a study (Savin-Williams & Ream, 2007) in which sixteen-year-olds who were OSA were almost 100% still the same way a year later, but those initially SSA had overwhelmingly changed to OSA. From these data, and confining the time period to adolescence, OSA is at least 25 times as stable as SSA. A lifelong figure would be not so extreme, and a rule of thumb would be about 15 times the stability. This does not argue for SSA stability.

The cultural history also shows large change of homosexual customs, sometimes within one generation. This does not demonstrate SSA stability, nor does it argue for likely genetic influence.

LeVay points out the huge shift in family patterns over the last few centuries, and gives an argument why possible homosexual genes could have become more prevalent.

But selective breeding experiments with animals require about a dozen generations to change behavior profoundly, and under LeVay's hypothesis, an extraordinarily selective human breeding pattern would be needed to produce a change in sexual orientation. It is highly doubtful that his mechanism can be correct for the timescale quoted. We must also remember that with today's smaller family sizes, there would be far fewer elder brothers than in the past!

Animal Homosexuality

LeVay also surveys animal homosexuality, finding that "homosexuality in the sense of a durable preference for same-sex partners, has not been widely described among non-human animals" (p. 69). Rather, there is what he describes as "broad bisexual potential." Homosexuality to him seems like a "second-best choice" for the animal, saying that "male-male partnerships often break up if females become available" (p. 67). He correctly points out that the parallels with human homosexuality are often exaggerated but he thinks longer-term preference is seen in domestic sheep and reports on brain changes observed in them that apparently correlate with SSA.

From my point of view, however, it is similarly a question of how far these brain changes are due to the psychosexually artificial conditions of farming and whether the brains have changed in the short term in response to these environments. I would also note the relative lack of mention of reproduction as a theoretical framework; this, rather than sexual orientation itself, is overwhelmingly important for animals.

How far can human and animal sexuality be compared in any case? The richness of human sexuality and family life makes comparing it with sex in animals like comparing a love sonnet with the grunt of a pig.

Is Change Possible?

Given his adherence to a strong prenatal theory of sexual orientation origins, what does LeVay think about the possibility of change in humans? Only homosexuality is dis-

cussed: "The majority viewpoint among mental-health professionals is that this so-called *conversion therapy* has little chance of success and can cause significant harm by reinforcing the gay person's negative self-image" (p. 12). No in-depth references are given for this, and such in-depth support does not exist.

LeVay quotes the Spitzer study and agrees that "at least a few highly motivated gay people can be helped to engage in and derive some degree of pleasure from heterosexual relationships and to pay less attention to their homosexual feelings. This should hardly come as a surprise, since we know that many gay people were heterosexually married and had children before coming out as gay" (p. 13). This simple and reasonable conclusion from the Spitzer study seems to at last be getting some unbiased attention! It is pretty refreshing these days to find an evaluation that is not totally blinkered. But he thinks therapy to stop gay self-hatred is more useful.

He cites literature in which some rare medical conditions seem to have caused a change in sexual orientation and would not rule out others being found. This is a much better approach than the ideological one that change is *a priori* impossible. I suppose it makes sense even from a strongly essentialist point of view that if the essence—the biological hardware—changes, very possibly the sexual orientation might also. He agrees that "sexual partner preference can change" (p. 286), at least under some conditions.

Although LeVay's book is about the influence of prenatal hormones, this section does not directly comment on whether such a link makes any change more difficult. The fact that the section is present at all suggests there is such a link in LeVay's mind and probably in the reader's mind as well. But the outcome seems to be a qualified agreement that the influence of the prenatal hormones is not absolute, and perhaps some forms of therapy might even have some effect under some conditions.

As to whether choice could be a factor in homosexuality, LeVay quotes a survey from the *Advocate* in the mid 90s that only 4% of gay men and 15% of lesbians said that choice had anything to do with why they were gay (p. 41). Such a survey sample is very

biased (as is any sample from clinical experience) but seems numerically to be in the right region for an estimate of "choice." This is because unbiased Gallup polls show that 90% of GLB people currently believe they were born that way, which leaves little room for a large choice element. The latter, rather than the *Advocate* survey, is probably the best available simple evidence that choice is a minor factor in the origin of SSA.

Trying Too Hard?

I think the reader of this book or any book dealing with this subject will be rather overwhelmed by the complexity of the subject. There are multiple aspects, multiple theories, and lack of consistency in experimental results and surveys. I think the general welter of inconsistent results is a mark of a discipline that is not yet mature. I believe there is a lesson to be learned from the gene-linkage surveys (not just for SSA) that appeared to be so exciting but were often totally wrong. I see also the piling up of multiple hypotheses, particularly in the field of the elder brother effect. There is some truth there, but I think the whole subject will ultimately have to be reconstructed more cautiously. Multiple hypotheses are fun, but more than two layers of them is stepping out onto the abyss. We need to wait until some of the findings are explained without (please) yet more layers of hypothesis.

LeVay's conclusion—"We [homosexual people] should be valued, celebrated, and welcomed into society rather than merely being tolerated" (p. 298)—is one of the few such "activist" comments, and the tone of the book is overwhelmingly academic.

Conclusions

The subtitle of the book seems to imply basically the same principle for development of homosexuality and heterosexuality—prenatal exposure to greater or lesser amounts of hormones. Although these have some effect, so do a multitude of other factors, and it is time to abandon a single-origin hypothesis. Rather there are multiple reasons for SSA that

include biological randomness, body shape, prenatal stress, early maternal interaction, early postnatal hormone effects, sibling interaction, sexual abuse, media influences, mental/fantasy life, early sexual experiences, paternal influence, and nonstandard masculine or feminine interests. Since some of these impact a few individuals to an unusually strong degree, there is no substitute for individualized clinical analysis. This may far better reveal "the reason why" one is gay or straight. Hopefully the mix of causes means that surveys of large populations can be summarized profitably by the sociologists.

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