Neither Genes nor Choice:

Same-Sex Attraction Is Mostly a Unique Reaction to Environmental Factors

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Abstract

This paper uses the seven largest twin registry studies to emphasize that same-sex attraction (SSA) is mostly caused neither by genetics (weak to modest influence) nor direct shared environment (very weak), but by many nonshared individualistic events and reactions, none of which is more than a small minority of total influences, and may well be differing reactions to shared environment. Twin studies sum up all influences (known and yet to be found) and their interactions, so this conclusion about the importance of nonshared factors is unlikely to change with future research into biological or social causes. The mean genetic percentages* for men and women are 22 and 33% respectively and are not significantly different statistically. They are almost certainly maxima, likely to halve with further research. Recent findings of nonshared environmental epigenetic causes (genetic expression influenced by the environment) lead again to a conclusion that the genetic influence has possibly been overstated. Nor is deliberate choice of orientation significant; even for adult sexual choice (e.g., heterosexual mate selection), chance predominates. For the development of sexual orientation (ten being the mean age of first attraction), deliberate choice must be a very unusual event.

* Erratum A misleading phrase has been removed

Introduction

Over the last fifty years, prolonged debate has raged over the question of where same-sex attraction (SSA) originates—is it in prenatal factors, such as genes, or postnatal factors, such as the style of family upbringing? The two groups of protagonists were mainly biologists (who believed in genetic causes) and psychologists (who believed in environmental causes). The purpose of the present paper is to present the evidence that idiosyncratic factors overshadow both the prenatal and parental (environmental) factors. This paper posits that SSA is mostly neither innate nor directly a matter of upbringing or social factors, but rather results from unique reactions to personal experiences. This conclusion is not likely to change with future research.

A study of most social factors possibly connected with SSA found that only a small percentage of overall SSA was accounted for by any specific influence, though all influences on adolescents taken together seemed highly significant (Bell, Weinberg, & Hammersmith, 1981). A later study confirmed those findings (Van Wyk & Geist, 1984). Thus, although social/family factors as a whole were quite significant, they were not significant individually. Research showed that there were many paths to SSA, and any given factor affected only a small minority of individuals. Obviously, some individual factors—such as sexual abuse—were very important to the individuals involved. Bell, Weinberg, and Hammersmith (1981) then totally erroneously stated that SSA was fixed and permanent in a person from childhood on—in other words, there was "tracking" from childhood SSA to adolescent SSA and on to adulthood. Hence, they concluded that their finding of significant social effects as a whole was only a statistical artifact.

The most significant single influence they found was childhood gender nonconformity. It used much weaker criteria than full Gender Identity Disorder as understood today, but even as the "most significant" single influence, it directly led to adult SSA in only 12% of cases—a weak effect.

The literature has subsequently stated erroneously that the work by Bell, Weinberg, and Hammersmith (1981) disproved that social factors had any general bearing

on SSA. Rather, Bell et al. could not demonstrate much importance for *individual* factors. This will be discussed in more depth in a forthcoming paper (in preparation).

Reacting to their modest result for individual social influences, Bell, Weinberg, and Hammersmith (1981)—without actual evidence—thought biological factors might be predominant. They did not discuss a possible role for chance, which from their data alone could have been a predominant part of the variance. The present paper, however, finds using twin studies, that chance (nonshared environmental factors) is indeed predominant, and shows that biological factors—like genetic factors—are likely to contribute less than 20% of the variance in the development of SSA.

The argument in this paper tries to reconcile some other contradictory findings—weak social factors for production of SSA being shown in sociological studies, but strong factors being found in individual clinical studies—by suggesting that erratic (unique) individualistic reactions to shared social factors are important as causes of SSA.

Many nontwin studies of various possible correlative biological factors contributing to SSA followed the 1981 paper (Blanchard, 2008; Kraemer, Noll, Delsignore, Milos, Schnyder, & Hepp, 2006). But none of the subsequent studies found a universal biological factor that might account for most SSA. The large number of such biologically oriented papers (about two dozen a year in PubMed alone) makes it superficially appear that biological factors must sum to a strong influence. However, since there can be overlap, synergistic, or competing influences, a summary measure is highly preferable—such as from twin studies. This paper demonstrates that twin study calculations show all biological factors—including those yet to be found—sum to only a weak to modest effect.

In this paper, identical twins will be identified as mz (monozygotic), and fraternal twins will be identified as dz (dizygotic).

Twin Studies as a Measure of the Various Contributions to SSA Influences

A central concept in twin studies is explaining the variation of something in the population—such as SSA—by calculating the relative contribution of genetics, shared postnatal environment, and nonshared postnatal environment (unique experiences or unique reactions to shared experiences).

The early post-World War II SSA studies by Kallmann (Kallmann, 1952a; Kallmann, 1952b) were performed on identical twins who were also mental patients. These studies found an overall 92% concordance for SSA (rather than the 100% sometimes quoted). These findings were never repeated in subsequent samples, possibly partly due to very lax and anecdotal SSA diagnostic criteria. Mz concordance is a simplified form of twin studies and compares shared and nonshared factors only. If the concordance was as high as Kallmann claimed, it would have meant shared factors (genetic and/or family factors) were very strongly influencing SSA development. Subsequent studies have not supported that finding.

Later studies followed the classical twin research design of comparing concordance in both mz and dz twins. Concordance in mz twins shows the influence of genetics, and concordance in dz twins allows for shared nongenetic influences. The greater the difference between the concordance of mz and dz twins, the greater the genetic influence. SSA research used samples drawn from the gay/lesbian/bisexual (GLB) community by a volunteer or "snowball" system, but at the beginning of the new millennium such studies were shown to have been subject to the known general problem that similar mz twins tend to volunteer for studies on a publicized topic and dissimilar mz twins do not (Kendler & Eaves, 1989). The results ultimately proved to be a great deal more biased than researchers had thought possible. The genetic influence fraction in those early studies, about 50%, is now generally believed to be significantly inflated—but, unfortunately, it is still quoted. This paper—which follows twin studies mainly since the year 2000—will suggest the true answer is likely to be below 20%. In other words, we've known for at least a decade that genetic influences on SSA are very little.

SSA studies based on twin registers, in which twins are enrolled on a voluntary basis simply because they are twins, probably produce much less bias than the "snowball" system. However, concordant twins (particularly mz twins) from such a register may still be disproportionately represented: participation in surveys that use twin registry members is not compulsory for any twins, and concordant twins tend to over-volunteer. The extent of this bias is not well-known, but is probably minimal, as seen by comparison of the results for SSA in Kendler, Thornton, Gilman, and Kessler (2000) and Bailey, Dunne, and Martin (2000). The results for SSA in these studies were very similar, though the first was a unique unbiased national survey and the second was drawn from a twin registry. Population studies, such as those from the highly documented health systems of the Scandinavian countries, will capture all twins with no bias, but do not register sexual orientation *per se*. Even these studies still need special surveys with participants' consent, which may again introduce bias through over-volunteering.

Modern Twin Registry Studies of SSA

Early twin registry studies contained such small numbers of twins that this paper concentrates instead on the major registry studies that have the largest samples. These large studies and their countries of origin include Buhrich, Bailey, and Martin (1991), Australia; Hershberger (1997), United States; Bailey, Dunne, and Martin (2000), Australia; Kendler, Thornton, Gilman, and Kessler (2000), United States; Bearman and Brueckner (2002), United States; Santtila, Sandnabba, Harlaar, Varjonen, Alanko, and von der Pahlen (2008), Finland; Langstrom, Rahman, Carlstrom, and Lichtenstein (2010), Sweden; and Alanko, Santtila, Harlaar, Witting, Varjonen, Jern, Johansson, von der Pahlen, and Sandnabba (2010), Finland. It is now common to find studies of a few thousand pairs of twins, and the last six referenced studies were generally individually larger than all the pre-2000 studies combined.

Varying measures of SSA have been used. However, we now know that the errors are so large that it makes no difference whether a criterion of attraction, fantasy, or

behavior was used to establish SSA. As a result, no differentiation was attempted in the present paper.

This paper emphasizes what has been implicit for some time: SSA follows the same general pattern seen in many other traits (Turkheimer, 2000) in which genetic factors are significant but modest; postnatal factors affecting both twins (such as upbringing) are apparently less important (quite often close to zero influence); and nonshared factors (usually described as nonshared environmental factors) are usually more important than either and usually more than 50% of total variance (=influences, approximately), meaning nonshared factors generally have a moderate to strong effect.

Psychologists reacted with frank disbelief and hostility to assertions in general twin studies that family influence was usually found to be so small, because their clinical experience with individuals unequivocally showed the opposite. Individuals they met were deeply affected by various unusual events in their histories. The conflict arose because the samples studied by each group were very different. Highly self-selected individuals in clinics have characteristics of those at one extreme end of a continuum, but prevalence of those characteristics often bears little relation to that of a carefully selected population-based sociological sample. Both conclusions, though conflicting, are therefore correct for their two samples, which consist of either the whole population or individuals (Whitehead, 1996). As described in that paper, clinical studies give unprecedented detail about individual psychological mechanisms that certainly occurred in the small sample described. On the other hand, surveys give a grand mean; the mechanisms revealed are often rather uninteresting and hide a wealth of significant detail. To some extent twin studies have fallen into the latter trap.

However, another important resolution of the conflict was the discovery that children reacted to shared family factors in differing ways rather than in similar ways (Plomin & Daniels, 1987). Overwhelmingly, there is individualized reaction, so the observations of the clinicians are valid for those they are studying. But this also means that *responses* to shared experiences (in a family, for example) are mostly not shared. In

other words, sometimes two children in the same family respond differently to the same environment. Individual perceptions of experiences and subsequent responses to those experiences seem to be the key factor in the development of same-sex attractions.

Results

This paper does not use the rigorously random twin study by Kendler, Thornton, Gilman, and Kessler (2000), which—though the result is much like the others—had to combine results from men and women. Nor does it use the survey of adolescents by Bearman and Brueckner (2002), because among the adolescents there was a major SSA measurement problem and enormous variability from year to year (Igartua, Thombs, Burgos, & Montoro, 2009; Savin-Williams & Ream, 2007).

The Santtila, Sandnabba, Harlaar, Varjonen, Alanko, and von der Pahlen (2008) survey was the largest, involving 2,334 pairs of twins. Like the second-largest survey by Langstrom, Rahman, Carlstro|m, and Lichtenstein (2010), it used the Scandinavian health records, ensuring a relatively unbiased sample.

Results: Calculated Genetic Fractions

The mean in figure 1 for men is (22± 20)% where the error is the standard deviation. It made no statistical difference whether this was recalculated restricting the results to attraction or behavior. All surveys were therefore pooled.

The errors are very large. The standard error (a measure of how far the result is from zero) is 4%, so the result is far from zero and is real, but a 22% genetic fraction is considered weak. Conventionally in the twin literature, 25, 50, and 75% are considered weak, moderate, and strong, respectively, based on the inherent mathematical relationship, since 0 is no relationship and 100% is a relationship totally dominated by the genetics. A result between 25 and 50% is considered modest.

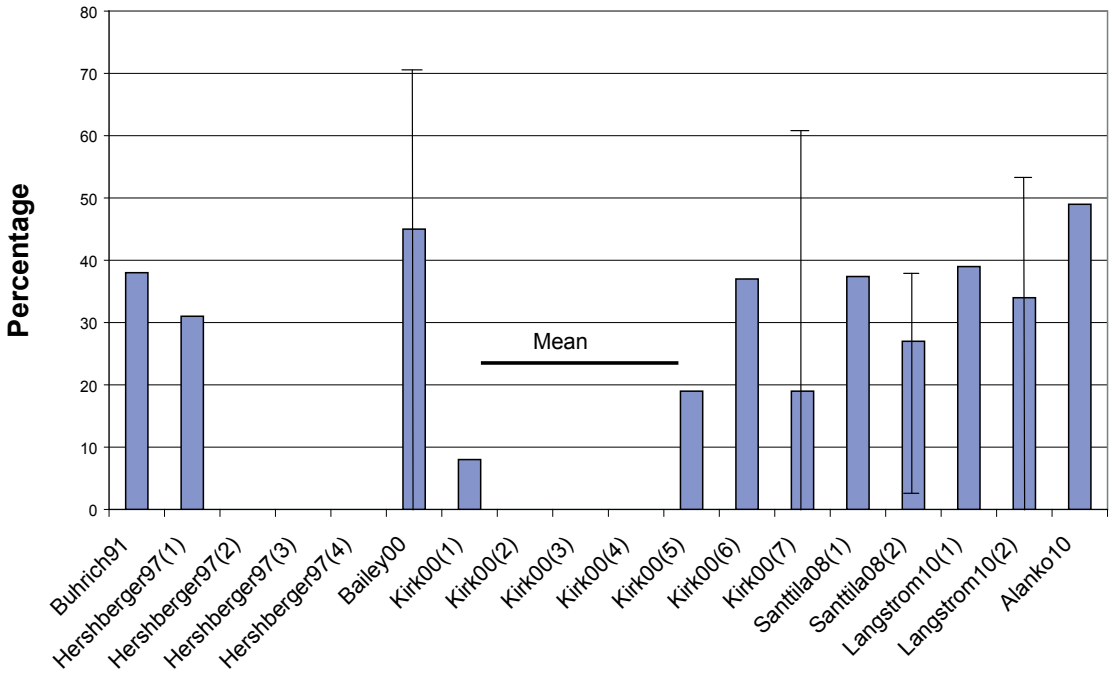

Figure 1. Estimates of genetic contribution to male SSA by various measures. Typical 95% error bars for selected studies are given. Absence of a histogram bar means the numerical result was zero. References and measurement basis: Buhrich, Bailey, and Martin (1991)—Attraction+fantasy+contacts. Hershberger (1997)(1)—Attractions when older than 25 years; (2)—SSA partners when older than 25 years; (3)—Sexual orientation (gay, bisexual, straight); (4)—Same, but modeling included siblings. Bailey, Dunne, and Martin (2000)—Sexual orientation: Kirk, Bailey, Dunne, and Martin (2000)(1)—Same sex (SS) feelings now; (2)—SS partners in last 12 months; (3)—Fantasy; (4)—Sexual orientation; (5)—Attracted once or more over life to date; (6)—Fantasy now (excitement or disgust at idea of SS contact); (7)—SS partners over life to date; Santtila, Sandnabba, Harlaar, Varjonen, Alanko, and von der Pahlen (2008)(1)—Potential to be SS-involved (fantasy); (2)—SS partners in last 12 months; Langstrom, Rahman, Carlstro¦m, and Lichtenstein, (2010)(1)—Any lifetime SS partners; (2)—SS partners over life to date; Alanko, Santtila, Harlaar, Witting, Varjonen, Jern, Johansson, von der Pahlen, and Sandnabba (2010)—Attraction+partners normalized to degree of libido.

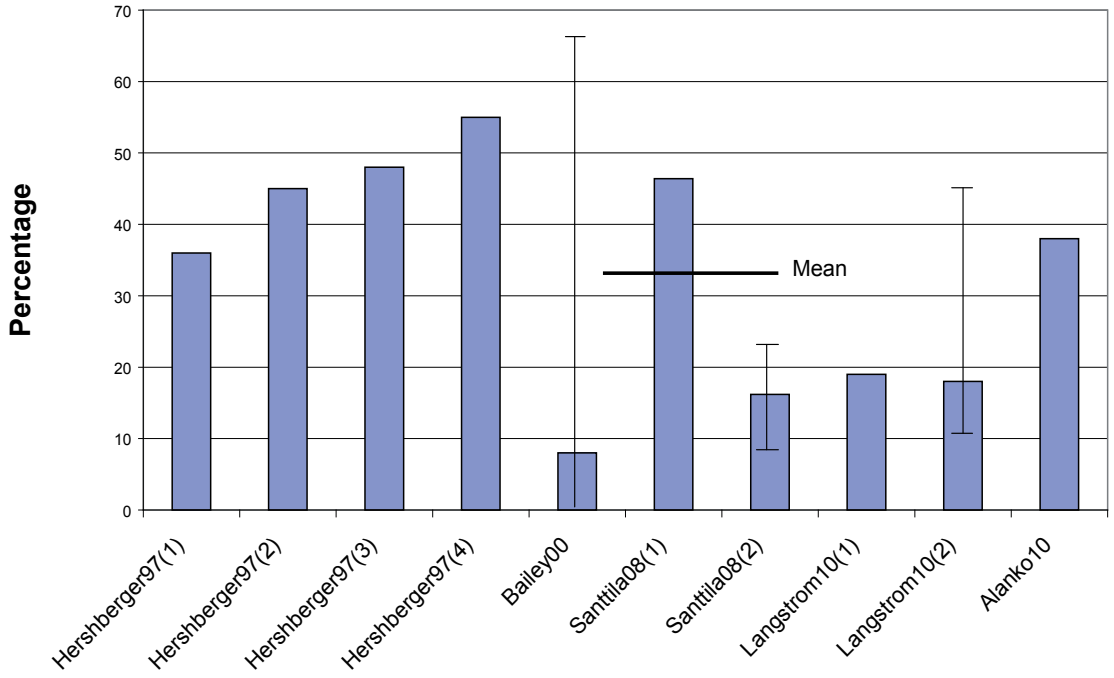

Figure 2. Estimates of the genetic contribution to female SSA. References as for figure 1, plus Kirk, Martin, and Bailey (2000).

The mean in figure 2 is (33 ±16)%, with a standard error of 5%. So a rough estimate of genetic contribution to female SSA is 33%. The result is far away from zero and is real, but its strength is only modest.

The results from the different sets of investigators are generally about the same within error. But when compared with classical twin studies on other traits, the errors are alarmingly large—a result that arises from the form of the statistical distribution of SSA in the population.

Even with so many SSA studies, the statistical errors are still so large that there is no statistically significant difference between the two percentages for men and women. The mean figures for the genetic content for men and women—22% and 33%, respectively—are still subject to the problems described later in the technical appendix, which will point out they are maxima and almost certain to reduce with further research.

Shared environmental factors in the studies cannot be distinguished from zero and are not graphed here. This is probably an illusion, as implied previously; in twin studies they often contribute to nonshared environmental effects.

Results: Nonshared Environmental Effects

In contrast, the errors on nonshared environmental fractions are much smaller. Figures 3 and 4 show the nonshared environmental fraction of SSA.

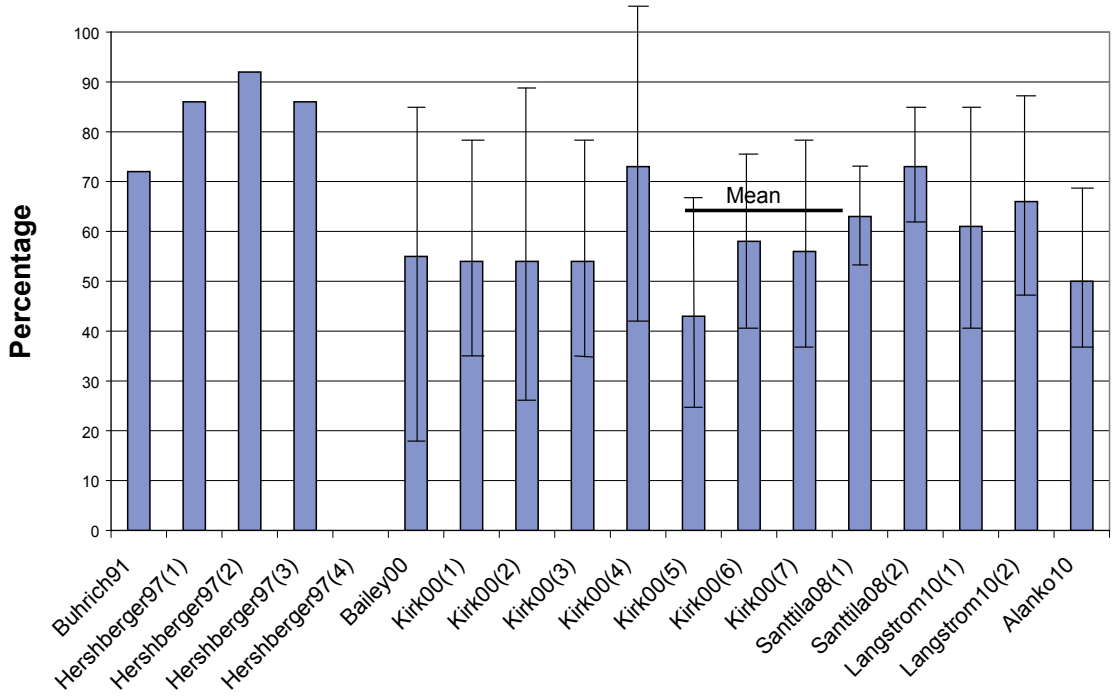

Figure 3. Nonshared environmental fraction for male SSA. The Hershberger (4) result is not missing, but is zero. Error bars are 95% confidence intervals, not available for the early studies. References as for figure 1.

The mean is (64±14)% and the relative standard deviation is much lower than for the genetic fraction estimations. This is a moderately precise estimation.

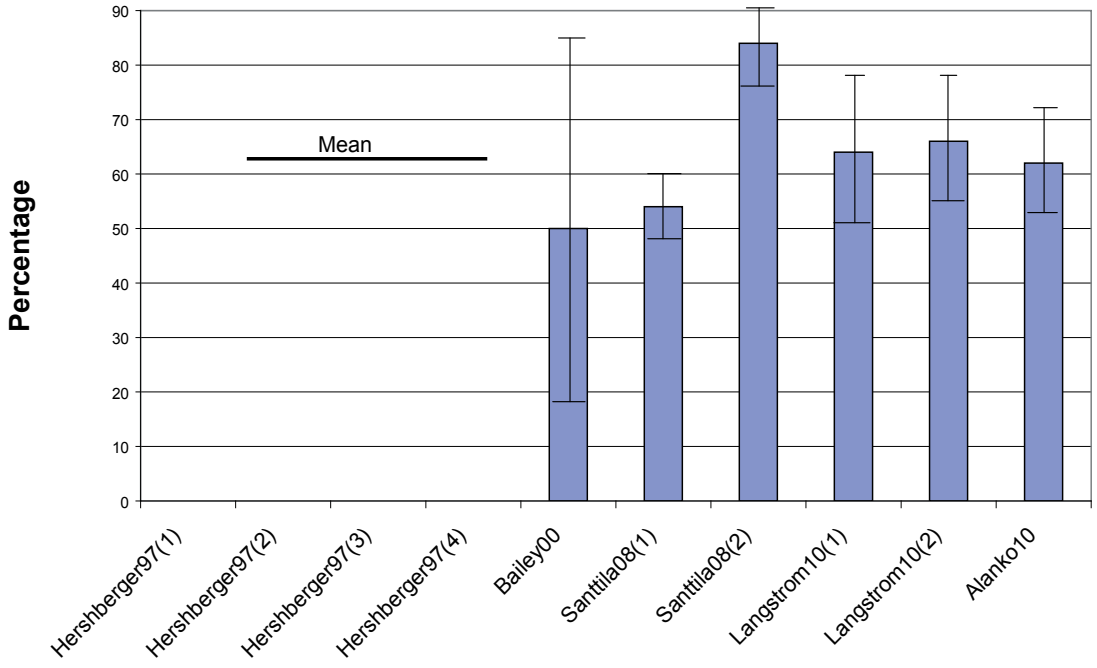

Figure 4. Nonshared environmental fraction from twin studies for females. References and same scale as for figure 2. (The Hershberger paper does not have female data for nonshared environment, only data for the genetic fraction).

The mean is (63±12)%, statistically the same as for the males.

Discussion

The relatively low results for the genetic fraction are in huge contrast to the genetically programmed events of puberty. Twin studies put the very strong genetic influence on pubertal events at 90% (Silventoinen, Haukka, Dunkel, Tynelius, & Rasmussen, 2008). This is much higher than the 22% and 33% means for SSA. The degree of any genetic programming must be much less for SSA than for puberty.

The calculated nonshared environmental contribution at 63 to 64% is moderate to strong. It is likely to increase with further research.

What Comprises Nonshared Environment?

From traditional twin studies, we therefore find in summary that SSA is predominantly (63%) produced by erratic, unique factors of a type that could affect one mz twin but not another.

An important outcome of the studies, therefore, is that the nonshared environment fraction is always two to three times the genetic fraction and has much smaller errors. Shared environment fractions are very low, consistent with zero—but, as mentioned, this paper suggests that varying reactions to shared environment are, in fact, very important to individuals.

Nonshared environment is characteristically elusive because it is erratic. There are probably very many individualistic experiences and factors that are very difficult to capture in surveys, explaining why general research into nonshared environment has been so unproductive. Turkheimer (2000) cites research that since 1987 had only accounted for 2% of such effects. However, in their study of socially problematic behaviors, Rodgers, Rowe, and Li (1994) accounted for a much larger proportion; they ascribed nonshared environment to features of the home that were experienced differently, so some research has succeeded.

Nonshared environment contains several components: (a) possible measurement error of SSA; (b) biological randomness; (c) differing random events affecting one mz twin but not the other; and (d) differing random psychological reactions.

The measurement error of SSA in adults is probably much less than in psychiatric studies of twins, in which conditions can be hard to diagnose. The error is probably not a large proportion of the "nonshared environmental" fraction.

There is some inherent randomness in gene expression. Gene expression is not a perfect clockwork-type system; it contains "noise" or variation in the expression of genes, depending on the circumstances or point in time (Bar-Even, Paulsson, Maheshri, Carmi, O'Shea, Pilpel, & Barkai, 2006). There is also randomness introduced when the

chromosomes recombine after the meiosis that forms the ova and sperm (Coop, Wen, Ober, Pritchard, & Przeworski, 2008). Those recombination events can be imperfect, introducing random errors.

This makes individual animals irreducibly different from each other; however, many researchers breed for uniformity and ensure that the environment is completely standardized. This effect has been known and puzzled about for many years. For example, breeding colonies of laboratory rats, however much inbred, always contain significantly variable-weight individuals. The same applies to cloned animals—and, therefore, human twins.

Other interesting mechanisms producing "epigenetic" differences within the category of randomness have become recently known; these involve environmental influences on genes and are considered further in the technical appendix.

This biochemical-connected randomness is a contributor to that part of the variance considered to be nonshared environmental. We do not presently know how large a percentage this is.

Some examples of differing random events that could influence one mz twin but not the other—especially among males—could include male sexual abuse, bullying, exposure to same-sex pornography, encounter with someone interested in same-sex experimentation, and bad experience during encounters with the opposite sex. In only a minority of individuals would any particular factor be a major reason for the development of SSA. In general, however, an unusual event—particularly of a sexual nature—has a formative effect that may outweigh routine years of other types of experience.

The nonshared environment may occur when individual children react differently to the same family environment or dynamics. For example, identical twins react differently to the same classroom (Oliver, Pike, & Plomin, 2008). Bailey and Pillard (1995) found identical SSA nonconcordant twins differed in some perceptions of childhood. Identical twins could also misperceive family dynamics in different ways.

Judging by the low values for shared environment in twin studies generally, these differing reactions to the same environment could be the predominant contributor to the random fraction.

All these random factors mean that it is likely each case of SSA will have an almost unique history, though some broadly common themes may be present.

Choice as a Possible Factor

Is deliberate personal choice one of the idiosyncratic, erratic factors involved in SSA? This seems very unlikely but is raised here mainly because it tends to be a possible issue associated with SSA when research results are discussed in the public arena. Most other twin study traits would not be given the same type of attention by the public. Whether choice has the usual adult meaning must be considered in relation to the age of first attraction.

Many papers show that the mean age of first attraction to the same or opposite sex (with 1 sigma error) is 10.9±4.5y for same-sex attraction and a similar 10.3±4.8y for opposite sex attraction (Hamer, Hu, Magnuson, Hu, & Pattatucci, 1993; Whitam & Mathy, 1986; D'Augelli, Grossman, Salter, Vasey, Starks, & Sinclair, 2005; Floyd & Bakeman, 2006; Floyd & Stein, 2002; Grossman, 2008). The standard deviation is exceptionally wide, and programmed biological events have a significantly smaller standard deviation (paper in preparation). When the referenced studies were published at the end of the twentieth century, first attraction to the same or opposite sex preceded puberty by nearly two years. Subsequent to puberty, attraction becomes more clearly sexualized, and identification as GLB usually occurs during adolescence. Deliberate choices about sexual preference prior to ten years are probably quite erratic and immature.

Even when clear choice is involved as an adult, there is evidence that random factors can be more important than the genetic elements. Thus in studies of opposite sex

attraction (OSA) mate selection and similarities in chosen partners, the genetic influence on mate characteristics was 34% genetic and 54% due to random factors—the familiar pattern in behavioral genetics (Philippe & Ann, 2005). One can easily imagine that there could be a significant degree of chance in finding a mate who is personally attractive. Even in this case, "choice" is overshadowed by chance. Another paper studying OSA concluded that the genetic component to most preferred love styles or forms of romantic attraction was zero, that there was a small to modest contribution from the shared environment, and that there was an overwhelmingly large contribution from the nonshared environment, ranging between 61 and 85% (Waller & Shaver, 1994). In both of these papers that examined choice, the nonshared environment was predominant.

Mate selection is probably one of the strongest examples of deliberate choice among life decisions—but, as seen above, even it has a strong random component. For children, the idea of such deliberate choice is even less applicable. It is hard to think of any major life decision that is made involving informed choice at the age of ten. Although cases probably exist, it must be very rare to find any individual who makes a conscious choice about SSA or OSA at that age and who can describe the event and process later. Contrast that to most adults, who can describe clearly the decision to live with a partner. This means that the unique factors rather than deliberate choice must be strongest among children.

The universal experience of those with SSA is that they neither deliberately chose it nor initially welcomed it; indeed, SSA is a significant factor in early suicide attempts. Similarly, the experience of those with OSA is that they did not deliberately choose it, in any common-sense use of the term *choice,* but merely reacted to life's circumstances in ways that seemed best at the time.

According to Bailey (1995, p. 103), "The argument over whether homosexuality is 'biological' or 'freely chosen' is perhaps the most common and the least productive version of the biology debate." The current analysis strongly backs his conclusion because it emphasizes the predominance of idiosyncratic factors over both.

Three Further Implications of Low Genetic Influence of SSA

There are three further implications showing that genetics have a low influence in SSA.

First, as demonstrated from the twin studies, SSA is *unusually* subject to production by erratic nonshared factors. The genetic fraction is much lower than usual compared with those for other traits; for example, a psychological component is typically nearer to but less than 50%. The high level of erratic nonshared factors for SSA is completely contrary to the common but uninformed assumption that SSA is usually produced by some inexorable deterministic process. This is exemplified by the anecdote of the nonconcordant mz twin pair in which the SSA twin insisted SSA must have a genetic origin (Bailey, 1995).

Second, the twin data sum up the influences of all shared and erratic factors, whether presently known or not. After more than seven large studies, the concordance percentages and estimated contributions to genetic, shared, and nonshared influences as estimated by traditional twin-study methods are not likely to change by large factors. Thus it is almost impossible that some genetic or shared social factor—whether already known or to be discovered in the future—will be shown to be a predominant cause of SSA for the general population. As an illustration of this, it is emphasized that all genetic and social factors together produce for male mz twins a low 11% pairwise concordance (Bailey, Dunne, & Martin, 2000). All such shared factors must be individually classified as relatively weak or modest influences, especially if there are more than one. This must be the ultimate conclusion of all the various pieces of research currently being undertaken. All these are interesting, but will not find a predominant cause. We have that conclusion already: nonshared factors predominate.

Third, the low results for the influence attributed to the shared environment leads to the conclusion that parental factors common to each twin are mostly not responsible—in other words, parents are unwise to hold themselves directly responsible for SSA in a

child. Instead, SSA has usually arisen from idiosyncratic immature reactions to factors (including family factors) that are shared by *all* children. The nonshared factors seem to include the child's perceptions of his/her experiences and his/her individual reactions to those experiences. One conclusion of the study of Bell, Weinberg, and Hammersmith (1981) was that 70% of SSA variation was not accounted for by social factors. The present paper concludes that a similar fraction is random factors, but concealing varying reactions to shared social factors.

Otis & Skinner (2004) studied which developmental factors had at least some perceived importance to those with SSA. Most participants were able to endorse at least part of one or more factors from a preselected list, generally of the type used by Bell, Weinberg, and Hammersmith (1981). The survey suggests that many respondents thought multiple causes could be involved.

Modern investigations of genetic contributions to psychological traits show that many genes are involved, each with a very small contribution. It seems likely that the random contributions to SSA will likewise be multitudinous, and that each will have a small effect (although some may have a strong effect in some individuals).

Conclusions

The title of this paper asserted that neither genes nor (deliberate) choice was directly responsible for SSA. Chance incidents and idiosyncratic personal reactions predominate, and this has been demonstrated in almost all the twin surveys that have used modern sampling techniques. The present weak to modest estimates of 22% for men and 33% for women for all shared prenatal biological influences are likely to decrease significantly with further research (see technical appendix). The present fairly strong estimate of 63% for both men and women for the nonshared influences is correspondingly likely to increase with further research. The statement of LeVay (2010) derived from twin studies saying that "genes exert a significant although not all-dominating influence

on sexual orientation" (p. xv) must be interpreted as meaning the lower part of the significance range.

Because of the calculated low genetic fraction, we are safe in saying that people are predominantly not "born that way"; in fact, SSA is a good example of relative lack of prenatal preprogramming. However, the basic attraction constituent of SSA is on average present or absent at age ten, and explanations of origin should concentrate on ages earlier in childhood but post-natal. Some development and sexualization of this attraction certainly occurs at later ages.

There has been, and still is, much research on prenatal causes for SSA. This is reasonable research in its own terms, but according to the twin data in this paper, any findings capable of impacting two identical twins similarly will be numerically minor in terms of total causes. SSA also seems usually not a matter of deliberate choice, because differing reactions to shared environment occur too young; immature reactions are much more erratic and varied than informed adult ones.

Many years ago, Kinsey and associates said that it is possible that the early initiation of sexual experimentation with a same-gender partner is essentially a random event (Kinsey, Reichert, Cauldwell, & Mozes, 1955, as cited in Bickham, O'Keefe, Baker, Berhie, Kommor, & Harper-Dorton, 2007). He may have been right.

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Technical Appendix

This appendix contains discussion of factors that are often highly technical but are an important part of the intellectual debate and will become more so. They tend to argue that the conclusions in the main body of the paper are quite conservative.

Epigenetics

Epigenetics is an emerging research current that describes how the environment influences gene expression. It is simply incorrect that genes control human characteristics in a deterministic, autonomous fashion. This has a potential impact on the twin studies described in the main text. It has been shown that gene expression is only 4% different in mz human twins near birth; more significant divergence emerges later due to epigenetic effects (Fraga, Ballestar, Paz, Ropero, Setien, Ballestar, . . . Esteller, 2005). Differences are more significant by age five (Mill et al., 2006) and continue to increase to old age by a factor of about 4. These epigenetic effects could contribute to different SSA outcomes, but those different SSA outcomes would mainly occur postnatally, when the environment is an actively contributing factor. These differences would decrease any effects of genetic preprogramming.

The Assumptions of Twin Study Analysis

There are rules for twin studies, and violating them leads in almost all cases to a genetic component that is too high. This paper asserts that the rules are often violated in SSA twin studies, resulting in genetic fractions that are significantly overestimated. This

paper also acknowledges that the researchers have checked many of the factors where they can.

For twin studies to be accurate in their conclusions about homosexuality, they must show that:

1. Mz twins did not volunteer for the study at higher rates than dz twins, nor did mz twins show unusual eagerness to answer intimate sexual questionnaires. (This "volunteer error" effect is one of the banes of psychological studies).

2. Families really do treat each of a pair of twins identically (the "shared environments" or "equal environments" assumption).

3. Homosexuality has a statistically "normal" distribution (bell curve) in the population.

4. There is no interaction between genes and environment.

5. People with the "homosexual gene" very rarely mate with others carrying the "homosexual gene."

6. The twins do not imitate each other; in particular, identical twins do not encourage each other to be homosexual (the "twin environment" effect).

7. Apart from being twins, the twins are very similar to the rest of the population (in other words, they are physically the same and about 1% are exclusively homosexual).

8. The calculations should give genetically consistent results when siblings, parents, and other relatives are included in the calculation model.

9. Whether an mz twin has an independent placenta or shares it with the co-twin makes no difference to the results.

Are These Rules Broken?

1. The volunteer error has been minimized (but not completely removed) by using modern twin registers. Another type of distortion can occur when twins refuse to take part

in the SSA section of a survey. Such twins tend to be more conservative, and less probably homosexual. Each of these factors tends to overestimate apparent genetic content.

2. Families treat each individual twin the same. Care given by parents amounts to a strongly individualized care that can be experienced as quite different from that given to a brother or sister. This could lead to differing SSA. But if in fact idiosyncratic reactions of children predominate anyway, this may not be a very important factor.

3. Normal distributions are respected. SSA certainly does not have a normal distribution in the population, and that is what causes the large error ranges. The endpoint of these particular mathematical distortions is to produce a "genetic" contribution result that is too high. Santtila and colleagues tried to allow for this mathematically, and presented evidence they had succeeded (Santtila, Sandnabba, Harlaar, Varjonen, Alanko, & von der Pahlen, 2008). But the calculated genetic fractions were much the same as those from other authors who did not allow for it. Either this is a small effect, or the results are dominated by other uncertainties.

4. There is a proper perspective on nature and nurture. Probably the most important criticism that has been leveled at twin studies is that they treat nature and nurture as totally separate influences that don't interact during human development. Is there an interaction between influences produced by genes and the environment? Of course there is. If interaction does occur between the influences of the genes and the environment among any population in a twin study, it has the effect of artificially raising the calculated genetic contribution (Eaves, Last, Young, & Martin, 1978; Eaves, Eysenck, & Martin, 1989; Lathrope, Lalouel, & Jacquard, 1984). Researchers are generally very critical of the idea that nature and nurture do not interact: "In a specific practical situation do we really believe . . . the . . . model is at all realistic? The answer is no" (Goodall, 1990, p.133). So, these interactions certainly exist. Let's look at an example. If a person were genetically inclined to become homosexual, would an environment that encouraged him to express his sexuality (for example, seeing homosexual porn or receiving advances

from homosexual men) have any effect on him? Of course it would. There is definitely interaction of the genes and environment. However, it may not always affect the results too seriously. In the Australian SSA study (Kirk, Martin, & Bailey, 2000) researchers tested for this specifically and couldn't find clear evidence of it—only a strong suspicion. It is also fair to say that when gene-environment interaction has been obvious in non-SSA studies, it has had only has a minor effect (Eaves, Last, Young, & Martin, 1978; Eaves, Eysenck, & Martin,1989). The possibility is disturbing, though, because it is an effect that is easily missed.

5. Do people with the "homosexual gene" or genes tend to marry each other more frequently than they marry those without the gene or genes? There are no such clearly known genes (Mustanski, DuPree, Nievergelt, Bocklandt, Schork, & Hamer, 2005), and this is therefore unlikely to be important. But if this effect existed, it would have an effect opposite to all the other factors mentioned here—it would underestimate the contributions from genes (Waller, Kojetin, Bouchard, Lykken, & Tellegen, 1990).

6. Do twins tend to imitate each other in homosexual development? Twins certainly do imitate each other (for example, they are known to imitate each other in antisocial behavior and in truthfulness or lying—Eaves, Last, Young, & Martin, 1978). It is quite conceivable that the same might happen in the development of homosexuality. Twins often have an unusually close bond, sharing intimately and reinforcing each other, particularly if they are identical. These environmental factors could lead to higher levels of homosexuality in identical twins, making the genetic content appear higher. Influence on each other could range from talking about SSA with each other to exploratory sex. Hershberger (1997) found statistical evidence in his sample that the mz twins had indeed influenced each other in the occurrence of their SSA.

7. It is quite doubtful that twins are completely similar to the general population. On average they start life smaller than other babies, and they have fewer verbal and social skills until as late as eight years (Powers & Kiely, 1994). The rate of child abuse among

twins is nearly three times higher than for the general population (Nelson & Martin, 1985). They tend to be found toward the bottom of the social scale in their schools and are often subject to harassment and teasing by schoolmates. Young male twins are often called "fairies" (Winestone, 1976), probably adding to self-perceptions of childhood gender nonconformity, which is one of the strongest predictors of later homosexuality. Twins are such good and sufficient friends to each other that their individuality and sexuality may not be entirely developed for social rather than hormonal reasons. For example, some studies have shown they are more likely to be unmarried than nontwins, though this effect was not found in the Australian twin study. The overall rate of self-evaluated SSA among twins was 3.1%, rather higher than 1.8% for an independent survey of prevalence in the general Australian population (Bailey, Kirk, Zhu, Dunne, & Martin, 2000). Other surveys also suggest SSA might be slightly higher for twins than for the general population. In summary, they are a somewhat suspect population for sexual surveys for a variety of reasons.

8. Inclusion of siblings has given contradictory results in SSA twin studies, worse than for other traits. For their research, Santtila and colleagues state that "attempts at fitting univariate and bivariate extended-family scripts for categorical data were not successful" (Santtila, Sandnabba, Harlaar, Varjonen, Alanko, & von der Pahlen, 2008, p.103). In other words, including nontwin siblings in a modified twin research design gave results that could not be interpreted or that were perhaps contradictory. Kendler and colleagues found a decrease in the genetic fraction, when siblings were included in the model (Kendler, Thornton, Gilman, & Kessler, 2000), and Hershberger (1997) could find no resulting genetic influence at all. This argues there may be a basic problem with the conventional genetic model used in all these SSA twin studies, and further investigation is necessary. This tends to point to a sibling social, rather than genetic, effect.

9. In traditional twin studies, the genetic fraction is probably further overestimated because of an effect of placenta on gene expression (whether mz twins have a shared

placenta in the womb or whether they had separate placentas, as dz twins do). Mz twins can have either, and it makes a difference. In a groundbreaking paper, Kaminsky and colleagues studied about 6,000 differences in gene expression between twins (Kaminsky, Tang, Wang, Ptak, Oh, Wong, . . . Petronis, 2009). Twins that shared a placenta were much more alike than expected. This is because the same blood supply ran through both twins and cells from each twin ended up in the other. Shared placentas occur in perhaps 25% of mz twins. Combining both kinds of mz twins, the average is still more similar than genetic theory says it should ideally be for the mz twins. The effect of this is to artificially increase the difference between mz and dz twin concordance on which the traditional twin method depends and hence overestimate the genetic fraction. Assuming an allowance needs to be made for the mz placenta effect, the average effect for all traits of not factoring it in would be that the genetic fraction is 15% too high (Kaminsky, Tang, Wang, Ptak, Oh, Wong, . . . Petronis, 2009)—quite a significant effect. Does this apply to SSA also?

It was already known in some of the above-cited SSA twin studies that mz twins were more alike than conventional genetic theory should allow. This could mean that overestimation of the genetic fraction due to mz excess similarity also applies to SSA. It has been theorized that this has been due to nonadditive genetic effects (Kirk, Bailey, Dunne, & Martin, 2000), but a simpler explanation is the placenta effect.

Allowing for this likely 15% overestimation would reduce the weak SSA 22% genetic contribution to 7% in men—with an error of zero—and from 33% to 18% in women. The genetic effect for the women would then also be classified as weak rather than modest.

Consequences of Assumption Violation

In most twin studies of homosexual populations, most of the assumptions that must be met if the results are to be trusted have been partly violated in such a

way that the genetic contribution is significantly overexaggerated. This also implies that the nonshared environmental fraction has been underestimated, if as usual the apparent shared environmental fraction seems to be low. Does this mean that twin studies are a completely unsuitable tool for gauging the genetic content of sexual orientation? Probably not. When other traits have been investigated closely for the effects of violated assumptions, the genetic proportion is over-estimated but is still real. So while it is a reasonable supposition as shown above that the 22% estimated figure for the genetic component of male SSA could be consistent with zero, the genetic influence for female SSA is more likely to survive further testing but is likely to be less than 20%.

In summarizing all of this, one may estimate that the genetic contribution to SSA as shown by twin studies is presently weak for men and modest for women, but may well downgrade ultimately to zero for men and weak for women. One can also estimate that the nonshared environment fraction will correspondingly increase.

Poor Penetrance?

Penetrance means that the genes for a trait may be present but do not exert their effects. So there could be genes for SSA that are, for unknown reasons, partly inactivated. This was raised as a possibility by Bailey, Dunne, and Martin (2000). Penetrance is usually judged by effects on relatives, particularly twin pairs. The pairwise identical twin concordance for SSA in men and women is typically 11 and 14% respectively (Bailey, Dunne, & Martin, 2000), meaning that identical twins are strongly discordant for SSA—they usually differ, and this might indicate poor genetic penetrance. We now examine the possibility, based on the interpretation of twin studies, that genetic factors are present but "non-penetrant" (Kastern & Kryspin-Sorensen, 1988) and produce this low concordance. The hypothesis would suggest that although responsible genes for SSA are normally present, their effects are for some reason not

being produced consistently in the nonconcordant twin. This would give rise to a low calculated genetic fraction.

This is unlikely. The huge database Omim (www.ncbi.nlm.nih.gov/omim)**,** which contains data on genetic variations and mutations in man, contains most of the quantitative data on typical penetrance. A penetrance as low as observed in the mz twins for SSA occurred in only 9% of cases.

A study on 6,578 human genes—nearly 30% of the total genome (York, Miles, Kendler, Jackson-Cook, Bowman, & Eaves, 2005)—showed that the expression of genes was 11.2 times more correlated in mz twins than dz twins. A much less extreme value from a much larger sample was found by Kaminsky and colleagues, but the correlation was in the same direction—positive (Kaminsky, Tang, Wang, Ptak, Oh, Wong, . . . Petronis, 2000). This means that discordant effects in mz twins are the exception rather than the rule. To observe strong discordance for SSA in both male and female mz twins seems even more unlikely.

We cannot meaningfully talk about penetrance until the genes are specified. This is because unless the genes are well-known, the effect of low-penetrance mechanisms cannot be differentiated from their absence, and other causes might instead be responsible. In the present case, about two decades of research on SSA-genetic association has found only genes that are individually not statistically significant (Mustanski, DuPree, Nievergelt, Bocklandt, Schork, & Hamer, 2005). More research is proceeding, but even if it yields positive findings it will need much replication and confirmation, particularly because of the previous conflicting findings in this field. It is likely, as for most traits, that SSA will be multigene, and such genes will eventually be found—but each will have a very weak and indirect influence by itself and individual confirmation will be quite difficult. Even if the influence of such genes is unequivocally established, it would be a further step to establish in this particular case the mechanism that produces poor concordance. At present, therefore, the idea of poor gene penetrance

is far too speculative and awaits much more research progress. The best conclusion right now is that unusually poor penetrance in the classic sense is not a factor.

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