

Twin Studies: the strongest evidence

Over the last decade, studies of twins have provided some of the strongest evidence that “our genes do not make us do it” - which makes this chapter probably the most important in the book. Results from twin studies are so far-reaching that they supersede many other studies we’ve mentioned so far.

In a nutshell, if you take pairs of identical twins in which one twin is homosexual, the identical co-twin is usually *not* homosexual. That means, given that identical twins are always genetically identical, homosexuality cannot be genetically dictated. No-one is born gay. The things that create homosexuality in one identical twin and not in the other have to be post-birth factors. Hold on to this simple thought as you navigate the complex world of twin studies in the pages of this chapter.

There is also good news in here for parents who hold themselves responsible for homosexuality in their children.

A note about the organisation of this chapter. The first section deals with homosexual concordance in identical twins, because it illustrates so much. The second section deals with the more conventional twin studies and their limitations. Readers may pass over the second section if it seems too technical and move on to the summary. We'll let you know when!

Twins have been invaluable to medical research for a long time, though sometimes in appalling ways. Twin study research probably reached its nadir during the Second World War, when Dr Josef Mengele, a brutal researcher at the Auschwitz concentration camp, deliberately sought out identical twins for experiments. Sometimes he would kill one twin by poisonous injection, dissect the twin to see the effects, then immediately kill the co-twin to see the differences.

The founders of twin studies were very frequently involved in the racial theories of the Third Reich, and actively suggested killing off defective humans. By contrast, the modern use of twin studies is overwhelmingly to help and heal. By the end of 2007 about 30,000 scientific papers in medical databases mentioned twins at a rate of a few thousand papers a year.

Traditionally, twin studies first compare identical twins to gauge the effects of genetics (a high similarity probably means high genetic influence), and then, if possible, add in non-identical twins, to give extra information about the relative importance of upbringing. *

Twin registers are the foundation of modern twin studies. They are now very large, and exist in many countries. A gigantic European one with a projected 600,000 members is being organised, but one of the largest in use is in Australia⁶¹, with more than 25,000 twins on the books. If we can look through a register of identical twins and find pairs in which one twin has SSA, we can then see if the other twin also has SSA, in other words what the degree of “pairwise” concordance is. Readers should note that sometimes a different measure is popularly used without explanation, called the “probandwise concordance”. This is a much less intuitive measure, needed for further twin studies calculations. It is often much higher than the

* We will follow that order in this chapter, but emphasise the identical twins because of the mathematical difficulties which arise when the non-identical twins are added in – making the results unusually difficult to interpret for SSA compared with other traits.

pairwise concordance and when used without explanation, can give the impression that genetic influence is much higher than it actually is. The pairwise concordances this chapter is focusing on are probably lower than anything you may have seen elsewhere. However, they answer the simple question – if one twin of an identical pair is SSA, what percentage of the co-twins are also SSA?

Jones and Yarhouse⁶⁵ mention pairwise concordance calculation for the important Bailey et al. (2000)⁶⁰ paper in which SSA was measured using one facet of an old Kinsey measure - attraction and fantasies combined. Jones and Yarhouse find that both for self-declared lesbians and gays the concordance is 14% and 11% respectively. Anyone can verify this in the original paper. The pattern looks like this for males and similar for females:



Figure 17

This means that for every nine sets of male twins, one of whom is homosexual, the other is homosexual only one time in nine, or 11% of the time, which is not very much. For this measure of SSA (attraction and fantasy) *identical twins usually differ*.

What 11% concordance means

At this point we probably need to re-emphasise what this means. It does *not* mean that 11% of identical twins have SSA. Numerous studies of western populations (Chapter 2) have shown that homosexuality is present in something between 2-3% of people, and this, of course, includes twins, eg. Figure 17 shows 100 hypothetical twin pairs taken from a twin registry. Of those 200 individuals only 4 (roughly 2-3% of them) [shown by the gray squares] have SSA. There are not enough pairs to show the rarer pairs *both* of whom have SSA and are concordant.

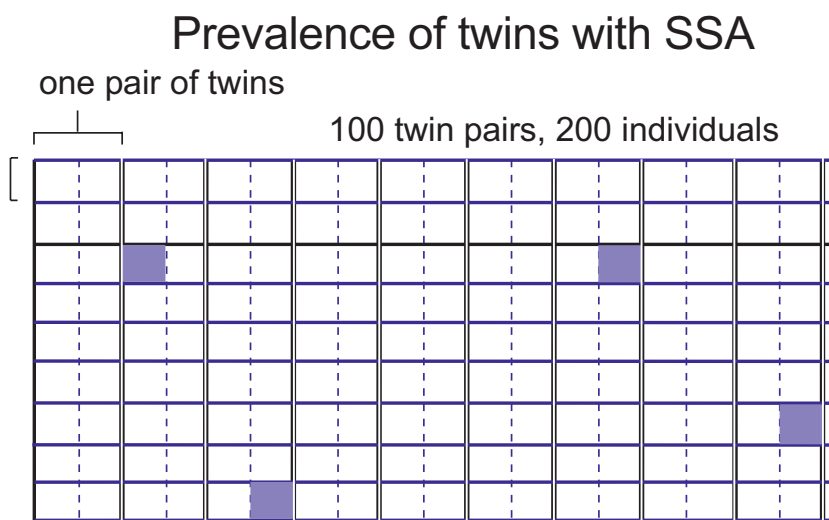


Figure 18

Nor does 11% concordance mean for any concordant pair, that they only, and none of the other twin pairs, are affected by genetic factors. All the twins (and everybody else in the world for that matter) are *equally affected by genetic effects, and environmental effects and chance*. Nor does 11% concordance mean that homosexuality is genetically inevitable for 11% of the homosexual population.

Eleven per cent concordance simply shows that when one of any number of twins from a general twin registry is homosexual, his co-twin is homosexual one time in nine, or 11% of the time.

It's also important to emphasise that genetic effects are not the only thing this degree of concordance shows. In twin studies "genetic" factors is a kind of catch-all for any *biological* common factors, eg things like womb environment before birth. But concordance is also a catch-all for common *environmental* factors. Identical twins not only have identical genes, but they (usually) grow up in the same family environment. So Figure 16 illustrates the *combined* effects of a common genetic inheritance *and* a common home environment. This is rather ambiguous but we shall see it still gives crucial information in the case of SSA. At only 11% concordance we have to say that for SSA, at first sight, it seems that neither upbringing nor genes is very important. Put another way, in homosexuality the practical effect of genes, other common pre-natal biological factors, and a shared home environment, is *weak*. But more about the common social environment later.

We also want to emphasise that the use of twin concordances we are making here is for illustration not for proof. To get a more accurate picture you also need to consider how often SSA occurs and/or what the concordance is for fraternal twins. However the fundamental point remains true when these are looked at: the largest single cause of SSA is random factors (meaning, factors affecting one of the twins and not the other), as we shall see.

We can now make our most important point:

Those with SSA are not born that way.

If common factors like genetics or womb environment predominantly cause SSA, then identical twins will *always* be identical for SSA – ie the SSA concordance would be 100%. But they are not, so post-natal random factors are causing SSA and making the difference. We could also sum up Figure 16 by saying that for SSA, *genes create a tendency, not a tyranny*. Even the tendency is weak. This is a critically important principle.

Twin studies cover every possible pre-natal biological influence - known and unknown

Our second important point is that because "*genes*" in twin studies include everything from the shared pre-birth biological environment, we already have the final answer about genetic influence in Figure 16, no matter how many details have yet to be scientifically "discovered". Twin studies sum up all the details, those already known to researchers, and those still unknown. And many more details will be discovered as more scientific papers emerge, usually announcing a new factor each year. It is remarkable, but twin studies summarise all the effects on a developing embryo that will ever be discovered, all the common factors. And, to repeat: at 11% the combined genetic effects are weak for SSA.

If this degree of concordance gains support (as generally anticipated) beyond the five research papers already consistent with it then the following conclusion will not change in the future either:

The predominant cause of SSA both in men and women is individual post-natal random reactions to biological and environmental factors.

However, even among academics this has not really been an important factor in thinking about the subject because research has tended to concentrate on the question, "Is it genetic?" and this has resulted in some problems of interpretation, even quite recently.

For example, the book, *Born Gay*⁴, uses twin studies (predominantly) to argue that 30% of SSA is due to genes and the remaining 70% to abnormal hormonal exposure in the womb. You may at once see the problem with that argument. If twins share the same womb environment then any hormonal effect on one twin would be experienced by the other – in other words, the effect is part of the *common* biological environment.

If, as the authors of *Born Gay* suggest, one of the twins is exposed to an extreme and unlikely hormonal influence causing homosexuality, then *both* twins should be homosexual. However, twin studies show they are not – in 89% of cases (Figure 17). Unusually high or low hormone levels are part of the shared pre-birth biological environment.

It makes no sense to say that abnormal hormone exposure accounts for 70% of homosexuality. As a shared effect, hormonal exposure belongs within the authors' 30% figure for the genetic influence – and is therefore weak.

In the next diagram we compare the degree of pairwise concordance for SSA with pairwise concordance for other human conditions. We concentrate on low concordance conditions. On the left is lung cancer. If one identical twin has it, the other almost always does not. This means that neither common environment nor genetics is responsible for lung cancer, but *chance* or *random* factors.

Low Pairwise Mz Twin Concordances

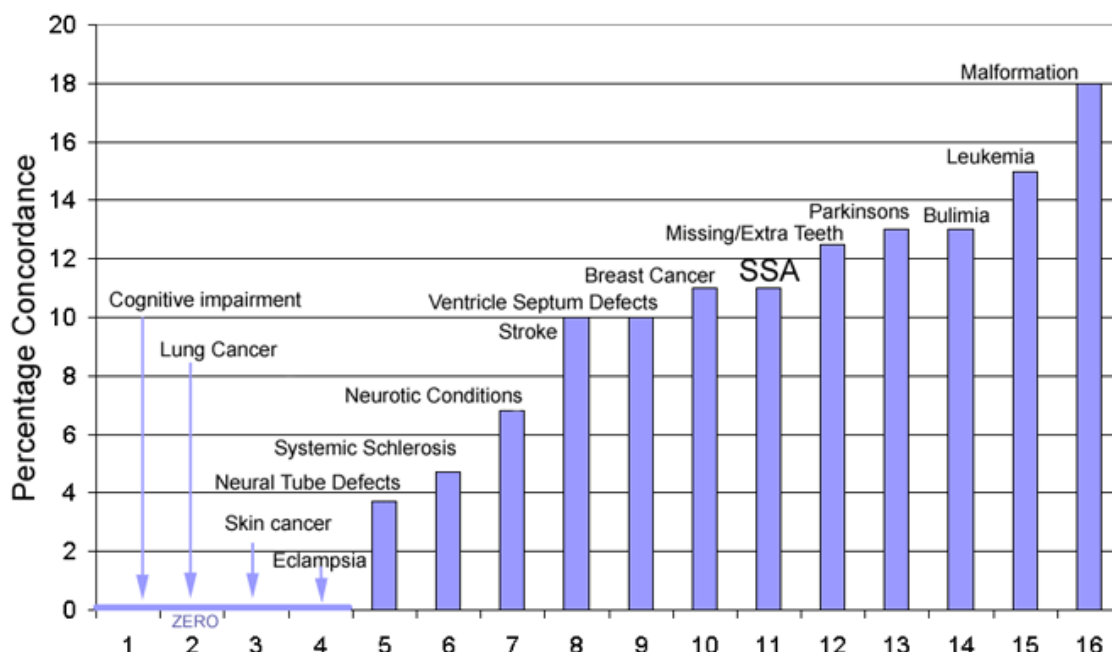


Figure 19. Low pairwise concordances for monozygotic twins. Taken from PubMed.

1. Cognitive impairment⁵ 2. Lung Cancer⁶ 3. Skin cancer⁹ 4. Eclampsia¹⁴ 5. Neural tube defects¹⁹ 6. Systemic sclerosis²⁰ 7. Neurotic conditions²¹ 8. Stroke²² 9. Ventricle septum defects²⁴ 10. Breast Cancer²⁵ 11. SSA⁶⁰ 12. Missing/extra teeth²⁶ 13. Parkinson's²⁷ 14. Bulimia²⁸ 15. Leukemia²⁹ 16. Malformation³⁰

SSA is dominated by chance, to a high degree. Few other conditions are so discordant except the cancers and stroke.

This conclusion should be spelt out again in a slightly different form from the way we did it above:

The largest factor in twin studies of SSA is non-common influences

Poor gene penetrance is a red herring

Another argument sometimes used in the genetic debate around SSA is that there is a gene which is important and responsible for the trait but its penetrance is poor. By this it is meant that an SSA gene is present but for unknown reasons probably concerned with the cell biochemistry, does not exert its effects. Low penetrance indeed exists; however classical twin studies cannot possibly untangle it from other chance effects in the environment. Rather, what must be done is to first find a gene for a trait and then show it is switched off in some conditions. From Chapter 9 we know no such gene has been found. We cannot

therefore just look at the low pairwise concordances and say a gene with low penetrance is responsible. Rather, following our present knowledge, we must say the effects of genes and common environment are very low, and chance dominates.

In addition, the norm is that low penetrance is unusual. And the concordance for monozygotic twins (identical twins) with SSA is so low that if poor penetrance is responsible it is unusually poor penetrance. So an argument that low penetrance is happening in this case is very unlikely indeed. The poor penetrance argument involves 3 layers of speculation: one, that SSA genes exist; two, that they are not exerting their effect consistently; three, that the low penetrance effect is unusually extreme for some hypothetical reason.

In contrast, left-handedness has a 13.5% concordance which is almost identical to that of SSA but the connection to known genes is well established.

One thing seems clear: any genetic contribution from an SSA gene is much less than in most traits for which genetic influence has been measured. SSA is 90% chance. SSA is in fact a good example of *not* being “born that way”!

One of the four papers giving a low level of concordance is by Bearman and Brueckner³ who in 2002 described part of a large ongoing study of adolescents in the USA, with tens of thousands in the initial sample. From this school-based sample they chose a large number of twins and other relations for genetic studies. This has advantages even over the twin registers – the twin register is voluntary, but the sample here was mostly not. However their final twin sample was smaller than the Australian set.

The results were that the SSA concordance between identical twins was 7.7% for males and 5.3% for females – lower than the 11% and 14% in the Australian study by Bailey *et al.* (2000). However these were adolescents, not adults, and we might be excused for thinking adolescents are peculiar in some way!

Three other studies support the above twin concordance; none show it is erroneous. (Earlier, higher concordances are universally acknowledged to have resulted from an unusual degree of “volunteer error”, and to be on a “probandwise” basis (see pp 1-2). It is also salutary to note that over the past decades, the better randomised the sample, the less the resulting concordance! In other words volunteer bias greatly exaggerated these early results, which are unfortunately still quoted widely.

Another interesting point is that SSA concordance for *both* men and women is unusually low. It would have been easy to imagine a strong genetic factor for men and not women or vice versa. But they are both low, and for both sexes: predominantly individual reactions to almost anything (something we are calling the random/chance/ or accident factor).

Effect of family environment

A second feature of these pairwise identical twin study data is the apparent minor effect of family environment (upbringing) on the development of SSA, because both prenatal effects and common family environment added together only produce an 11% pairwise concordance. Fuller studies, which include non-identical twins, usually also show that for SSA the effect of upbringing is low and even less than that of genes. Parents take note: according to twin studies of SSA, *you are usually not directly involved in making a son or daughter gay.*

Those familiar with classical twin study results will immediately notice that this is a common pattern. Calculations from twin studies for many, many traits usually show family influence is less than genetic influence. However the lack of family effects is a controversial issue. Developmental psychologists just can't believe it - they notice many and obvious effects of the family environment in their studies. But the twin studies experts have stuck to their statistical conclusions, and argue that the third factor, randomness, is even more important than genes or family. We agree, but will later explain why family effects *seem* so small.

It is indeed true that there is a lot of evidence (hetero)sexual orientation is usually developed first in the family through imitating the gender patterns of the same-sex parent and siblings, though soon the peer environment becomes much more important. But we want to say that random/chance factors affect this learning. Perhaps we should note here that Robert Plomin, whom we met in earlier chapters, has devoted some decades of his career to finding what produces differences in family members and also twins. One important conclusion he has come to is that genes tend to make people more similar, and environmental factors tend to make them differ more.⁸⁶

What might random environmental factors be?

Random factors could include: perhaps the sexual abuse of one twin but not the co-twin; perhaps one twin is suddenly exposed to gay pornography and develops a habit, but his co-twin does not; maybe one twin misunderstands his longing to be like confident, popular boys and wonders if he is gay; perhaps one is persistently unlucky with girls, unlike his brother, and seriously questions whether he may be gay. Lastly there are differing influences from friends and experiences that are not common to the twin pair.

Twin study researchers say it is random factors (chance occurrences), which predominate in the development not just of homosexuality but many other traits. Frequently they say they are unable to find the effect of shared environment (upbringing) and at most grant it a low influence. From the point of view of twin studies, if the question is asked: is SSA mostly nature or nurture? the answer would be, *Neither, it is mostly accidental events*. This is an unexpected and probably unwelcome answer to the decades old nature/nurture argument!

The apparent importance of randomness seems at odds with many of the other studies in this book. There seems plenty of evidence that parents and family-life can have a huge effect on the development of SSA. What's going on?

The answer is: both can be true!

More about the (extra-)ordinary factor, the chance or random event

Our brains have a way of filtering out the routine and remembering the unusual. For example, we don't remember every cup of coffee we've had; we edit out the vast majority and remember only the unusually bad or unusually good. What is rare or unusual stays in our mind. (This is probably the reason why people persistently bet on dark horses, an irrational behaviour which has puzzled psychologists, but been exploited by bookies for centuries). Similarly the routine years of good care that children receive from parents fade into the background and tend to be overridden by a few unusual events in the family which can be very good, or very bad.

Some of the extremely unusual, or rare, incidents are therefore extraordinarily powerful influences. This includes early sexual experience. Sexual activity is not usually observed by children round the home – so pornographic images falling into the hands of an insecure but hormonal adolescent can burn themselves in the memory and be an influential factor for many years. The unusual random event impresses itself on our memory, and begins to have a disproportionate influence on us. Examples of other powerful unusual factors are given later in this chapter.

Different perceptions of the common environment

This probably explains the apparently minor effect of common environment in twin studies. The *perception* of the common family environment and upbringing can be quite different in the eyes of different members of a family. Bailey conducted interviews with a number of identical twins discordant for SSA. (ie one had SSA the other didn't). He found they had perceived the same family environment differently. These different perceptions show up in the data as random occurrences and they are. But what they also are, are *different reactions to the same environment*. They often represent the reactions of a twin, who can, for example, mistakenly take it into his head that his parents don't like him nearly as much as his brother. Parents often can give a wrong impression to an immature mind, and no-one is really to blame. Nor has the child chosen this perception. It just happened, though it may be quite inaccurate. Virtually all researchers would agree that choice has not been a significant factor in the development of SSA.

Twins can react differently. This is a good picture for us of how children who are not twins can also react very differently in the same circumstances.

Many gay men and women in the process of recalling incidents which they believe were instrumental in the development of their SSA will recount clear early memories of things done and said in families, which engraved themselves into memory and deeply influenced their later choices.

A slightly unusual bodily form or defect may sometimes be taken obsessively to heart by one child, but not another. This will also masquerade as something random.

This means that:

upbringing and common environment are showing up after all – but heavily disguised as random factors. Put another way, the random category in twin studies carries within it a significant influence of upbringing and family environment.

So, the random contribution found within twin studies is too high - inflated by upbringing and common environment while appearing to exclude them. Much more research is needed to detail the important factors since it is these random, yet often environmental events, which are predominantly responsible for SSA.

What are the implications for parents in all this? Children are children, and immature. They need to be checked from time to time. Have they really perceived an important event in the family accurately? What has really been the influence of some unusual event? Do you need to diminish its importance or explain it for them?

Perhaps your son or daughter is gay. Probably it has had nothing to do with you. But you may have been faced with unjust accusations about their upbringing when in fact your real motives were quite different. Take your opportunities to show what your real feelings were, in a way that will convince them.

Perhaps in one of the rarer cases you really were distant from the child, and this led to deeper damage than you appreciated. However you may now have the advantage of knowing the true situation and working to make amends. A long and hard task, but possible.

Factors known to be important to at least some people with SSA

Otis and Skinner⁸⁷ as shown in the following diagram found factors endorsed by their group of SSA people which had at least some influence in the development of their SSA.

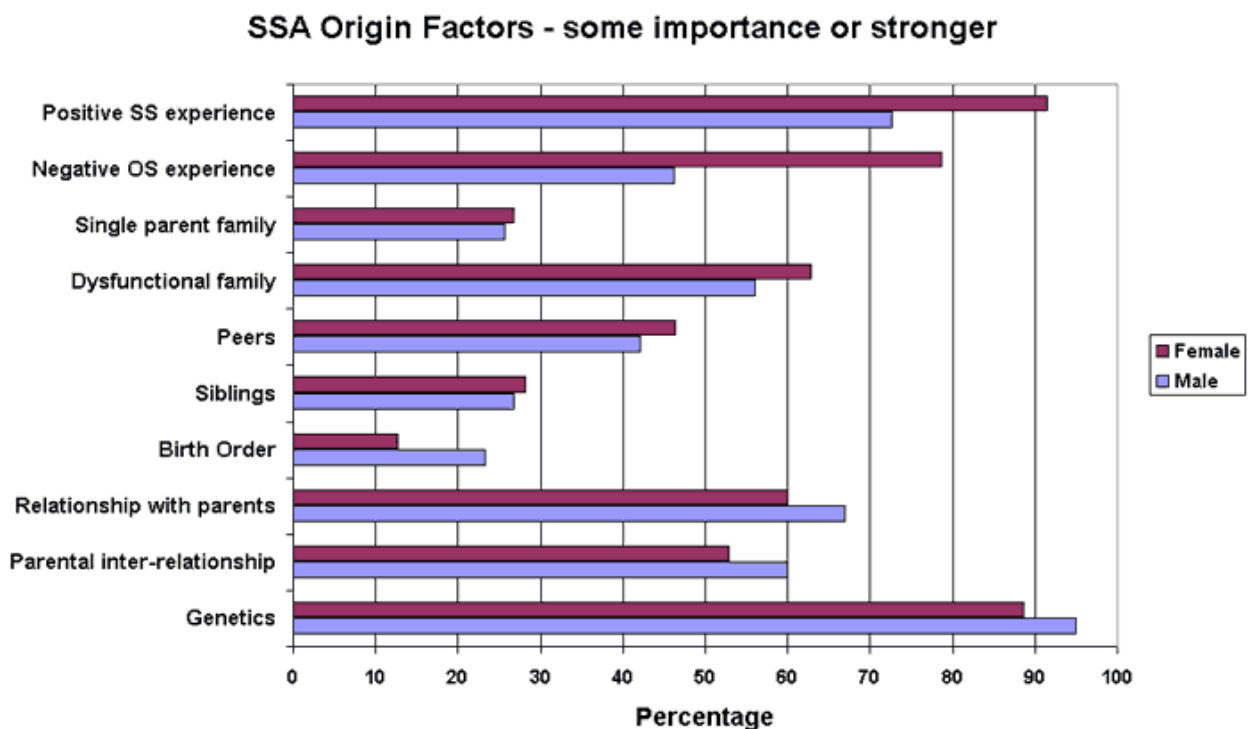


Figure 20. Factors thought by gay and lesbian people to have at least some importance in the origin of their SSA.

This diagram shows the inaccuracy of the popular impression that SSA development is a vast mystery. Most people with SSA can point to several factors which were of some importance.

Of course no-one directly experienced genetics as an important factor! But perhaps some thought that features of their body were important, and perhaps they obsessed about them. Many of us do! But in most cases we can guess that they heard about the influence of genetics from the media, and from gay sources and chose that as one of the explanations. (The numbers of SSA people believing homosexuality is genetic went up 400% between the 1940s and 1990s.)

The diagram shows that many factors were important. Calculation shows that most people must have ticked a number of factors. It thus seems *even for an individual that multiple factors are involved*. This reflects the mainstream view as expressed by Sir Michael Rutter: "The great bulk of psychological traits and of mental disorders is multifactorial in origin."⁵⁰

We now give an even more extensive list of things that people may react to, and, in so doing, become SSA if other factors are in place. These reactions are the "chance" factors we have been talking about. We have drawn them from personal accounts we have received, and from the literature. Most people with SSA will say "Most of these factors were totally irrelevant to me, but a few were important". The important ones will differ from person to person. In no case will such a factor be important to a majority. Some are reactions to body features, hence "genetic". Others are much more environmental, and include chance meetings and individual reactions. Some may appear improbable on first reading, but all have been important to *someone*. A few of the genetic ones are obviously not from personal experience, but only are alleged by various people. All factors in the lists have the potential to be reacted to differently by different people. Some factors are in both lists.

Some SSA people will identify with nothing on the list, and we would suggest to them they think about the events or feelings which did seem important. We suggest that if you have mulled these things over more than a hundred times, they are important and formative to you and may even be a mind habit.

Reactions to factors of predominantly genetic origin:

Polycystic ovaries

Obesity (in women)

Poor coordination (in men, particularly in sports, the converse for women)

Unattractive/ "unfeminine" physical features (in women)

Physical handicap (can include deafness, other handicaps)

Retiring temperament/tomboy temperament (men/women respectively)

Artistic predisposition (men, particularly if allied to poor sports coordination)

Left handedness (both sexes. Included because probably 60-70% "genetic")

Intersex conditions (this is a special case, because gender identity does not correspond to chromosomal identity. The vast majority of SSA people are not intersex.)

Visio-spatial defects/aptitude (in men or women respectively)

Novelty seeking (in so far as this is genetic, it can lead to trying many unusual sexual experiences)

Fluctuating asymmetry (can overlap left-handedness and irregular physical features)

X-chromosome inactivation (if atypical and extreme)

Inherent gender atypicality (genetic influence is generally found to be high for this feature)

Older brothers (men)

(15 factors)

Reactions to factors of predominantly environmental origin

Polycystic ovaries

Obesity (women)

Sexual abuse (same-sex for males, and opposite sex for women)

Gay pornography(mostly men)

Older brothers(men, included here because the physiological argument is highly speculative)

Reactions to parents (no identification with same-sex parent: sometimes misperceptions)

Sexual experimentation (particularly, prolonged with same sex)

School peer pressure (defensive withdrawal, mainly by males)
Bullying (mainly males)
Divorce (impacting perceptions of sexual adequacy in both sexes)
Verbal abuse (particularly about gender atypicality)
Discrimination (mainly reinforcing a position already adopted)
Parental negative messages (about gender conformity)
Parental encouragement to be gender atypical (often by default for amusement)
Poor social skills (more important for males)
Bad luck in love (leading to self-questioning)
Liberal cultural environment (encouraging experimentation)
Rebelliousness (a rejection of same-sex stereotypes)
Political climate (lesbian/feminist solidarity)
Fashion (extreme concentration on aesthetic values – men)
Soul mate (quest for deep intimacy – women)
Easier sex (men – less commitment required for same sex)
Maternal stress (daughters affected only)
Prescriptive cultural environment (many anthropological examples)
Middle age (women, may coincide with family leaving home)
Gay culture attraction (coincidence of values)
Habit (repeated pattern of relationship sometimes with early origins in family)
Sensual factors (seeking repetition of pleasure)
Urban environment (anonymity perhaps a factor.⁸⁵ (Laumann *et al.* 1994)
Slimming pills (Taken by mothers during pregnancy and affecting daughters)
Marriage resistance (lesbian)
Resistance to categorisation (women, leading to predominant reaction to whatever is the prevailing environment)
Gay social pressure (mainly on bisexuals – to be either gay or straight)
Envy (of attractive same-sex attributes)
Adoption (possible disturbance of modelling)
Alleviation of depression (different orientation depending on affect)
Feelings of rejection
Exercise of power (men particularly historically)
Single parent family (fewer role models)
Fetishes
Passivity
OSA intimacy problems (overlaps poor social skills)
Shyness (similar to poor social skills)
Dreams (particularly sexual, leading to questioning of orientation)
Chance encounter (with an attractive same sex partner)
Bad opposite sex experience
 (46 factors)

An interesting thing about the two lists, is that the “environmental” one is three times as long, and that is simply because there are more influences and events in a lifetime than in one limited body! That suggests that although dominant environmental causes for SSA do not exist, they may together comprise the majority of factors which are important to people. Of course you may have a different list. See if you can make one with more “genetic” factors in it. We suspect it is difficult. *The genetic list also shows that genetic effects themselves are very indirect. What is important is the individual cognitive/emotional reaction to the genetic trait.*

It's now time to look at the rather more murky traditional twin studies. If you want to skip this material, [click here](#) to go directly to the chapter summary at the end. Congratulations on getting this far!

Identical and non-identical twins

The traditional studies use both kinds of twins, trying to disentangle the relative contributions of genetics, common environmental experiences, and non-common experiences.

There were about a dozen twin studies of homosexuality up until 2007, of which the last few are the most important. Earlier studies suffered severely from acknowledged volunteer errors – twins who were similar in sexual orientation tended to preferentially volunteer, and this distorted the results. Finally large studies were done using the twin registers. These should greatly diminish the problem, but not eliminate it, because even on a register, twins have to agree to take part in a given study. Perhaps again, those twins who were both SSA agreed too frequently. No-one really knows.

Even these studies, the best to date, encountered another problem: the unusual mathematical form of the occurrence of SSA. Basically, in these studies as in the general population, there were lots of heterosexuals, relatively few bisexuals and rather more homosexuals than bisexuals. Among women there were relatively more bisexuals and the mathematical problems were not so great. But the fundamental mathematical twin study pre-condition of sampling a group with a normal distribution in the population (represented by a bell curve) was strongly violated, with the result that the errors on the estimate of the genetic content were *much* larger than usually found in twin studies of other traits. This makes the studies which use identical and non-identical twins together, more difficult to interpret than twin studies on other traits, as seen in the following tables.

Table 1. Genetic contribution for male same-sex attraction. In this rather unusual comparison we give the results for three facets of SSA, attraction, sexual orientation and activity separately as found in different studies, all using twin registries or similar. The tables give the calculated “genetic” or common influences as a percentage and the usual 95% error range in brackets. For Hershberger no error range was given but is calculated from data in his paper, for Bearman and Brueckner an error range is estimated by us. The latter was adolescents rather than adults.

Factor	Bailey et al. ⁶⁰	Hershberger ⁵⁹	Bearman and Brueckner ³	Santtila et al. ⁸⁸
Attraction to men	0.45% (0 – 71%)	31% (0-60%)	0.0% (0-70%)	
Self-reported sexual orientation	0.0% (0 – 54%)	0.0%		
Frequency of male partners	19% (0 – 61%)	0.0%		27% (2.7-38%)

The three factors are traditional ones (due to Kinsey) and have proven reliable estimators in the past. We present them separately, but several papers give a combined measure. These results could be consistent with almost any percentage, and for attraction, often thought to be most fundamental measure, average out to the (weak) 10% estimated elsewhere in this book. The results for women have somewhat lower errors:

Table 2. Genetic contribution for female same-sex attraction. Other parameters as for Table 1.

Factor	Bailey et al. ⁶⁰	Hershberger ⁵⁹	Bearman and Brueckner ³	Santtila et al. ⁸⁸
Attraction to women	32% (5% – 45%)	36%	0.0% (0-25%)	
Self-reported sexual orientation	37% (25% – 46%)	48%		
Frequency of female partners	52% (23% - 69%)	45%		16% (8.3-24%)

But the error ranges are still very large compared with twin studies on other traits. Bearman and Brueckner included full siblings in their comparison, but found the relative percentages bore no resemblance to any plausible genetic contribution.

The results from the four different sets of investigators are generally within error about the same, but from the viewpoint of traditional twin studies those errors are distressingly large.

When the twin concordance results for the fundamental attribute “attraction” are combined and recalculated, the genetic fractions for men and women respectively are $(36\pm 23)\%$ (which makes the value probably consistent with zero), and $(12\pm 17)\%$ which is zero within error. In contrast the values for the non-shared fraction are significantly different from zero and about 50%. That is generally true for the factors in the table, and combined measures.

Kirk *et al*⁶¹ re-examining the Australian data, welded together same-sex feelings, mental attitudes to homosexuality, and number of partners, into a new simultaneous measure of SSA that gave a stronger genetic component with much smaller errors. This was tweaked slightly until it gave the best result. But we must ask, if the traditional Kinsey SSA measures which seem so simple cannot be relied on, doesn't this study with its new measures need repeating? And doesn't it throw into question many previous studies?

There are other reasons for being very careful about these studies. There are rules for twin studies, and it happens that violating them leads in almost all cases to obtaining a genetic component which is too high. We must suspect that has happened, but acknowledge that the researchers have checked many of the factors where they can.

The Rules of Twin Study Analysis

For twin studies to be accurate in their conclusions about homosexuality, it would have to be shown that:

1. Identical homosexual twins did not volunteer for the study at higher rates than fraternal homosexual twins, nor those twins showing unusual eagerness to answer intimate sexual questionnaires. These factors create “volunteer error”, 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 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 - particularly, identical twins do not encourage each other to be homosexual.
7. The twins, apart from being twins, are very similar to the rest of the population (e.g. they are physically the same and about 1% are exclusively homosexual.)

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 the survey. Such twins tend to be more conservative, and less probably homosexual. (Homosexual respondents usually speak much more freely.) Each of these factors tends to over-estimate apparent genetic content.
2. Do families treat twins the same? (The “equal environments” assumption) Parents may claim they are utterly fair and even-handed and treat all their children the same way, but in fact they tend to respond to their children according to each child's differently expressed needs. Although this difference in style is usually not consciously planned, it amounts to a strongly individualized care that can be experienced as quite different from that given to another sibling. Parents can even have favorites, or one twin can mistakenly think that they have. Thus, non-identical twins may be treated quite differently from one another, contrary to the assumption of twin studies that they are treated identically. For example it is known that mothers of non-identical twins treat them with differing

warmth, but treat identical twins almost the same^{7,8}. If, for argument's sake, different parental treatment of fraternal twins contributed to a "defensive detachment" (of the kind discussed in Chapter 3) by a "disadvantaged" co-twin, twin study methodology would artificially inflate the genetic influence. In other words, twin studies would interpret an environmental factor as a genetic factor. The equal environments assumption was specially checked by Bailey *et al.* in the Australian study,⁶⁰ and the distorting effect for their study seemed small. But this equal environment assumption is violated by elusive random factors like idiosyncratic misperceptions of the family environment by one twin of a pair, and this can be very hard to pick up in standardized tests.

3. Homosexuality certainly does not have a normal distribution in the population, and that is what caused the large error ranges. The endpoint of these particular mathematical distortions is to produce a "genetic" contribution result which is too high.
4. Is there an interaction between effects produced by genes and the environment? Of course there is. That is almost the definition of a living organism. Probably the most important criticism which has been leveled at twin studies is that they treat nature and nurture as totally separate variables that don't interact during human development. If interaction does occur between the genes and the environment in any population under investigation in a twin study, it has the effect of artificially raising the calculated genetic contribution^{8,10,11}. Researchers are generally very critical of the idea that nature and nurture do not interact. "In a specific practical situation, do we really believe that the model is at all realistic? The answer is 'No'," says one statistician, Goodall.¹² So, these interactions certainly exist. Let's look at an example. If a person were genetically inclined to become homosexual, would an environment which encouraged him to express his sexuality (e.g. homosexual porn, or advances from homosexual men) have any effect on him? Of course it would. If he were brought up in an all-female environment and completely isolated from men all his life, could he have a homosexual relationship with a man? How could he? An environment containing men is essential. There is definitely interaction of the genes and environment. However it may not always affect the results too seriously. In the Australian study⁶², they tested for this specifically, and couldn't find clear evidence of it, only a strong suspicion. It is also fair in general to say that when gene-environment interaction has been clearly found it has had only a minor effect^{8, 11}. It is worrying though, because it is an effect which is easily missed. Very similarly the effects of genes and environment may not simply add together when they occur, but have a multiplicative effect. This effect certainly exists, and has been noticed in studies of personality. If this effect exists in SSA studies it would once again greatly overestimate the genetic contribution. According to one authority on psychiatric epidemiology Michael Rutter⁵⁰ "the traditional notion was that additive effects for genetic environmental influences would constitute the norm, and it is now apparent that this assumption must be rejected."
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? The best estimate at present is that there are no such genes, 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.¹⁵
6. Do twins tend to imitate each other in homosexual development? Twins certainly do imitate each other (e.g. in antisocial behaviour, in truthfulness or lying).⁸ 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 twins. 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. Older studies are very contradictory on the latter, so we make no comment. Hershberger⁵⁹ found statistical evidence in his sample that the identical twins had indeed influenced each other, in the occurrence of their SSA but did not find a genetic contribution in any case!
7. Perhaps most importantly, it is very doubtful that twins are completely similar to the general population. They start life smaller on average than other babies, and have fewer verbal and social

skills till as late as eight years.¹⁶ The rate of child abuse among twins is nearly three times higher than for the general population.¹⁷ They tend to be found towards 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”, probably adding to self-perceptions of childhood gender non-conformity, one of the strongest predictors of later homosexuality.¹⁸ The rate of hypospadias (congenital deformations of the penis and a marker of low exposure to testosterone in the womb) for twins² is 90% higher than normal, so their hormonal status is a little questionable, though the pairwise concordance for hypospadias is about 3 times that for SSA, meaning that low testosterone is not very likely to be the main cause of SSA in males. Twins are such good and sufficient friends to each other that their individuality and sexuality may not be entirely developed. For example, they are significantly more often unmarried than non-twins (33-40 percent of male twins may remain unmarried, and 22.5-26 percent of female twins – rates which may be about double those in the rest of the population, though this effect was not found in the Australian twin study. The overall rate of SSA among twins was 3.1%, slightly higher than 1.8% for an independent survey of prevalence in the general Australian population.⁶² Other surveys also suggest SSA might be slightly higher for twins than for the general population.) Summarising, they are a somewhat suspect population for sexual surveys.¹³

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, and in such a way that the genetic contribution is significantly over-exaggerated. Does this mean that twin studies are a completely unsuitable tool for gauging the genetic content of sexual orientation? At this stage, we can only judge by looking at much more sophisticated studies of other traits which have been investigated most closely for the effects of violated assumptions. In general, the genetic proportion is over-estimated as the assumptions are violated, so that the real influence is significantly lower, but it is still real. So it is a reasonable bet that the 10% figure for the attraction component of male SSA might represent some real though very minor genetic contribution but even so is too high. How much might it fall? We don't know, but it is quite likely that for males, it could ultimately fall significantly below 10%. Because twin studies so easily exaggerate the genetic contribution, it is much more likely that a study showing a lower figure is closer to the mark. Similarly the attraction result for lesbianism is very likely to be too high at 22% and may be finally near 10%.

The meaning of a 50% genetic contribution

Let's be very generous and grant that the genetic proportion of influence might be 50%. What does that mean? Does the 50% percent figure dictate behavior anyway?

Few studies of behavior actually report a genetic contribution of more than 50% for any trait, but many are around the 50% mark.

Obviously, even for something as greatly inherited as height, there is still some environmental effect. For example, a child fed a poor, protein-deficient diet will not grow as tall as one who regularly visits hamburger bars.

But even if genetic contribution is about 50 percent, does it mean that a behavior or personality is inevitable? Let's look at a few other twin studies that have come up with a trait close to Kirk *et al.*'s 50% for female homosexuality, and see how far that 50% figure actually determines a behavior. A few in this category are divorce^{42,43}, depression²³, altruism⁴⁰, religiosity⁴¹, fundamentalism¹⁵, psychological inpatient care³⁵, fear of the unknown³⁴, and perhaps alcoholism³⁹. We know enough about some of these to know that divorce, alcoholism, religiosity, and inpatient care are not genetically destined! The authors of the paper which found such a high genetic contribution for divorce were apologetic. Obviously, they remarked with some embarrassment, divorce does depend on another person. Other critics remarked dryly that even legal processes like divorce seemed subject to genetic influence!

An even higher genetic influence is seen for bigotry at 70%⁴⁵. It makes most people uneasy, to hear that racial prejudice has 70% “genetic influence”. Racial prejudice in the genes?! But the history of the last 50 years has shown that even for racial bigotry, people change. In the end it's your decision, isn't it? Somewhat easier for some people than others, but impossible for no-one reading this.

So it mightn't be easy, but with help, some of these traits that are apparently half inherited - even religiosity! - can be avoided. Significant intervention might be required for a long time, but Alcoholics Anonymous, Prison Fellowship, Marriage Guidance, and numerous support groups show that nothing is inevitable in these categories. Why should homosexuality be any different?

Even if the genetic factor is as high as 50% - and there are many reasons why it almost certainly isn't - homosexuality is not destined.

And don't forget: a 50% genetic factor does not mean homosexuality is 50% inherited. Homosexuality cannot be significantly inherited because only about 8% percent of the sons of homosexual fathers are also homosexual.^{47,48}

Genetic influence varies with environmental factors

"Genetics" in the twin study context is not a definitive statement about a *fixed* genetic content in any trait - and it is very important to understand this. It is a *relative* percentage only - relative to the contributions from common environment and non-shared environment. In twin studies the percentage "genetic influence" is used as a kind of snapshot of a balance at any one time and place between genes and a changing social environment. In twin studies, the measured genetic contribution is entirely malleable - it rises and falls inversely to environmental effects. That is, if genes are exerting a strong effect, but then opposite-effect environmental influences are brought to bear, the relative genetic contribution will drop accordingly. For example, twin studies in Australia have showed a genetic contribution to tonsillectomy which has varied over time, depending particularly on one environmental factor: whether or not tonsillectomy was medically fashionable.⁴⁹ When it was, the environmental contribution increased, and the relative genetic influence fell.

It is possible to deliberately increase the relative strength of genetic influences. You simply ride along with them, encourage and practice them. There are some fascinating Swedish and other studies on twins⁵⁰⁻⁵⁴ which show that genetic influences, particularly on mental processes, increase with age, right up to age eighty. How does that happen? When children are young, environmental influences - rules, habit formation, controls on behavior - are strongest. In adulthood more personal autonomy allows relaxation of some of the rules, so the genetic factor increases slightly in importance. In old age, people with far fewer pressures on them revert to what comes easily; they can please themselves more, relax. They can let the genetic influences predominate, or even encourage them. Many with disciplined habits will maintain a strong "environmental" influence on themselves, but, at least in some Western countries, on average, the contribution of genetic influences increases with age. Other studies show that genetic influences on children of school age diminish in highly-regulated households, but in households where the children are less restrained, the genetic influence is stronger.⁵⁵⁻⁵⁷

Genetic contribution to certain traits can vary from country to country and period to period. For example, the genetic contribution to height, which is 90 percent in the west, is far lower in Egypt, where family influences are far more important.⁵⁸ That is, in some third world countries, the height a person grows to may depend on the way limited food is shared round the family (some cultures preferentially feed boys, or oldest boys). Changes in social mores and increasing availability of food can swing the balance back toward a genetic predominance.

A recent Spanish twin study¹ looked at genetic expression and found it increasingly differed between identical twins with age. In other words environmental effects were changing the effects of the genes. This in turn probably depended on details of the lifestyles. This meant at age 3 the twins expressed their genes 4% differently, but at age 50, 22% differently. Even the influence of the genes can therefore be affected. What way are you going to affect yours?

This raises the question - how much are you going to go along with natural tendencies you have, and how much are you going to challenge them, if necessary all your life?

Summary

Homosexuality is not genetically determined. If it were, identical twins would show 100% concordance and no modern twin study on any behavioral trait has come remotely near that figure. In fact SSA is a good example of a trait little influenced by genes.

The simplest illustration that homosexuality is not completely genetic is pairwise concordance, which shows that a male co-twin is also homosexual only one time in nine, 11% of the time (Figure 16). This is very far removed from genetic determinism. And remember, this figure does not show that 11% of identical twins have SSA, (only 2-3% do) or that only 11% of homosexual twins are genetically affected and 89% are not.. All twins (all humans) are affected alike by genes ,and common environment and random factors.

The most recent and reliable twin studies (based on twin registers) still have huge error limits, making the genetic effect consistent with zero, and many factors suggest that the estimate values for genetic influence have been exaggerated. However non-shared environment is predominant and significant. Homosexuality fits poorly inside the twin studies model: the high ratio of heterosexuality to homosexuality and relative lack of bisexuals makes it unlike most other traits measured in twin studies.

However even if we accept the highest genetic effect that twin studies have produced – error bars and rule violation aside – don't forget what genetic effect means in twin studies. It is not a measure of how much a trait is inherited. It is, more accurately, a measure of the balance between environment and genetic input into a trait at any one place at a point in time. Genetic influence is something that rises and falls in direct response to the amount of environmental intervention. An opposite environmental influence can reduce a genetic effect to something relatively negligible.

But even if, for the sake of argument, we accept the highest twin study genetic figure of 50% as correct as an estimate for SSA, comparison with other traits showing genetic influences of comparable strength (eg divorce, altruism, religiosity, fundamentalism, depression, extroversion) makes it clear that homosexuality is not inevitable or fixed.

Twin studies also tend to eliminate the effect of family life and upbringing. But they pop up instead in the random factors – which, remember is the strongest category in twin studies - in the form of rare and indelibly remembered incidents that happen in family life, and in the form of different perceptions of the common family environment by different children.

The genetic effect of twin studies translates in real life into a weak and indirect effect. The scenario of a boy who was not very well co-ordinated and poor at sports, was rejected by his peer group, confused his emotional longing to belong with his sex drive and developed same sex attraction is about the extent of the genetic effect in homosexuality. A girl might be big-busted (genetic) and attractive, raped one day by a man, making her disown herself as a woman, so setting up a compensating search for re-connection in another woman. The psychological effects of the event may be devastating. The genetic effects which led to it are weak and indirect.

Ultimately, it doesn't matter much whether the genetic contribution is large or small. It doesn't fully determine our behavior. Any genetic influence can be counteracted with an opposite environmental influence, and an environmental influence can be counteracted with an opposite environmental influence. We are not the inevitable victims of our personal histories either.

Genes produce a tendency not a tyranny

You can foster or foil your genetic tendencies

You can prescribe either feast or famine for those tendencies.

The battle is not really at the level of our genes. It may not have been at the level of our conscious choices in the past, but it *is* at the level of our conscious choices right now.

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