CHAPTER TEN

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 quantitative, so they greatly focus and sharpen the results of 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 (a monozygotic (MZ) 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 predominant 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.

Four other points to take note of on the way through:

- Saying a trait is, e.g 10 % "genetic" is nothing extraordinary. There is at least a 10 % genetic effect in anything humans are and do, simply because without bodies we can't act in the environment at all. "Genetic" effects are experienced by everyone because we all have bodies. So homosexuality is like any other human trait
- Any genetic effects are mostly quite indirect
- For SSA they are weak
- They become relatively less important in the face of contrary environmental input

There is also good news in this chapter for parents who (usually mistakenly) hold themselves responsible for homosexuality in their children, either genetically or socially.

[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 classical twin studies and their limitations. Readers may pass over the second section if it seems too technical and move on to the summary.]

SECTION ONE

Twin studies

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 Josef Mengele, a researcher at the Auschwitz concentration camp, deliberately sought out identical twins for experiments. Sometimes he would kill one twin by poisonous injection, dissect that twin to see its effects, then immediately kill the co-twin to see the differences.

The founders of twin studies were very frequently involved in Third Reich theories of Aryan racial superiority and in favour of ethnic cleansing. Today twin studies are used constructively. By June 2010, about 33,000 scientific papers in medical databases mentioned twins and new papers are being published at the rate of a few thousand 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 quickly add non-identical twins (fraternal/dizygotic (DZ) twins), to give extra information about the relative importance of upbringing.*

*We will follow that order in this chapter, but emphasise identical twins because of unusual mathematical difficulties for SSA studies which arise when the non-identical twins (fraternal or dizygotic, DZ twins) are added in.

Concordance for SSA

Twin registers are the foundation of modern twin studies. They are now very large, and exist in many countries. A gigantic European twin register with a projected 600,000 members is being organised, but one of the largest in use is in Australia,[1] with more than 25,000 twins on the books, all of whom are prepared to assist in general research. If we use a register of identical (MZ) twins and find pairs in which one twin has SSA, we can then see if the other twin does or does not have SSA. This finding gives what is called the *pairwise* concordance. Readers should note that often a different measure is used without explanation or warning, called the *probandwise* concordance. This is a much less intuitive measure, needed for classical twin study calculations. It is often much higher than the *pairwise* concordance and when used without explanation, can give the impression that genetic influence is much higher for the trait under study than it actually is. The pairwise concordances in this chapter answer in an intuitive way the simple question—if one twin of an identical pair is SSA, what percentage of co-twins are also SSA?

Jones and Yarhouse,[2] for the important Australian Bailey et al. (2000)[3] SSA twin study paper, find that for self-declared lesbians and gays the pairwise concordance is 14 % and 11 % respectively. Anyone can verify this in the original paper. Five, mostly later very large studies, gave very similar results. The pattern is similar for males and females, looks like **Figure 22**.

Figure 22. Concordance for SSA in identical twins is one in nine.

This means that for every nine sets of male identical (MZ) 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. That is, *identical twins usually differ.*

What 11 % concordance means

What does what 11 % concordance mean?

It does *not* mean that 11 % of identical twins have SSA. Numerous studies of western populations (Chapter Two) have shown that homosexuality (including bisexuality) is present in something between 2-3 % of people, and this, of course, includes twins, e.g **Figure 23** 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 therefore concordant.

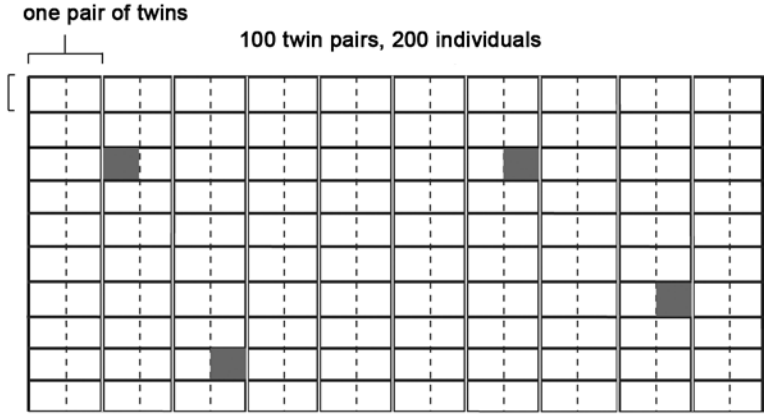

Figure 23. Prevalence of SSA in twins

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 exposed to genetic effects, 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 a twin pair from a general twin registry is homosexual, his co-twin is homosexual one time in nine, or 11 % of the time.

Twin studies give information about family environments

It's also important to emphasise that in twin studies concordance is not only a kind of catch-all for any *biological* factors in common, e.g things like biological environment before birth, but also for common post-birth *environmental* factors. Identical twins not only have identical genes, but they (usually) grow up in the same family environment. **So Figure 22** illustrates the *combined* effects of a shared genetic inheritance *and* a shared 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 at first sight for SSA, it seems that neither genes nor upbringing is very important. Put another way, in homosexuality the practical effect of genes, other shared pre-natal biological factors, and a shared home environment, is *weak*. (More about the shared family environment later.) So if shared factors are not important what is? In SSA it is non-shared factors: things happening to one twin but not the other, or a personal response to an event by one of the twins and not the other (that response having far-reaching effects.)

We will call this non-shared fraction, the *random* or *chance* factor and for SSA it is 89 %.

We also want to emphasise that the use of pairwise 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 in the general population and/or what the concordance is for fraternal twins, (see Section Two, on classic twin studies.) However the fundamental point will remain true: the largest single cause of SSA is random factors (meaning, factors affecting one twin but not the other).

Same-sex attraction is not inborn

We can now make our most important point:

Those with SSA are not born that way.

If factors in common like genetics or conditions in the womb overwhelmingly cause SSA, then identical twins will *always* be identical for SSA, i.e the SSA concordance would be 100 %. But they are not 100 % concordant for SSA, so it is clear that post-natal random factors are mostly responsible for SSA. We could also sum up **Figure 22** by saying that for SSA *genes create a tendency, not a tyranny.* Even the tendency is weak. This is a critically important principle.

In the discussion here we concentrate on adults and postpone until Chapter Twelve the implications of even lower SSA concordances in identical adolescent twin students in the 2002 paper on SSA by Bearman and Brueckner.[4] *** ***

Twin studies cover all possible shared biological influences—known and unknown

Our second important point is that because *"genetic"* in twin studies includes everything from the shared biological environment (**Figure 22**), twin studies reflect all genetic/biological influences, those known and those not yet known. It is a statement of the realities no matter how many details have yet to be scientifically discovered. And many more details will be discovered as scientific papers continue to find new factors at the rate of about one every year. Remarkably, twin studies summarise all the shared biological effects on developing twin embryos that will ever be discovered. And, to repeat: at 11 % the combined genetic effects are weak for SSA.

**In 2002 Bearman and Brueckner described part of a large ongoing study of tens of thousands of adolescent students in the USA. From this sample they chose a large number of twins and other relations for genetic studies. The SSA concordance between MZ twins was only 7.7 % for males and 5.3 % for females—lower than the 11 % and 14 % in the Australian study by Bailey et al. (2000). But in Chapter Twelve we show that SSA adolescents are a special case—generally changing their attractions from year to year.

This degree of concordance now has the backing of half a dozen major twin studies so is very unlikely to change. So 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.

As clear as this conclusion is, it has not been foremost in the thinking of academics because research has tended to concentrate on the question, "Is SSA genetic?" and this has caused some problems of interpretation,

For example, the book, *Born Gay*[5] 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. (The authors argued that either high or low hormonal exposure for one identical twin but not the other could account for non-concordances in SSA.)

But this is fallacious. If twins share the same womb environment, then any hormonal effect on one twin will be experienced by the other. It is well-known in DZ (fraternal) twins (on separate placentas) that a girl is very slightly masculinised by the testosterone from her brother twin. It follows then that MZ (identical) twins, sharing the same sac and in many cases a joined blood circulation, will be even more mutually influenced.

Unusually high hormone levels are part of the shared pre-birth biological environment and unusually low levels in one twin tend to be compensated for by the other twin, creating a levelling out of exposure.

So, 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 would be affected by it and both would be homosexual. However, twin studies show they are not—in 89 % of cases.

As a shared effect, hormonal exposure properly belongs in the twin study "genetic category," which the authors rate at 30 %. This is a weak to modest effect.

SSA concordance compared with concordance for other conditions/traits

In **Figure 24** we compare the degree of *pairwise* concordance for SSA with *pairwise* concordance for other traits and conditions, to give some perspective. We concentrate on low concordance conditions. On the left is lung cancer with an almost zero concordance. This means that if one MZ twin has it, the co-twin almost always does not. This illustrates that neither common environment nor genetics is responsible for lung cancer, but *chance* or *random* factors.

SSA at only 11 % concordance is therefore strongly dominated (89 %) by chance. Few other conditions produce such MZ twin differences except the cancers, stroke and criminality (not shown).

This conclusion should be spelt out again in a slightly different form: the largest factor in SSA twin studies is non-shared influences,

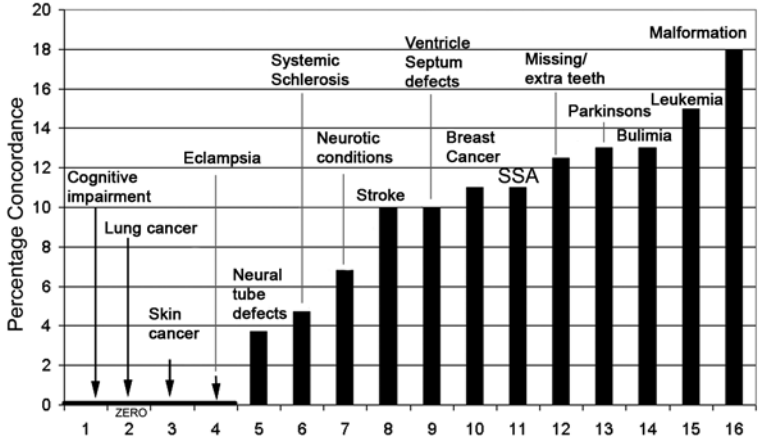

Figure 24. Some low pairwise concordances for identical twins. (Taken from PubMed.) The male figure is given for SSA.

1. Cognitive impairment[6] 2. Lung Cancer[7] 3. Skin cancer[8] 4. Eclampsia[9] 5. Neural tube defects[10] 6. Systemic sclerosis[11] 7. Neurotic conditions[12] 8. Stroke[13] 9. Ventricle septum defects[14] 10. Breast Cancer[15] 11. SSA[1] 12. Missing/extra teeth[16] 13. Parkinson's[17] 14. Bulimia[18] 15. Leukemia[19] 16. Malformation[20]

i.e random or chance events: things happening to one twin but not the other, or different perceptions of, e.g upbringing or a one-off event, causing it to have special significance for one but not the other.

In contrast to SSA, left-handedness has a 13.5 % concordance similar to that of SSA. The difference is that the gene/left-handedness connection is now much better established, while nothing clear has been found for SSA. Even given the genetic connections to left-handedness, it is still overwhelmingly due to non-shared environment—chance.[22]

Gene penetrance

Poor gene penetrance is a red herring

An argument sometime advanced to explain low concordance in twin studies for SSA is poor gene penetrance. This holds that there is a gene which is important and responsible for the trait but for unknown reasons (probably connected to cell biochemistry) it does not exert its effects in those without the trait. Low penetrance indeed exists but classical twin studies cannot possibly untangle it from other chance effects in the environment. However, to argue poor gene penetrance you must first find the gene (or genes) involved, then show that they switch off in some conditions. But, as discussed in Chapter Nine, no such homosexual gene has been found or is expected to be. In addition, penetrance as an explanation for gene effects is unusual. Sir Michael Rutter says "Penetrance is not very usual for single gene effects."[21] Thirdly, the concordance for MZ 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 three layers of speculation:

- that SSA genes exist;
- that they are switching off in one twin and not the other
- that the penetrance effect is unusually weak for SSA compared with other traits

Low penetrance is no explanation for low pairwise concordances. Rather, from the perspective of our best present knowledge, the effects of genes and shared environment are low, and random events dominate.

Summary

One thing seems clear: any genetic contribution to SSA is much less than in most traits for which genetic influence has been measured. SSA seems 90 % a result of random factors. SSA is in fact a good example of *not* being "born that way"!

Higher SSA concordances from pre-register studies (before 2000) are now agreed to have resulted from an unusual degree of "volunteer error" and are often given as *probandwise* concordance (see p176), which is considerably higher than the 11 % result.

It is also salutory to note that the better the twin sample, the lower the SSA concordance, i.e the lower the genetic influence. In other words volunteer bias greatly exaggerated those early results, which are unfortunately still quoted widely.

Some might expect concordances for male SSA to be different from concordances for lesbianism, but concordance for *both* men and women is unusually low. At 11 %-14 % this suggests that shared genetic and environmental factors are weak in both cases and that something else is going on. Again we posit idiosyncratic responses to random/chance factors.

Minimal effect of family environment

A second feature of these pairwise MZ twin study data is the apparent minor effect of family environment (upbringing) on the development of SSA. Both shared biological effects and common family environment added together produce only an 11 %-14 % pairwise concordance. Fuller studies, which include DZ twins, also usually 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, either genetically or through parenting.*

Those who know classical twin study results will immediately recognise a common pattern. Results from twin studies for very

many traits show family influence is less than genetic influence. However lack of family influence is a controversial issue. Developmental psychologists didn't believe it—they noticed many obvious effects of family environment in their clinics. But the twin studies experts stuck to their statistical conclusions, and argued that the third factor, randomness (in our terms individual reactions to chance events) is even more important than genes or family. They then found that many shared family influences were hidden in that random group, so family effects only *seem* very small but they are hard to separate from the true random effects.

There is a lot of evidence (see Chapter Three) that (hetero) sexual orientation is usually developed in the family first through imitating the gender patterns of the same-sex parent and siblings, though soon the same-sex peer environment becomes much more important. But random/chance factors strongly 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. Importantly he concludes that genes tend to make people more similar, and environmental random factors tend to make them differ more.[23]

What might random environmental factors be?

Random factors could include: perhaps the sexual abuse of one twin but not the co-twin; perhaps reactions to perceived parental preference of one twin above the other; maybe one twin is exposed to gay pornography and develops a habit, but his co-twin does not; maybe one male twin misinterprets his intense envy and admiration of confident, popular boys and wonders if he is gay; perhaps one is persistently unlucky with girls, unlike his co-twin, and seriously questions whether he may be gay; one might be the target of denigrating sexual innuendo from other males, but not the co-twin; a slightly gender-atypical physical feature may sometimes be taken obsessively to heart by one child, but not another.

Twin study researchers say it is random factors (chance occurrences), which predominate in the development not just of homosexuality but many other traits. From the point of view of twin

studies, if the question is asked: is SSA mostly nature or nurture? the answer would best be, *Neither, it is mostly chance events.* This is an unexpected and probably unwelcome answer to the decades-old nature/nurture argument!

So family effects and randomness are both important. But how can a family effect appear as randomness? The linking idea is that the unusual random event is very influential.

More about the (extra-)ordinary factor, the chance 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 fades into the background and tends to be overridden by reaction to a few events in the family—which assume great significance for one child, but not another.

Some of the extremely unusual incidents are therefore extraordinarily powerful influences. This can include 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 hormonally charged adolescent can burn themselves into the memory and affect sexual responses. Unusual random events can impresses themselves on our memory, and affect our responses and behaviour for years. Many homosexual men and women, recalling incidents which they believe were instrumental in the development of their SSA, will recount clear early memories of one particular thing done or said in families that deeply influenced their later choices.

Examples of other powerful unusual factors are given later in this chapter.

Different perceptions of the same environment

The different way two people can describe the same incident helps us understand why the effect of the shared family environment seems so small in twin studies. The environment is the same but the *perception* of it and one's upbringing can be quite different in the eyes of different members of the same family. Bailey conducted interviews with a number of identical twins discordant for SSA, i.e one had SSA the other didn't. He found ways in which they had perceived the same family environment differently. These different perceptions show up in the twin study data as random occurrences, and they are. But what they also can be, are *different reactions to the same environment*. They may represent the reactions of a twin, who for example, mistakenly takes it into his head that his parents don't like him nearly as much as his brother. Parents can often 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 intentional choice has not been a significant factor in the development of SSA.

An illustration of this divergent reaction is a study which showed that MZ twins experience the same classroom differently.[24]

MZ twins can and do react differently to the same circumstances. (Of course, children who are not twins can also react very differently.)

Therefore:

> Upbringing and shared social environments are showing up after all, but heavily disguised as random factors. Put another way, the random category in SSA twin studies carries within it a significant influence of upbringing and family environment, responded to differently.

So, the random contribution includes upbringing and common environment while appearing to exclude them. Much more research is needed on this, 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. Check from time to time. Have they really perceived an important event in the family accurately? How deeply did an unusual event affect them? Do you need to diminish its importance or explain it for them?

Perhaps your son or daughter is gay. Probably its origin had nothing to do with you. But you may be blaming yourself, or others may be blaming you when its roots really lie in major misperceptions of events, and some resolution might be possible.

In rare cases, parents may have been more deeply implicated, e.g long-term emotional distance from a child or abusive treatment. Personal reform and making amends may help reduce the distance and lessen the drive for same-sex love over time.

Factors important to some people with SSA

There is a wrong but popular impression that SSA development is a great mystery. But in many ways it's no more mysterious than the development of heterosexuality. And most people with SSA can point to several factors which were of some importance.

Otis and Skinner[25] in a non-twin study identified some of them by sampling a group of SSA men and women who said the factors in **Figure 25** had at least some influence on their orientation.

Of course no-one directly experienced genetics as an important factor! But perhaps some thought that a physical feature was important, and perhaps obsessed about it. Many of us do! But in most cases we can assume they had heard about probable genetic influence from the media and from gay sources and ticked the box.

A little calculation shows that most people ticked a number of factors. It thus seems, *even for an individual, that multiple factors are involved.* This reflects the mainstream scientific view about behavioural traits as expressed by Sir Michael Rutter: **"**The great bulk of psychological traits…is multifactorial in origin."[21]

We now give an even more extensive list of things that people may react to. The "thing" and the reaction to it can contribute to SSA if other factors are in place. These things and reactions to them are the "chance" factors we have been talking about. We have drawn them from personal accounts of people with SSA and from

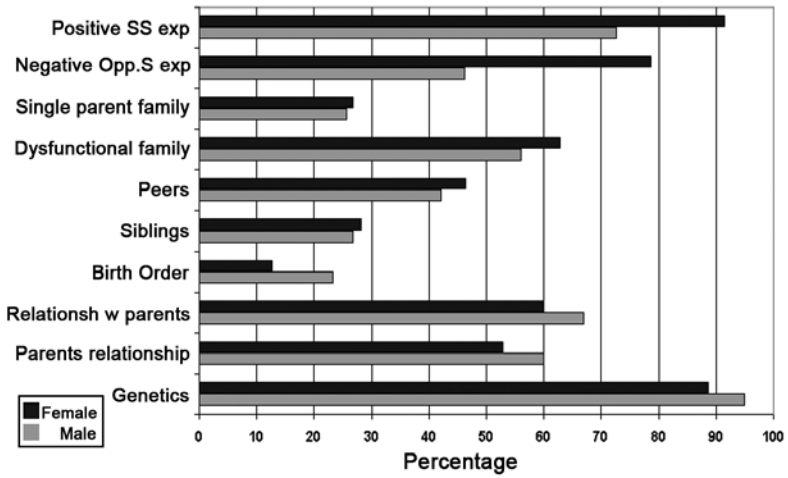

Figure 25. Factors thought by gay and lesbian people to have had some causal connection to their SSA

the literature. Most people with SSA will say "Most factors on the list were totally irrelevant to me, but a few were important". The important ones will differ from person to person; in no case will one factor be important to the majority. Some are reactions to body features, hence "genetic". Some of the genetic influences are not from personal experience, but are claimed to be significant. Others are much more environmental, and include chance meetings and individual reactions. Some may appear highly improbable on first reading, but all have been important to *someone*. Some factors are in both lists.

Some SSA people will identify with nothing on the list. If so a little thought might turn up something which is significant. If an event or feeling has been mulled over numerous times for years, it is important, possibly formative and may even be a mind habit.

Reactions to factors of predominantly genetic *origin: (16 factors)*

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

- Fluctuating asymmetry (includes left-handedness and irregular physical features)
- Inherent gender atypicality (genetic influence is generally found to be high for this feature)
- Intersex conditions (this is a special case, because gender identity may not correspond to chromosomal identity. The vast majority of SSA people are not intersex.)
- Congenital disability
- Left handedness (included because there is a modest genetic contribution)
- Novelty seeking (in so far as this is genetic, it can lead to trying many unusual sexual experiences)
- Obesity (in women)
- Older brothers (men)
- Physical handicap (can include deafness, other handicaps)
- Polycystic ovaries
- Poor coordination (in men, particularly in sports, the converse for women)
- Retiring temperament/tomboy temperament (men/women respectively)
- Unattractive/ "unfeminine" physical features (in women)
- Visio-spatial defects/aptitude (in men or women respectively)
- X-chromosome inactivation (in mother and if atypical and extreme)

Reactions to factors of predominantly environmental *origin (46 factors)*.

- Adoption (possible disturbance of bonding and modelling)
- Alleviation of depression (having SS sex to lessen depression.)
- Bad luck in love (leading to self-questioning)
- Bad opposite sex experience
- Bullying (mainly males)
- Chance encounter (with an attractive same-sex partner)
- Discrimination (mainly reinforcing a position already adopted)

- Divorce (impacting perceptions of sexual adequacy in both sexes)
- Dreams (particularly sexual, leading to questioning of orientation)
- Easier sex (men—less commitment required for same sex)
- Envy (of attractive same-sex attributes)
- Exercise of power (demonstrating dominance)
- Fashion (extreme concentration on aesthetic values—men)
- Feelings of rejection
- Fetishes (partialisms)
- Gay culture attractive (shared aesthetic appreciation—males)
- Gay pornography (mostly men)
- Gay social pressure (mainly on bisexuals—to be either gay or straight)
- Habit (repeated pattern of responses)
- Liberal cultural environment (encouraging experimentation)
- Marriage resistance (lesbian)
- Maternal stress (SSA women only, affected by stress in mother)
- Middle age (women, may coincide with family leaving home)
- Obesity (women)
- Older brothers (men, included here because the biological immune argument is highly speculative)
- OSA intimacy problems (overlaps poor social skills)
- Parental encouragement to be gender atypical (often for amusement)
- Parental negative messages (about gender inadequacy)
- Passivity
- Political climate (lesbian/feminist solidarity)
- Polycystic ovaries
- Poor social skills (more important for males)
- Prescriptive cultural environment (many anthropological examples)

- Reactions to parents (no identification with same-sex parent: sometimes a result of misperceptions)
- Rebelliousness (a rejection of same-sex stereotypes)
- Resistance to categorisation (women, leading to resisting the prevailing gender environment)
- School peer pressure (denigration for lack of masculinity—males)
- Sensual factors (seeking repetition of pleasure)
- Sexual abuse (same-sex for males, and opposite sex for women)
- Sexual experimentation (prolonged, with same sex)
- Shyness (similar to poor social skills)
- Single parent family (absent male role mode for boys)
- Slimming pills (Taken by mothers during pregnancy and affecting daughters)
- Soul mate (quest for deep intimacy—women)
- Urban environment (opportunity and anonymity a factor)
- Verbal abuse (particularly about gender atypicality)

The "environmental" list is three times as long. 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. *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.*

SECTION TWO

It's now time to look at the rather more complex classical twin studies. This next section will amplify the points already made. Skip it if you wish. Congratulations on getting this far!

Classical twin studies (identical and non-identical twins)

Classical twin studies use both MZ and DZ twins, trying to disentangle the relative contributions of genetics, shared environmental experiences and non-shared experiences. Classical studies look at more than just twin concordance.

The last half dozen twin studies on homosexuality (1998-2010), are the most important. Studies earlier than 1998 suffered severely from volunteer errors, i.e twins (particularly MZ) who were similar in sexual orientation tended to volunteer for the SSA twin studies in much greater numbers than those who were dissimilar, and this distorted the results, exaggerating the genetic content much more than researchers thought possible. Later large studies were done using the twin registers and when volunteers signed up they didn't know they would be asked about SSA. Below we display only registry-derived results. These should greatly diminish the bias problem, but not eliminate it, because even on a twin-register, twins have to agree to take part in a given study. Probably again, those twins who were both SSA and knew it, disproportionately agreed to take part. No-one really knows the extent of the problem.

Even these studies, the best to date, encountered another problem: the unusual mathematical form of the occurrence of SSA in the general population. In twin studies the trait under analysis should present as a bell curve, e.g height in a population would produce a bell curve, with most people of average height, tapering away to very few at the extremes. However, the shape of the histogram for varying degrees of homosexuality is like this upper-case J inverted left to right. (Among women there were relatively more bisexuals and the mathematical problems were not so great.)

But this fundamental mathematical twin study pre-condition for SSA 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 SSA studies which use MZ and DZ twins together more difficult to interpret than most other twin studies.

Researchers used a variety of measures of SSA. Most think that current attraction is the most best measure, and the other measures, (self-identification, same-sex activity) are more culturally influenced. However the results below have such large errors, that it turns out it matters little which measure is used.

We don't describe here the detailed mathematics, which is a learning curve even for graduates. But, in examining a trait, twin studies attempt to find the relative contributions to that trait of genetics, shared environment (family, social groups) and non-shared environment, i.e random effects.

The results, shown in **Figure 26**, are very scattered and have large calculated errors (often consistent with zero for genetic effect) and no measure gives a more solid result than any other. The figure shows genetic-fraction calculations for different SSA measures.

The mean for men is $(22 \pm 20)\,\%$ where the error is the standard deviation. It makes no statistical difference whether this is recalculated restricting the results to attraction or behaviour. The errors are very large. A $22\,\%$ genetic fraction is real but usually considered weak.

For women the mean is $(37 \pm 18)\,\%$. So a rough estimate of genetic contribution to female SSA is $37\,\%$. The result is real but its strength is only weak to modest in twin study terms.

Figures 26 and **27** did not use the rigorously random study by Kendler[33] (not a twin registry but random sampling of the population), which combined results from men and women and did not allow separation of the two sexes for the Figures (though their answer is much like the others).

The Santtila[37] survey was the largest (2334 pairs of twins) and like the second largest by Langstrom et al.[36] used the Scandinavian health records (effectively compulsory twin registers), ensuring a relatively unbiased sample.

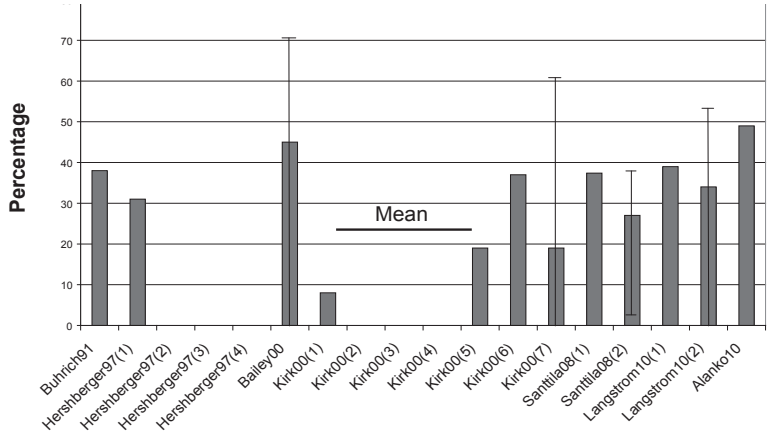

Figure 26. Estimates of genetic contribution to male SSA by various measures. Typical 95 % error bars for selected studies are given. References and measurement basis: Buhrich[26] Attractions plus fantasy plus contacts: Hershberger[27](1)Attractions when older than 25y: (2) SSA Partners when older than 25y: (3) Sexual orientation (gay, bisexual,straight): (4) Same, but modelling included siblings: Bailey[3]: Kirk[1] (1) 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[28](1) Potential to be SS-involved (fantasy): (2) SS Partners in last 12 months: Langstrom[29](1) Any lifetime SS partners: (2) SS partners over life to date. Alanko[30] Attraction plus behaviour allowing for libido. The Kirk paper reports results from the Australian study more usually known as headed by Bailey.[3]

The end results from the different studies are generally within error about the same, but compared with classical twin studies on other traits those errors are disconcertingly large, and also prevent estimation of the genetic content for OSA.

The mean figures for the genetic content for men and women, 20 % and 36 % respectively, are much less certain for men than for women, but are probably maxima because both are still subject to the problems described later in this chapter. The percentages will almost certainly reduce with further research.

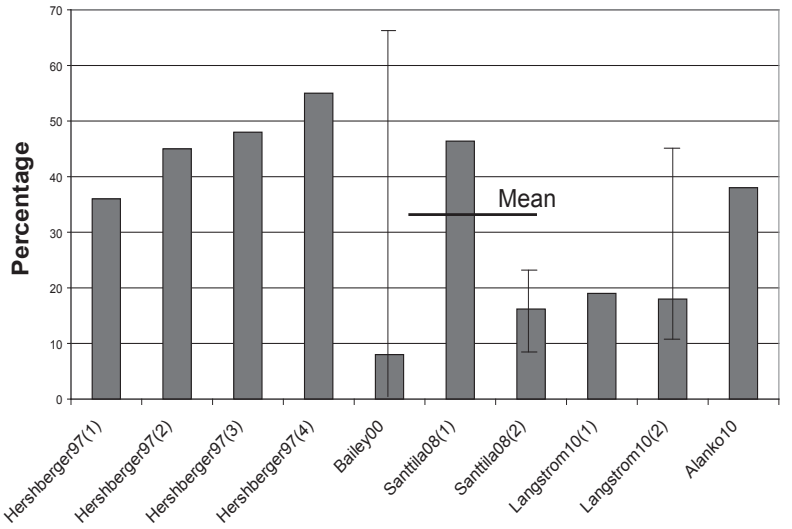

Figure 27. Estimates of the genetic contribution to female SSA. References as for Figure 26 plus Kirk.[31] Typical error bars are shown.

It is a little ironic that the genetic content appears significantly less for men than women, because it is the males that have always been anxious to ascribe their attractions to "genetics". The women have preferred to believe their attractions are under their own control.

The non-shared fractions for men and women are shown in **Figures 28** and **29.**

An important outcome of all the SSA studies is that the "non-shared environment"/random fraction is always larger than the "genetic fraction" and has much smaller errors on it.

The non-shared environment contains several components. These are (a) possible error in measuring (defining) SSA (b) biological randomness (c) differing random psychological reactions (d) differing random events affecting one MZ twin but not another. The measurement error is probably much less than in, e.g psychiatric studies of twins, in which conditions can be hard to diagnose.

An example of biological randomness, (b) above, is a kind of "biological noise" at the biochemical level of cells. This may produce different weights in a colony of genetically identical

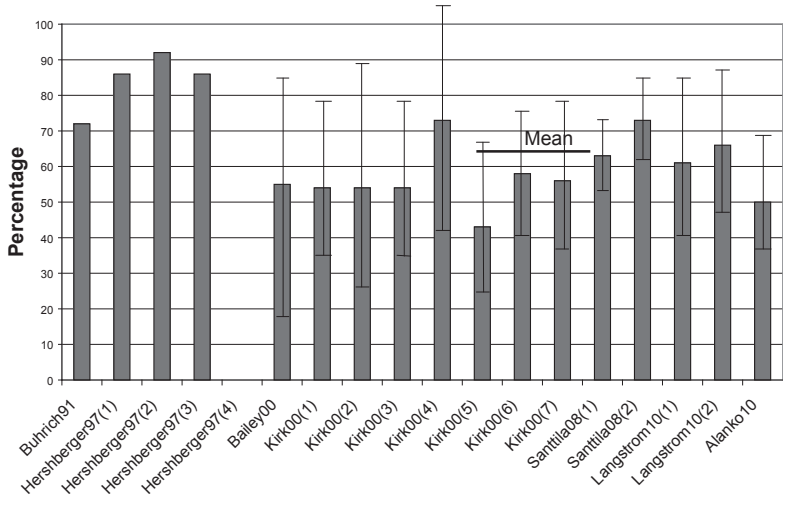

Figure 28. Estimates of the non-shared environmental contribution to male SSA. References as for **Figure 26**

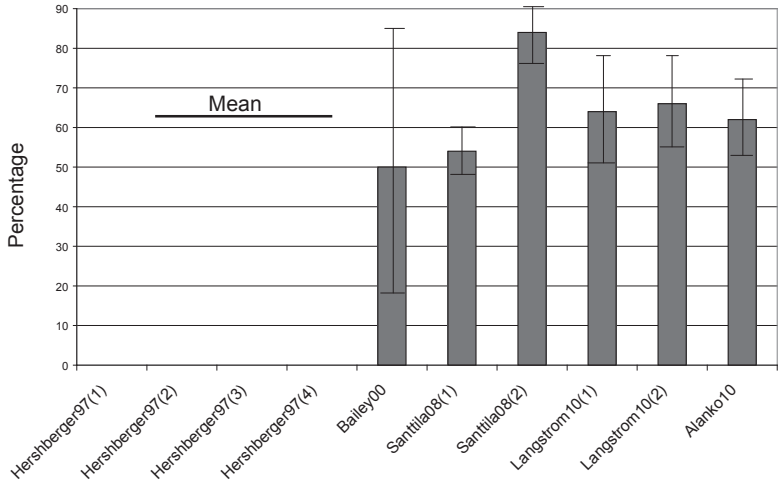

Figure 29. Estimates of the non-shared environmental contribution to female SSA. References as for **Figure 27.**(Hershberger gives no results for female non-shared environment)

animals however much researchers breed for uniformity and no matter how standardised the environment. This "noise" effect has been known and puzzled about for many years. Some people may try to argue that biological noise creates homosexuality. If it does exist then it would appear in the random factors fraction.

As mentioned above, the Bearman and Brueckner adolescent twin study was not considered in this section. However it has an important implication. It was a very large study but there was a calculated 0 % genetic contribution to SSA. The implications for teenagers who think they have SSA and that it is genetic? No it isn't, and what is more, in 98 % of cases the same teenager will be heterosexual the following year (see Chapter Twelve). This huge swing over such a short time stands in huge contrast to the genetically programmed events of puberty, which appear in twin studies to be about 90 % genetic.[32] This is also much higher than the 20 % and 36 % genetic fractions for SSA. The degree of genetic programming must be very low for SSA compared with puberty.

The rules of Twin Study analysis

There are rules for twin studies, and violating them leads in almost all cases to a genetic fraction which is too high. We now survey the rules. We think they are often violated in SSA studies, but accept that researchers seek to eliminate bias 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, or show unusual eagerness to answer intimate sexual questionnaires. (This "volunteer error" effect is also 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—particularly, identical twins do not encourage each other to be homosexual. (The "twin" effect.)

7. The twins, apart from being twins, are very similar to the rest of the population, e.g they are physically the same and the same percentage are homosexual as for the non-twin population.)

8. 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 minimised (but not completely removed) by using modern twin registers. However, 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. (Homosexual respondents usually speak much more freely.) Each of these factors tends to overestimate apparent genetic content.

2. Do families treat twins the same? 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 and varying insistence. Although this difference in style is usually not intentional, it amounts to a strongly individualised care that can be experienced as quite different from that given to a brother or sister. Parents may also have favourites, or one twin can mistakenly think that they do 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 DZ twins treat them with differing warmth, but treat MZ twins almost the same.[33,34] For argument's sake, if different parental treatment of DZ twins contributed to a "defensive detachment" (of the kind discussed in Chapter Three) in a co-twin, twin study methodology would exaggerate the genetic influence. The equal environments assumption was specially checked by Bailey et al. for a few factors in the Australian study,[3] and the distorting effect for their study seemed small. But the 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 standardised tests.

3. Homosexuality does not have a normal distribution in the population, and that is what causes the large error ranges in twin study results for SSA. The endpoint of these particular mathematical distortions again produces a genetic fraction which is too high. Santtila and colleagues tried to allow for this mathematically, and presented evidence they had succeeded. But the calculated genetic fractions were much the same as those from other authors who did not allow for it. So it may be only a small effect.,

4. Probably the most important criticism which has been levelled at twin studies is that they treat nature and nurture as totally separate influences that don't interact during human development. But interaction between genes and the environment is almost the definition of a living organism. If interaction does occur between genetic influences and the environment in any population in a twin study, it again has the effect of exaggerating the genetic contribution.[34,35,36] 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.[37] 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.

On the other hand, if the man were raised in an all-female environment isolated from men all his life, could he have a sexual relationship with a man. How could he? An environment containing men is essential. There is obviously interaction of the genes and environment.

However it may not always affect the results too seriously. In the Australian study[31], they tested for this bias, and couldn't find clear evidence of it, only a strong suspicion. It is also fair to say that in studies of other traits, gene-environment interaction has

only had a minor effect[34, 36] However this 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? We have already discussed (Chapter Nine) that none have been found, so this is unlikely to be important. However, if this effect existed it would *under*estimate the contributions from genes.[38]

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.[34] It is quite possible it might happen with homosexuality. Twins often have an unusually close bond, sharing intimately and backing each other up, 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. Mutual influence could range from talking about SSA to exploratory sex with each other. Studies are very contradictory on the latter, so we make no comment. Hershberger[27] found statistical evidence in his sample that the MZ twins had indeed influenced each other in the occurrence of their SSA.

7. It is very doubtful that twins reflect 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.[39] The rate of child abuse among twins is nearly three times higher than for the general population.[40] 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," the kind of labelling that can create self-perceptions of childhood gender non-conformity, one of the strongest precursors of later homosexuality.[41] The rate of hypospadias (congenital deformations of the penis and a marker of low exposure to testosterone in the womb) is 90% higher than normal[42] compared with non-twins, so their hormonal status is a little questionable.***

***See p137 for hypospadias. The pairwise concordance for hypospadias is about three times that for SSA, meaning that low testosterone is not very likely to be the main cause of SSA in males. It is more likely that another known factor, placental insufficiency, is responsible for much of the increased percentage[66] of hypospadias

Twins are such good and sufficient friends to each other that their individuality and sexuality may not be entirely developed. For example, they may be twice as likely to be unmarried as non-twins, 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.[31] Other surveys also suggest SSA might be slightly higher for twins than for the general population. Summarising, it is very doubtful twins reflect the general population, so they are a somewhat suspect population for sexual surveys.

8. In traditional twin studies the genetic fraction is probably further overestimated because of the effect of chorionicity on gene expression—meaning whether MZ twins have a shared placenta in the womb or separate placentas (like DZ twins). MZ twins can have either and it makes a difference! In a very important paper, Kaminsky et al. (2009)[44] studied about 6000 differences in gene expression between twins. If the twins had a shared placenta they were much more alike than they would have been without a shared placenta *because* twins sharing the same placenta share the same blood supply. (About 25% of MZ twins share the same placenta.) When both kinds of MZ twins are combined (those sharing the same placenta and those with separate placentas), the average concordance for SSA is higher than it should be. Sharing the placenta exaggerates the difference between MZ and DZ twins (the classical twin method depends on this difference) and hence overestimates the genetic fraction. Assuming an allowance needs to be made for the MZ placenta effect, the average effect for all gene expression is that the genetic fraction is 15% too high.[44] Does this apply to SSA also? No-one knows, but probably. This would reduce the 22% genetic contribution to SSA to 7% (for men) and from 37% to 22% (for women). The genetic effect for the women would then also be classified as weak.

Breached rules exaggerate genetic fraction

In most twin studies of homosexual populations, most of the assumptions that must be met if the results are to be valid have been breached, and in such a way that the genetic contribution

is significantly overstated. Does this mean that twin studies are a completely unsuitable tool for gauging the genetic content of sexual orientation? No. When other traits have been investigated closely for the effects of violated assumptions, the genetic proportion is over-estimated but is still real.

10% a more likely genetic fraction

So it is a reasonable conclusion that the 20% estimated figure for the genetic component of male SSA is too high but represents some real though very minor and indirect genetic contribution. It is quite likely that for males it could ultimately fall significantly below 10%. Similarly the attraction result for lesbianism is very likely to be finally near 10%.

In summary we estimate that corrections made to this over-estimated genetic fraction for SSA reduce it to about 10% for men and women.

The meaning of a 22-37% genetic contribution

Let's be generous and grant that the genetic proportion of influence on SSA might be 22-37%. What does that mean? Does a 22% genetic figure dictate behaviour anyway?

Church attendance is also close to 22% "genetic."[45] If we don't think church attendance is very "genetic"; then we should view SSA the same way.

Even much higher percentages still do not dictate behaviour. A few in the 50% category are divorce,[46] depression,[47] altruism,[48] religiosity,[49] fundamentalism,[38] psychological inpatient care,[50] fear of the unknown,[51] perhaps alcoholism,[52] and most interestingly homophobia!![53] Are they changeable? 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 cynically that even legal processes like divorce seemed genetically influenced these days!

Homophobia?! Prejudice in the genes?! Unlikely! But the history of the last 50 years has shown that even for homophobia, society's attitudes clearly change. It's somewhat easier for some people than others, but not impossible for anyone reading this.

So it mightn't be easy, but with help even some of these traits that look half inherited can be avoided. Significant intervention might be required for a long time, but Alcoholics Anonymous, 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 37%—and there are many reasons why it almost certainly isn't—homosexuality is not destined.

Genetic fraction changes with environmental input

Even a 22% genetic factor does not mean homosexuality is 22% inherited. Homosexuality is not significantly inherited because only about 8% of the sons of homosexual fathers are also homosexual.[54]

"Genetic" 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—fluctuating depending on influences from the common environment and non-shared environment.

In twin studies the "genetic fraction" is used as a kind of snapshot at any one time and place of a balance between genes and a changing social environment. If genes are exerting a strong effect, but then opposite-effect environmental influences are brought to bear, the genetic fraction will drop. For example, researchers found strong genetic influences in the United States on smoking for those born in the 1920s, 1930s, and 1950s, but lower genetic influences for those born in the 1940s (WWII cigarette shortages) and 1960s (cancer findings). Legislation in the 1970s and subsequently prohibited smoking in public places reducing this genetic influence still further.[55] That is, the environmental contribution increased, and the relative genetic influence fell.

To increase the relative strength of genetic influences simply ride along with them, and practise them.

There are some fascinating Swedish and other studies on twins[56-59] which show that genetic influences, particularly on mental processes, increase with age, right up to age 80. How does that happen? When children are young, environmental influences—rules, habit formation, controls on behaviour—are strongest. In adulthood more personal autonomy allows relaxation of some of the rules, so the genetic fraction increases slightly, i.e elderly people with far fewer pressures on them can revert to what comes easily; they can let 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.[60-62]

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 % in the West, is far lower in Egypt, where family influences are far more important.[63] 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 study[64] looked at genetic expression and found it increasingly differed between MZ twins with age. At age three the twins expressed their genes 4 % differently, but at age 50, 22 % differently. In other words environmental effects were changing the effects of the genes. This in turn probably depended on details of the lifestyles. The influence of genes can therefore be affected. What way are you going to affect yours?

Which raises the question: how much are we going to go along with natural tendencies we have, and how much are we going to challenge them, if necessary all our lives?

Summary

Homosexuality is not genetically inevitable. If it were, identical twins would show 100 % concordance for SSA and no modern twin study on any behavioural 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 genetically enforced is *pairwise* concordance, which shows that a male co-twin is also homosexual only one time in nine: 11 % of the time (**Figure 22)**. This is a long way 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 (and every human being) are affected alike by genes, shared environment and random factors.

Homosexuality fits inadequately into the more complex classic twin studies model: the high ratio of heterosexuality to homosexuality in the population means homosexuality does not conform to the bell-curve model used in twin studies, making it unlike most other traits measured in twin studies.

The most recent and reliable twin studies (based on twin registers) still have large error limits, and many factors and rule violations suggest that the estimated genetic influences are too high. However, non-shared environment (the effect of random events and idiosyncratic reactions) is predominant and significant.

Twin study results tend to eliminate the effect of shared family life and upbringing, making it appear they have no effect. But they *are* present in the form of different perceptions of the common family environment by one of the twins and in rare or unusual events (random factors) that occur in families and can have an unforgettable and disproportionate impact. Remember random factors are the strongest category in twin studies.

The 22-37 % "genetic" estimate earlier in this chapter from classic twin studies is much less than the typical figure of 50 % found in studies of all other traits, and much less again than the 90 % "genetic" influence on puberty, showing that genetic programming of SSA is minimal.

Comparison with other traits showing higher genetic influences than SSA, e.g divorce, altruism, religiosity, fundamentalism, depression, extroversion, homophobia, makes it clear that homosexuality is not inevitable or fixed.

> The genetic effect of twin studies translates in real life into a weak and indirect effect for SSA. The scenario of a boy who was, e.g poor at sports, artistic and sensitive, is about the extent of the genetic effect in homosexuality. A girl might be, e.g big-breasted. The boy may be bullied, withdraw from his male peer groups and go on to see himself as gender atypical; the girl may be raped, and decide she doesn't like men or want to be a woman. The psychological effects in each case may be devastating and lead to SSA but the genetic effects which lead to it are weak and indirect.

Ultimately, it doesn't matter much whether the genetic contribution is large or small. It doesn't determine our behaviour. 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 feed them or starve them.

The battle is not really at the level of our genes. The traits we end up with may not have been consciously chosen in the past, but can be subject to our conscious choices right now.

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