10

Twin studies— the strongest evidence

Over the last decade, studies of twins have provided some of the strongest numerical evidence that "Our genes do not make us do it"— which makes this chapter probably the most important in this 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. Ten per cent 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

Twin studies

Twins have been invaluable to medical research for a long time, though sometimes in lethal 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 supporters of ethnic cleansing. Today twin studies are used constructively. By September 2013, about 50,000 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.*††

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

†† 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.

register with a projected 600,000 members is being organised, but a few registers already have more than 33,000 pairs of 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 twin researchers often use a different measure 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] examining 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. 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.*

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, and looks like **Figure 26**.

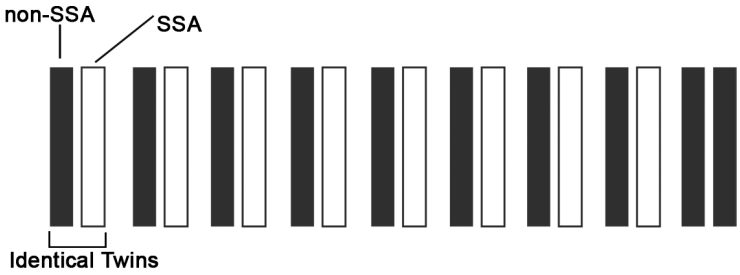

Figure 26. Concordance for SSA in identical twins is one in nine—11%

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 27** shows 100 hypothetical twin pairs taken from a twin registry. Of those 200 individuals only 4 (roughly 2-3% of them) [shown by the grey squares] have SSA, but all are discordant. There are not enough pairs to show the rarer pairs *both* of whom have SSA and are therefore concordant.

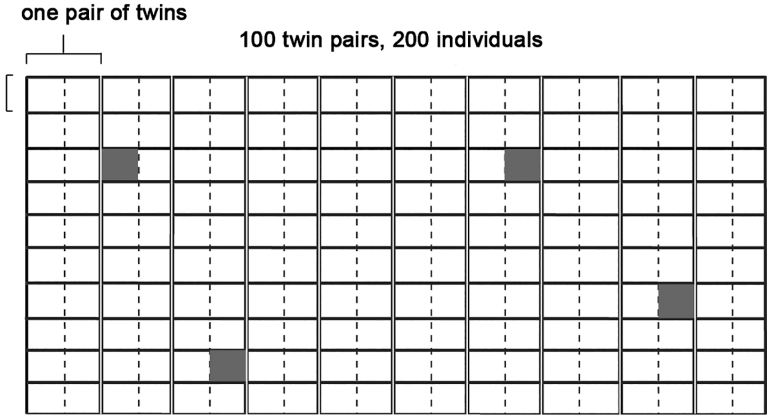

Figure 27. Occurrence of SSA in twins

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 26** 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, on this basis, 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 **Figures 26-27**

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 26**), 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.

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 diverted attention from the individual erratic factors.

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‡‡ In 2002 Bearman and Brueckner[4] 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).[1] But in Chapter Twelve we show that SSA adolescents are a special case—generally changing their attractions from year to year.

SSA concordance compared with concordance for other conditions/traits

In **Figure 28** 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.

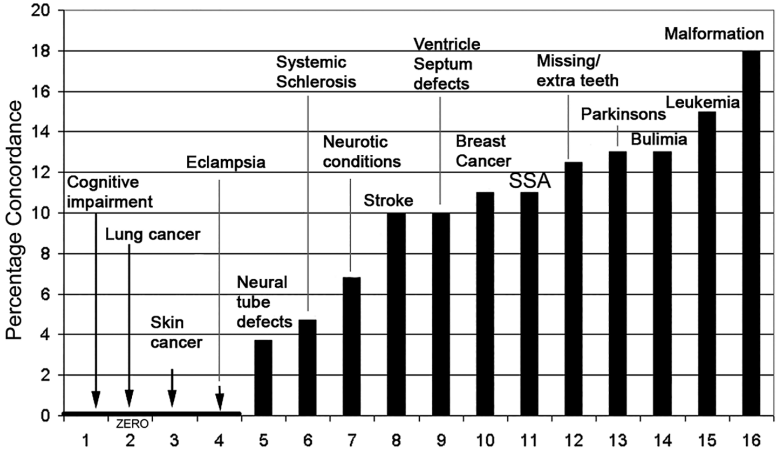

Figure 28. Some low pairwise concordances for identical twins. (Taken from PubMed.) The male figure is given for SSA
1. Cognitive impairment[6] 2. Lung Cancer[7] 3. Skin cancer[8] 4. Eclampsia[9] 5. Neural tube defects[10] 6. Systemic sclerosis[11] 7. Neurotic conditions[12] 8. Stroke[13] 9. Ventricle septum defects[14] 10. Breast Cancer[15] 11. SSA[1] 12. Missing/extra teeth[16] 13. Parkinson's[17] 14. Bulimia1[8] 15. Leukemia[19] 16. Malformation[20]

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, 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.

Left-handedness has a 13.5% concordance similar to that of SSA. Left-handedness is still overwhelmingly due to non-shared environment—chance.[22] This book proposes the same for SSA.

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 single 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. But, Sir Michael Rutter says "[poor] penetrance is not very usual for single gene effects."[21] A check of the largest database on penetrance showed that this might happen for a single gene in about 10% of cases only. 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 p157 on), which is considerably higher than the 11% result.

It is also salutary 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 say this is 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. A representative view is held by therapist Oliver James: *Twin Studies: a Discredited Method*.[46] 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.

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.

Epigenetic effects are another random factor.[45]

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 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 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 impress 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.[49] (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

SSA development is not a great mystery. 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 29** 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. This also meant they thought that deliberate choice was not a factor.

A study by Herek et al.[5] found 88% of gay people thought no choice was involved.

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]

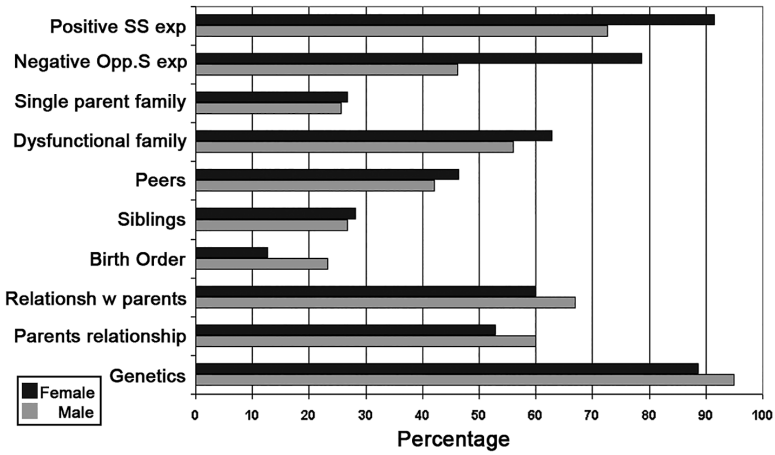

Figure 29. Factors thought by gay and lesbian people to have had some causal connection to their SSA

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 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: (18 factors)

- Artistic predisposition (men, particularly if allied to poor sports coordination)
- Auto-immune thyroid condition in mother
- 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 was believed to be a modest genetic link between homosexuality and left-handedness)
- 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)
- Teenage pregnancy (? hormonal influence)
- 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 (49 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 affecting 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)
- Mental problems in the home
- 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)
- Sibling same-sex incest
- 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)
- Teenage pregnancy (negative reaction to men or femininity?)
- 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.*

Classical Twin Studies

The previous material presents the conclusions of twin studies in a rather intuitive way. When done more mathematically, both identical and non identical twins are involved and the method produces three

numbers; percentage contribution from genetics (common prenatal factors); family environment; and factors which affect even identical twins differently.

Estimation of the genetic percentage for heterosexuality is difficult because of mathematical problems, but Hershberger[27] found a result of 18-26% which is much lower than one would expect. SSA should be about the same or lower. Whitehead[66] found a similarly low result from considerations about the timing of puberty.

For homosexuality, the last half dozen twin studies (1998-2013)[1,3,26,28,29,30,31] are the most important. These large studies were done using the twin registers and when volunteers signed up they didn't know they would be asked about SSA. This should greatly diminish bias, but even on a twin-register, twins have to agree to take part in a given study, and we don't know exactly what effect this has.

Researchers used a variety of measures of SSA. However we found that different measures did not affect the final percentage conclusions. These were: the mean genetic fraction for adult men is (22±20)% and for women (37±18)% (the errors are the standard deviation of the mean). The percentages are weak to modest, and the errors are large. Another important conclusion is that the "nonshared environment"/random percentage is always larger than the "genetic fraction" and has much smaller errors on it. The non-shared influences were 78% and 63%.

The Bearman and Brueckner adolescent twin study[4] was not included, but is very important. It was a very large study but had a calculated 0% genetic contribution to SSA. The implication 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 is very different from the genetically programmed events of puberty which appear in twin studies to be about 90% genetic.[32] The degree of genetic programming must be very low for SSA compared with puberty.

The technical details of these studies are discussed elsewhere[48] and depend on many assumptions, almost all of which overestimate the genetic percentage. In general though, when analysed in detail, there is still some real genetic percentage, though we estimate it could be about 10% for both men and women.

The meaning of a 20-37% genetic contribution

Let's be generous and grant that the genetic proportion of influence on SSA might be 20-37%. What does that mean? Does a 20% genetic figure dictate behaviour anyway? Certainly not.

Church attendance is also close to 20% "genetic."[34] 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% "genetic" category are divorce,[35] depression,[36] altruism,[37] religiosity,[38] fundamentalism,[33] psychological inpatient care,[39] fear of the unknown,[40] perhaps alcoholism,[41] and most interestingly homophobia!![42] 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.[43]

"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 nonshared 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, and 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.[44] 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. Which raises the question: how much are we going to go along with natural tendencies and how much are we going to challenge them?

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 26).** This is a long way from genetic determinism.

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 strongly suggest that the estimated genetic influences are too high. In any case, 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 each of the twins and in the form of those 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 from classic twin studies is much less than the typical figure of 50% found in classic 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., slight of build, poor at sports, artistic and sensitive, is an example of the kind of role the genetic effect might play in male homosexuality. The boy may be bullied, withdraw from his male peer groups and develop longings for connection that become eroticised. A girl might be, e.g., big-breasted. She 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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