My Genes Made Me Do It!

Homosexuality and the Scientific Evidence

NE AND BK WHITEHEAD
Scientists discover the gene for cable knit
Introduction

*My Genes Made Me Do It!* (the title is facetious) is an attempt to place in the public arena the scientific facts about homosexuality—particularly the information that the homosexual orientation is not inborn or hard-wired, and that sexual orientation can naturally undergo huge change.

The West has been subject to such a campaign of misinformation and disinformation in the last 20-30 years that its public institutions, from legislatures and judiciaries to the church and mental health professions widely believe that the homosexual orientation is innate—in the sense of biologically imprinted—and therefore unchangeable.

The implications of this are that anyone who makes the following scientifically true statements is considered to be the one who is misinformed.

- sexual orientation is not inborn but develops over some years in response to an individual’s response to life events—as many human predicaments do
- homosexual orientation can change, i.e., half the homosexual population naturally moves towards heterosexuality over time (without any therapeutic interventions), and further and faster with counselling and support
- The same-sex-attracted are not 10% of the population but (including bisexuals) much closer to 2.5%.

The West has lost its way on this issue, and today we are seeing the outcome.
The mental health professions

In the West now, mental health professionals in many jurisdictions are unable to offer “reparative therapy” for people with unwanted same-sex attraction. They are often under policy constraints to counsel clients towards acceptance of their sexuality.

The American Psychological Association (APA), which tends to set the trends in mental health policies in the West, has been under unremitting pressure for years to ban reorientation therapy for people with unwanted homosexuality. It tends to rubber-stamp its Gay and Lesbian Task Force reports and in 2009 endorsed an assessment of sexual reorientation therapy rejecting it as probably harmful and change as dubious. The Task Force making the evaluation was comprised of activists in gay causes, most themselves publicly identified as gay. Every practitioner of sexual reorientation therapy (at least five highly qualified people) applying for inclusion on the committee was rejected by the APA’s President Brehm.*

The report applied ridiculously high standards of proof that re-orientation therapy worked—standards not required of any other therapy. In its determination to show that change could not occur the Task Force ignored the psychological literature showing evidence of a great deal of change.

Nonetheless, other professional organisations follow suit with little appreciation that the APA stance on homosexuality is political, and not scientifically grounded.

The judiciary

In the judiciary, homosexuality has steadily gained status as an “immutable characteristic” (like skin colour and gender) so that it has become widely unconstitutional in many countries to discriminate against it in any way—with the inevitable result that it also becomes unconstitutional to withhold marriage licences. Marriage is no longer distinctively a contract between a man and a woman able to naturally procreate.

The churches

The crises in the Roman Catholic and Anglican denominations are the outcome of the stance that homosexuality is something that is innate and impossible to change. The media have presented the sexual abuse by priests of children as pedophilia; we have rarely heard the word “homosexuality.” But 99% of the abuse has been against young men past the age of puberty; in other words the crisis is not about a few errant priests who have molested children, but about priests with a homosexual orientation who have sought sexual connection with post-pubertal males.

The Roman Catholic church has a significant amount of homosexuality in its priesthood (we estimate about 10%; much higher than in the general population), but though it counsels celibacy in its priests, it is only beginning to appreciate the scale of the crisis, i.e., just how many homosexual priests have sought refuge in its ranks, and the effects of a policy that fails to take account of the extent to which sexual orientation can change. Rather, priests are expected to be celibate.

The Anglican communion has gone further than the Roman Catholics, particularly in the USA and Canada, where the denomination has divided so thoroughly over the ordination of gay bishops and priests and the sacrament of marriage for practicing gay couples, that some of the faithful are placing themselves under foreign bishops, while gay and gay-friendly US bishops and clergy refuse to back down. Merciful men like Archbishop Desmond Tutu have been caught in the falsehood. The Archbishop equates homosexuality with skin colour and asks, therefore, why we don’t want homosexuals “to give expression to their sexuality in loving acts?”, since “it is becoming increasingly clear they can do little about [their sexual orientation].” These attitudes naturally filter down to people in the pews, whose opinions are already shaped by the misinformation that homosexuality is “genetic” and that 10% of the population is gay. They will also quote the attitude of Christ who is inclusive and loves all men and women. Compassion is better than judgmentalism, and anything but full acceptance is judgmentalism and homophobia. Ordination of practicing gays becomes the compassionate act. This view is also increasingly held in other denominations.
Governments

Governments with strong social agendas have been both victim to misinformation and propagators of it, so the idea that homosexuality is just another minority category that needs special protections now runs wide and deep in Western Governments. Political debates don't even consider the scientific facts. Few politicians would give alcoholics or the obese or gamblers special protections in law because they realise these particular personal difficulties can be overcome. Homosexuality belongs far more in this company than in that of skin colour or gender, but that is not generally known or believed.

The United Nations

In the UN the pressure is on non-Western governments by Western representatives to globally end discrimination against gays. The message: all member states must pass legislation enshrining homosexuality as a human right in all cultures. The declaration is nonbinding, but has been signed by at least 66 countries, most of them Western, and the pressure will continue. The debate, driven by gay activism and its backers in high places takes it for granted that the issue is one of a large minority denied the right to protection for something as basic and unchangeable as eye colour. This is not the truth: homosexuals (including bisexuals) are a tiny proportion of Western populations with a condition as responsive as many other human disorders to support and good counselling, the will to change and hard work. In this middle ground there is still plenty of room to make sure people with a homosexual orientation are protected from the malicious and bigoted.

The media

The media, under pressure to condense information to soundbites and headlines, or more often because it is frequently a purveyor of information passed on by special-interest groups seeking publicity, often gives the public skewed facts. Usually (in our experience) the science is misrepresented. We're left with a headline that says something like, “Gay gene discovered,” or “Genetic basis to homosexuality,” or “Homosexuality found in rams.” Any reputable geneticist begins to cry! But most of
the rest of us make a mental adjustment of sorts—“Well, I guess if it’s genetic there’s not much they can do about it… and if animals do it too, then it must be just a natural part of life.”

“Most of the rest of us” are Mr and Ms Average Citizen, and the people in the preceding paragraphs: the bishops, clergy, laity; members of the judiciary, politicians, psychotherapists, counsellors, teachers, state servants, community leaders, parents. We are not specialists in homosexuality. We are busy people who often only have time to scan the headlines, or absorb the first couple of sentences on the TV news, or follow the policy directions from head office.

**Education**

Students are increasingly provided with counselling support if they believe they might be homosexual. This has come in response to pressure from policy makers and the gay lobby to protect “homosexual” students at school. But it is not generally understood that almost all 16-year-olds who think they are homosexual now will, one year later, believe just as firmly, that they are heterosexual and in fact go on to develop heterosexually. Some will become homosexual, but to offer gay-affirmative counselling and contacts to teenagers finding their way through the wobbly process of acquiring stable heterosexual gender identity is a stumbling block to acquiring it rather than a stepping stone. Children showing evidence of GID (Gender Identity Disorder, now known as Gender Dysphoria) and parents of these children can instead be offered solutions to recognise and resolve contributing factors rather than affirm what is possibly a developing homosexual orientation.

**The gay community**

In the gay community itself more than 90% of gays now believe genes are a significant factor in their orientation—a ten-fold increase in fifty years.† Few people know enough to tell them differently. And because of the current climate in the psychological and counselling professions few know how to help them change if they want to. The only other path left is the fight for equal freedoms—and Western human rights-focussed

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governments are easy prey to gay activist assertions that they are a minority with innate and immutable characteristics that need special protections.

So much of what people in the West believe about homosexuality now, is not the truth. The blind are leading the blind. It suits some people to believe what they do, but many others genuinely don’t know what to believe and would welcome the truth if they only knew where to find it. Here is a very basic piece of truth. There is nothing fixed or final about the homosexual orientation and its natural expression—homosexual behaviour. No politician, church leader or member, judge, teacher or counsellor, or homosexual person, or friend or family of a homosexual person, needs to feel forced into a position on homosexuality based on the apparent immutability of the homosexual orientation. Homosexuality is not inborn, not genetically dictated. Nor for that matter is heterosexuality or any other human behaviour. In fact our genes do not make us do anything. Whether it’s homosexuality, a foul temper, bed-wetting or addiction to chocolate, our genes have very little to do with it.

*Any genetic influence is weak and indirect*

In human behaviour everything is nature and nurture. Without genes you can’t act in the environment at all, and without the environment your genes have nothing to act on. No behaviour, including homosexuality, results solely from genes. At least for homosexuality this book argues that the level of genetic influence could easily be as low as 10%, the balance of 90% coming from the environment. And that 10% is not a direct genetic influence. Every human being has a 10% genetic influence on behaviour. A man or woman may have long fingers but that doesn’t make him a player of Liszt. If it did, we might say there was a genetic influence on his choice to take up piano and play Liszt. A man may have compact build and good co-ordination but that doesn’t make him another Roger Federer. If it does, we might say there was a genetic influence on his choice to follow in Federer’s footsteps. In homosexuality the genetic factor can be any physical characteristic that might make a man or woman feel gender atypical. But many people with SSA have none of these.
For other human behaviours genetic influence may be as high as 50%, but nothing about that is fated either. Probably the best tool for measuring genetic influence on any behaviour (studies of twins) makes it quite clear that the genetic content of any behaviour drops commensurately with whatever environmental interventions of an opposite kind are brought to bear upon it. In other words, even if homosexuality did have a genetic content of 50%, opposite environmental influences could almost nullify it.

In accounts of genetics or social environment and SSA, you will often find statements that the link is weak, or moderate or strong. This is often misleading. Physicists may say a link within physics is strong, but when sociologists say it is strong, the physicists would say, “Rubbish, that’s weak!” Difference disciplines use different standards, and “soft” sciences have a low one.

In this book a more objective standard is used which relies on how strong the effect is (in statistical terms the percentage of variance explained). Total domination by an effect would be near 100%; moderate influence would be 50% and weak, down near zero —perhaps 10-20%. By this standard almost all sociological influences are weak to modest, so inevitably this book will say the same; nothing is a strong, overriding, and universally applicable influence in the development of SSA.

*My Genes Made Me Do It!* attempts to bring scientific objectivity into the debate about homosexual orientation and its many implications. In the following pages you will read what orthodox science tells us about homosexuality, and you can draw your own conclusions. Don’t let the numerous references persuade you that this book is for academics and scientists only. The references are listed for those who want to refer to the original research but the text is accessible to the average reader.

Because the scientific evidence so clearly shows sexual orientation can and does change we dedicate this book to those heroic people who, against a strong tide of Western public opinion, have found the courage to change their sexual orientation.

This is the 6th edition of *My Genes Made Me Do It!*, the first was published 20 years ago. The years since have only strengthened the book’s original conclusions. Although there have now been many studies of biological factors none has come close to showing an overwhelming influence on homosexuality. Twin studies, in particular—which provide
the best quantitative estimate of the genetic contribution—have continued over the last decade to lower their estimates of genetic input into homosexuality. In addition, recent work on the role of histones (Chapter One) in gene expression hints at a much greater environmental role than twin studies have factored in. The first edition of *My Genes Made Me Do It!* suggested a figure of 10% genetic influence, both weak and indirect. Nothing has happened over the period to make us alter that view. This edition further emphasises the role of the predominant random factors, including some indirect random genetic factors. It also contains quite a number of new arguments not used elsewhere. Almost all have now been published in peer-reviewed journals.
About Us

Our research into homosexuality started in 1987 when we met a married man, who told us he had been a promiscuous gay man for more than 20 years and a gay political activist for more than half that time. We got to know him and his (female) wife. He introduced us to the news that gays didn’t always stay gay, and to other same-sex attracted people who were on a similar journey out of the lifestyle and orientation.

Six years later the first of our several books on homosexuality was published. *Craving for Love*, by Briar Whitehead (Monarch UK, 1993, 2003, www.cravingforlove.nz), interviewed scores of people with a homosexual orientation who were at varying stages in the process of change. The book looked at causes of homosexuality and the process of change. The second was a submission to a New Zealand Government Select Committee during the passage of gay rights legislation. It defended the rights of gays to change their orientation if they wished; gay activism had intended to use the legislation to make assisted change of orientation a discriminatory act.

This, the 6th updated edition of *My Genes Made Me Do It!*, is the result of a 30 year review of more than 10,000 papers from all sides of the debate: scientific, sociological and psychological, including those written by gay scientists hoping to find a genetic or biological basis to homosexuality. The first edition of the book was published in the USA in 1999.

Neil Whitehead (PhD) biochemistry) has worked for 40 years as a research scientist in New Zealand and around the world. Briar Whitehead is a journalist, writer and editor of this edition of *My Genes Made Me Do It!*
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If I really wanted to get to know you, would it help if you offered me an analysis of your DNA? Or a chunk of your cellular fat and carbohydrate? Would an understanding of the way your genes produced the protein in your fingernails help me figure out why you bite them when you’re nervous? Would the configuration of the nitrogenous bases in your DNA help me understand why you have a preference for cordon bleu on Saturdays? Is it the chemistry of the paint that makes a Rembrandt Self Portrait what it is? Is it vibrational physics that makes Beethoven’s Symphony No 7 so magnificent?

We could argue that the chemistry of paint and vibrational physics adds something to the portrait and the symphony. But most of us would say they don’t have much to do with it.

Mainstream geneticists react in much the same way when people try to argue human behaviour—particularly, for the purposes of this book, homosexual behaviour—is dictated by genes. For the geneticists the argument was settled 30 years ago. Almost every behaviour is both nature and nurture. Rather frustrated, geneticists mutter, “What are
these activists doing, trying to turn back the clock and argue homosexuality is only genetic?!”

Sir Michael Rutter in his book *Genes and Behaviour* says,

Any dispassionate but critical review of the research leads to the clear conclusion that there are substantial genetic and environmental effects on almost all types of behaviour and all forms of psychopathology or mental disorder… None of the findings are in the least bit compatible with a genetically deterministic view.\(^{21}\)

However this book will argue that any genetic influences on homosexuality are weak and indirect and about 10% of total effects. (Everybody has at least that level of genetic content to their behaviour; without genes no human behaviour of any kind is possible at all.) It will also say that of the environmental influences on homosexuality, chance—an individual’s reaction to random life events—is the strongest. By reaction we mean a reaction that starts to become habitual, structuring itself into the personality, leading to homosexual responses.

We shall frequently call homosexuality “SSA” (Same-Sex Attraction) and heterosexuality “OSA” (Opposite-Sex Attraction). SSA is more appropriate because homosexuality is not sexual in origin, though can become so in practice. Same-Sex Attraction more accurately expresses this strong connection to people of the same gender.

In this first chapter we will argue that SSA is too common to be dependent on a single gene or its mutation, or even many genes. Similarly it is too common to be a biological developmental error, but could plausibly be a psychological trait. For all of us—homosexual or not—genetic structure and function only hint at the people we ultimately can become. They have very little to do with our sexuality.

**Some fundamentals of genetics**

But first, let’s visit the nucleus of a single human cell for a moment and look at some of the fundamentals of genetics.

If we pick any nucleus at random from one of the cells in our bodies about to divide, almost all of us will find forty-six chromosomes inside. Each chromosome is made up of one strand of deoxyribonucleic acid (DNA) highly-folded, and made up of an extraordinary twisted ladder
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of 60 to 185 million rungs depending on the chromosome (Figure 1). If you joined, end to end, each unfolded, untwisted chromosome in a single cell you’d have about three billion rungs. That’s a lot of rungs! If you climbed each rung at the rate of two a second, sixteen hours a day, you would spend your whole lifetime getting to the top, and at the end of it you would only have climbed your own height in DNA. Any molecule as long as that is not stable in water and is always breaking spontaneously. So there is an army of enzymes constantly repairing it in many places, like groups of engineers with sandbags on a dyke threatened by flood-waters.

**Figure 1.** *Left:* Double stranded DNA molecule. Missing from the outside on each strand are phosphate groups. *Right:* On a much larger scale the molecule is curled round protein globes called histones. (More on histones later in the chapter. The highly folded DNA on the right occurs only during cell division.)
DNA in several ways is a marvellous measure of what you are. Fearfully and wonderfully made? We haven’t seen more than a glimpse so far!

Groups of the rungs on a single strand comprise what we call genes. Genes are typically anything from 1600 to 4000 rungs long. Scientists estimate everyone has 22,000 genes. The collection of genes for an organism is called its genome. The process of finding genes was so well established by 2006 that it was possible to catalogue all the genes in one small bacterium in only four hours. The minimum number of genes for a viable scientifically-designed cell was estimated to be 256. The largest was of the minute Amoeba dubia which is about 200 times the size of the human genome. By 2010 it was even possible to make a simple synthetic DNA capable of making a bacterial cell function and reproduce. One paper mentioned genomes on 178 species of bacteria which live on or in humans. So the analysis of the human genome was only a first step. Now, even a Neanderthal genome has been analysed and those of many hundreds of lesser animals.

There are some exceptionally large genes, particularly for the protein titin, which is 50,000 rungs long, and forms a molecule which, like a spring, pulls back a muscle fibre after it is stretched.

There are whole families of genes which act as back-ups for each other.

However about 90% of the spiral ladder contains no genes. There had been some puzzlement about the function of these “waste” stretches of DNA but by 2015 researchers had shown even they had an important function as regulators of gene function.

The rungs of the DNA ladder are actually chemical bonds between “nitrogenous bases” at the ends of the rungs. These bases are various combinations of carbon, nitrogen, oxygen, and hydrogen, and look something like a rather skewed infinity symbol. Yes, infinity is in your DNA! In DNA, there are only four bases, each with exotic names. For the sake of simplicity let’s call them letters. (A and T) thymine and adenine always join together to form one type of rung, and (G and C) guanine and cytosine always form the other type of rung. One rung might be adenine and thymine (AT) and the next rung the same again, or thymine and adenine (TA), or cytosine and guanine (CG), or guanine and cytosine (GC). (Adenine appears to be the basis of one compound which
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makes us desperate to sleep. We hope this account won’t!) The arrange-
ment is shown in Figure 1. The ladder sides, between the rungs, are
sugars! The number and sequence of letters on one strand of the DNA
ladder represent special coded information which determines the trans-
fer of hereditary information from one generation of cells to the next
and from one generation of humans to the next. The entire chromo-
some is made up of 64 different 3-letter sequences of code all of which
can be reduced to a table taking up less than half a page in a textbook.
These 3-letter sequences would correspond to one amino acid (a small
component of protein). The biochemical machinery in the nucleus
also makes a copy of the gene: a secondary, smaller, slightly different
and more mobile piece of nucleic acid called ribonucleic acid (RNA),
which is transferred out of the cell nucleus into the “body” of the cell
where more biochemical machinery then uses it as a template to make
specific proteins. Complicating it still further, some of the RNA in many
species, can pass on some information from generation to generation
independent of DNA, within the nucleus and also the mitochondria,
the little energy-producing organelles within the cell.

What the gene really does

If it’s not clear already let’s spell it out! The gene’s function is biochem-
ical. The DNA contains genetic coding that spells out the instructions
for making (mostly) proteins: usually one gene for one protein. In fact,
the process DNA—>RNA—>Protein is so basic to genetics that it has
been called the Central Dogma of biochemistry, and likened to a kind
of cellular software. Proteins are made up of various combinations of
about twenty little molecules, called amino acids. Each group of three
bases (letters) on the ladder is a code specifying one individual amino
acid which should link with other amino acids, similarly produced, to
form a protein. For example, the triplet GTA codes for the amino acid
histidine, while GTT codes for glutamine. The sequence, types, and
numbers of amino acids largely determine the nature of the proteins.

With a process as complex as this it is not surprising that errors
happen. One third of routinely produced proteins contain errors, and
are immediately broken down and recycled. This may be because they
have been folded into an incorrect three dimensional shape rather than
the correct one—many of these incorrect shapes are toxic to the cell.
We could sum this up crudely and rather incorrectly, by saying “genes make proteins, not (sexual) preferences.” (Actually they are only recipes for proteins, and don’t do the work themselves.)

If the DNA is correctly “read” and its recipe precisely followed, the “right” proteins will be produced in the cell and the gene will have been “expressed.” If, however, the process is blocked, either through biological accident or through normal feedback mechanisms at higher levels, the gene is said to have been “repressed.” In simple organisms, most genes are expressed, but, in complex organisms, only about 10-15% are expressed in any one organ. For example, genes coding for proteins involved in the development and function of the eye will be repressed in cells in the region of the toenail. The pattern of proteins produced depends on the pattern of repression.

Some of the proteins are also enzymes. They act as catalysts in chemical reactions producing more proteins, carbohydrates, and lipids (fats) from smaller components, i.e., from amino acids, simple sugars (such as glucose), and fatty acids, or they break larger molecules to smaller ones. This means far more than just 22,000 unique proteins are produced; estimates range from 200,000, to as high as a few million, and perhaps one tenth of those in a single type of cell.

Biochemists themselves rarely appreciate how complex a single cell is. To use a metaphor: one single fertilised ovum, for example, resembles a vast plain crammed with about a billion dancing figures on a complex grid, either spinning alone or briefly forming long chains or small groups or circles, only to break away and form thousands of others. There are about one billion biochemical reactions each second (plus or minus a factor of ten) within this single cell—a dazzlingly complex mesh of actions, interactions, reactions, feedback and control paths, and cooperation and interference, causing thousands of genes, and all the gene products within the cell, to interact. More than 100 trillion other cells in this potential human body have yet to develop in the same way and begin to interact with each other in this extraordinary dance of life.

* This was calculated from the energy used by a typical cell compared with the energy of a typical chemical bond.
Is behaviour genetic?
The standard genetic model is that behaviour is both nature and nurture, but a few people argue that genetic function goes much further. Sociobiologists particularly, hold that all human behaviour is genetically predestined, coded into the genes. Some researchers have sought to find a link between genes and SSA. We’ll look in detail at some of these arguments in later chapters, but right now let’s continue to look at basic genetics and see what general statements can be made about genetic influence and determinism in relation to sexual behaviour.

No gene can do anything by itself
“Researcher finds gay gene” was the way the media headlined the news of American geneticist Dean Hamer’s claim to have found a link between genetics and homosexuality in 1993 (Chapter Nine). But that’s not what Dean Hamer was claiming, at least publicly. Hamer said: “We have not found the gene—which we don’t think exists—for sexual orientation.”
Hamer knew that any attempt to argue the existence of a “homosexual gene”—a single, apparently autocratic, gene governing homosexuality—is nonsense, genetically. There is no single gene governing sexual preference or any other preference. There is no gene for smoking, dancing, or making sarcastic remarks.

Why is this so? Because, for a gene to even be expressed, it has to be acted upon by the products of another expressed gene or genes. It probably takes combinations of products from at least five separate genes, and sometimes as many as twenty separate genes, to activate a single gene in a single cell into expressing itself. The products may come from some obscure part of the molecular dance or sometimes from outside the cell. No gene is an island—it interacts with other genes. In this biochemical ecology it is almost impossible for any one gene, or a minor combination of genes to completely control all the others, though a small group of genes does determine (usually) the body form and organisation of organs in the body and the expression of all other genes during development. The simple world of monk Gregor Mendel and his peas—in which single traits like tallness, colour and seed shape are each determined by a single gene is almost never seen in human genetics. One paper found 567 interactions between 268 of the genes in yeast. How many would there have been for the whole genome? It is quite possible
the complexity is too great for humans to grasp. Hamer would have been happier if he had found several interacting genes. It is very unlikely that a single gene is responsible for SSA.

Could SSA be a result of sudden mutation?

It’s highly unlikely the gay community or geneticists would accept such an explanation, but from a biological point of view, could SSA possibly be the result of a mutation?

What causes a mutation? It can be something as simple as one wrong DNA triplet code in a critical place. The effect might be like a plane crashing in the middle of the group of dancers. They may form new circles and groups to try to compensate for the deaths of their companions, but things will never be the same again, even though the cells contain several enzymic mechanisms for repair which work quite effectively.

But if many genes are involved, many genes would have to mutate simultaneously, which is so unlikely that no geneticist would accept it happens under natural conditions. If we argue instead that there could be a mutation in a single one of the critical basic control genes, homosexuality is far too common in the population to fit such a hypothesis. (See later in this chapter.)

There are many conditions now known to scientists that have been traced to specific single gene locations or chromosome faults: muscular dystrophy, familial colon cancer, Huntington’s disease, cystic fibrosis, sickle cell anemia, Down’s syndrome, hemochromatosis (abnormally high storage of iron from the diet), multiple exotoses (a disorder of cartilage and bone), haemophilia, polycystic kidney disease, Lou Gehrig’s disease (fatal degenerative nerve ailment), and neurofibromatosis. These are physical conditions resulting from breakdown of biological processes, or faults in genes. They are not behaviours, though distinctive behaviours may result from them — as in Down’s syndrome (“simple” behaviour). There are known to be more than 10,000 gene effects due to mutation in the human organism — most of them creating the kinds of physical defects just mentioned and with the availability of the human genome pattern that number is growing fast. But attempts by scientists to pin specific behaviours down to single gene defects or specific genes are proving very difficult and often unproductive. The suggested genetic
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links to behaviour usually only link to negative behaviours such as schizophrenia (see Chapter 9), and many of the findings have been retracted in the face of the repeated failure of further independent laboratory tests.

Let's look at one of the of the most direct results of mutation on human behaviour known so far and examine the implications. It's a rare condition associated with aggression, in a study of Dutch men, and is probably an example of the maximum genetic contribution to a behaviour you are likely to see. People without the condition have an enzyme in their bodies called monoamine oxidase A, which performs a simple oxidation of basic compounds called amines. Dutch men affected with the syndrome completely lack the active enzyme, because a genetic mutation has made a minor change of one of the amino acids making up the enzyme. The defective gene is passed on by the mother. Alleged behaviour results include aggression, arson, attempted rape, and exhibitionism, behaviours that were described as “disturbed regulation of impulsive aggression.” The aggressive behaviour in the Dutch men varied greatly over time and in type, and—according to the authors—could have been linked to levels of fear, anger, and frustration, possibly related to the borderline mental retardation that is part of the syndrome. Experiments with drugs to specifically inhibit the production of this enzyme in depressed but otherwise normal adults who usually produce it, raised levels of aggression (“mania” or “hypomania”) by 65% in the subjects, but aggression also rose by 50% in those who took the placebo. So we have to say although this created a tendency, it was not very strong. Also, the condition arising from the mutation was easily controllable: after counselling the Dutch men were able to lead virtually normal lives and their antisocial behaviour almost disappeared. The variation in behaviour, the dubious rise in aggression levels despite inhibition of the enzyme, and changes after counselling disprove a genetically dictated aggression.

So—to summarise:

One of the most closely genetically-linked human behaviours known to science is only weakly influenced.

Even if behaviours are linked to genes, environmental interventions (e.g., diet, counselling) can greatly modify or even eliminate the behaviour (Chapter Ten).
As Plomin remarks,

If a certain form of psychopathology should be caused primarily by genes it might be mistakenly assumed that psychotherapy and other environmental intervention would be useless. This pessimistic point of view is simply wrong.\textsuperscript{6}

\textbf{Percentage of SSA too high to be a mutation}

There is another reason SSA cannot be caused by a mutation in a single gene. The occurrence of homosexuality is too high (see Figure 2).\textsuperscript{†} In each genetic disorder from a mutation, only a very small proportion of the population is affected, in each case, about 0.025\% at most. All conditions combined affect only about 1\% of the total population.\textsuperscript{9} Homosexuality, at 2.4\% of the population does not fit into the category of genetic disorders or epigenetic effects because its occurrence is 90

\begin{figure}[h]
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\caption{Percentage of population with genetic disorders compared with homosexuality}
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\textsuperscript{†} Typical data taken from PEDINFO on the internet at http://w3.ihl.uab.edu in 1999, and verified from another source in 2010).
times too high. (Epigenetic means alteration of genetic expression by outside influences, this expression sometimes being passed on to the next generation.) So SSA does not seem to be a mutation.

Angelman and Prader-Willi syndromes are examples of epigenetic alterations which are discussed in the next section.

Any behaviour links are with many genes

If we’re going to argue that human sexual behaviour is dictated, or influenced, by genes in any way, then many genes are involved. But the “many genes” hypothesis doesn’t explain homosexuality either because as we’ll see, it changes too fast from generation to generation.

In very simple organisms, one or two genes do govern simple behaviours. Researchers found that when certain genes were repressed or disabled in some way in an offspring, a certain behaviour suddenly disappeared. For example, the sandhopper’s feeding behaviour is dependent on a single gene which produces an enzyme that breaks down complex sugars into simple, sweet sugars. This single gene can appear in several forms in sandhoppers each form producing a different enzyme, breaking down different complex sugars. So, different sandhoppers have different favourite foods because they go for different complex sugars. But, if the gene producing that particular enzyme is disabled or repressed in the offspring of a particular sandhopper, that generation is no longer interested in its parents’ favourite food.4

It is a genetic truism that if simple organisms in selective breeding experiments lose in the next couple of generations a clearly defined, consistent behaviour, then that behaviour can be said to be governed by a gene or perhaps a few genes. The same is true if the gene/genes is/are expressed or restored in the organism in the next couple of generations, and the behaviour returns.

This means the opposite is also true: if a behaviour changes slowly and steadily over many generations (as in selective breeding for example), then, many genes are responsible.

One of the longest studies on mammalian behaviour ever undertaken was done on thirty generations of mice.5 Thirty mice generations is equivalent to about 1000 years of human lineage. The mice were deliberately bred to create two strains of behaviour: activity and passivity, tested by aversion to space and intense light. Those that reacted positively
(exploring the space) were active, those that didn't react so strongly were passive. Active (exploratory) mice were then mated with active, and passive with passive, and the offspring re-tested. What happened was a slow, steady and gradual change of behaviour over 30 generations: the active mice became more active (fearless) and the passive became more passive (fearful), until they froze with fear in most circumstances. Similar results have been found in mice bred for exploratory behaviour; alcohol sensitivity, preference, and withdrawal; various types of learning; aggressiveness; and nest building. Plomin, has commented about this gradual change of behaviour: “Th[is] steady divergence…provides the best available evidence that many genes affect behaviour.” Drawing on other studies, he said that if only one or two genes had been involved, the mice would have sorted themselves abruptly into one or other of the two groups within just a few generations. Other geneticists concur with Plomin. When there are slow shifts in behaviour with each generation (as in the breeding of dogs for specific behaviours), they believe that many genes are interacting—probably many hundreds of genes—with each contributing a tiny part of the whole effect.

**Histones: interaction between genes and environment**

We mentioned that the DNA chain is wound round histones (Figure 3). Histones are unusual, extremely alkaline proteins, and it is becoming increasingly clear that they have a very important role in controlling what the genes do; in fact they are another layer of control just above the genes. For reasons not fully understood there are three major patterns of histones in all organisms from bacteria to humans. The way they act on the genes depends on the extent the histones are chemically changed by the addition or removal of acetyl and/or methyl groups, little simple clusters of atoms which are essentially acetic acid (vinegar) and methane (natural gas) though biochemists think that description far too simple.

These chemical changes are partly accomplished by cell biochemistry, and partly by diet (e.g., folic acid and the amino acid, methionine). But, significantly, the pattern of changes is also strongly affected by early social interactions—classically, for rats, by grooming by the mother. For our purposes the critical principle is that changing the histone pattern alters behaviour, and quite often dramatically.
Can genes create sexual preferences?

We mentioned above the mice bred to be either fearless or fearful in open spaces and intense light, a process that took 30 generations, and was thought to involve many genes. In recent histone experiments, offspring of these same mice were handled every day in a controlled but nurturing way by the lab technicians. Control groups of fearful and fearless mice were not handled at all. At the end of the experiment the histone patterns of handled and unhandled mice proved to be 20% different. But the interesting point was that in one generation the fearful mice that received handling became 3x as exploratory as the fearful mice that were not handled (Figure 4).

In other words, although the slow generational change in the earlier breeding experiments eventually gave rise to about a 7-fold difference between fearful and fearless mice, handling in just one generation produced a much faster and greater difference—about 10x as great. So, changes in the histones produced by handling happen very much faster and are much larger than behaviour changes produced by genetic changes in selective breeding programmes. The histone pattern has a very significant part to play in gene expression or inhibition. Although we are talking only of mice at this point, it is reasonable to assume the same process is happening in humans.

Figure 3. The effect of histone changes on the DNA. Acetylation (Ac) of the histones allows genes to be expressed, deacetylation represses genes.
Rather than a gene recipe for behaviour we are now looking at histone patterns for behaviour. This makes the whole quest for connections between particular genes and some behaviours look rather irrelevant because it is becoming increasingly clear that thousands of genes are involved in behaviours rather than hundreds.‡ The search for a responsible individual gene seems doomed.

But the most important conclusion of this research is that early social interactions in particular (and it’s reasonable to assume all sorts of life experiences) affect the histone pattern.

We are at the beginning of a large change in scientific thinking, in which histones, and how they are altered by environmental factors will be very important. Although both nature and nurture will always be involved, right now the pendulum is swinging back to environmental influence.

In Chapter Eight we will look at how histones are involved in formation of sexual behaviours in mice.

In the active/passive mice experiment there was also a control group of mice—a group that was left alone to breed randomly over the same thirty generations. What happened to that group? There was no

‡ The authors equate a 20% difference in histone patterns with effects on 20% of total genes. The human genome contains about 22,000 genes; 20% of 22,000 genes is at least 4000 genes.
significant change in behaviour. At any one time, the behaviour of those mice was about the average of the active and passive groups. As in the active/passive groups, there were no sudden random fluctuations of behaviour, as there would have been had the behaviour been controlled by only a few genes.

In a similar example, several years ago in a study published in *Nature Genetics*, scientists used two strains of fruit-fly selectively bred in opposite directions for 40 years to either prefer high flying or low ground flying. This experiment continued for 1000 generations! So it was even more extreme than the mouse experiments which were only for 30 generations. The two strains (inevitably) were called “hi5” and “lo”! Scientists were able to check about 5000 genes (about one third of the total predicted for fruit-flies) and found 250 which were significantly associated with the two different styles of flying. Rather a lot! Of the 250 they chose four to examine in detail and by transplanting them into another strain of fruit-flies and greatly magnifying the effects, proved eventually that the four genes had a small effect on high or low flying. Yes, some effect, but small.

The effects of the genes could not have been predicted from their functions. Some controlled wake-sleep patterns, and another was a “nuclear importin” which imports proteins into the nucleus of the cell.

Moving from mice to humans, the involvement of many genes is also clear if we look at human IQ. We know that many more than 100 genes are involved in human IQ because at least 100 separate gene defects are already known to individually lower IQ.6

Similarly if genes connected with heterosexual or homosexual behaviour are found there are likely to be many of them, and they will probably have cell functions only very indirectly related to homosexuality or quite irrelevant to it.

This is so widely accepted that some authors propose it is a basic law: “A typical human behavioral trait is associated with very many genetic variants, each of which accounts for a very small percentage of the behavioral variability.”25

**Implications for sexual behaviour of “many genes”**

When many genes are involved, changes in behaviour take place very slowly, over very many generations. If homosexuality is caused by many
genes how can it suddenly make an appearance in a family the way it does? Like the mice, or fruit-flies, the typical genetic pattern would be a gradual change in the family over about 30 generations from heterosexuality through bisexuality toward homosexuality—a few percent with each generation over the course of perhaps thirty generations. Similarly, homosexuality would only slowly disappear in the descendants (if any) of a homosexual person. Any other proposed mechanism is highly speculative.

Behaviours which do change slowly over the generations in a family or society are much more likely to be genetically influenced or determined, but homosexuality changes too swiftly to be genetically controlled or influenced by many genes.

How could “genetic” homosexuality maintain itself in the population?

There is another objection to the idea of a genetically produced homosexuality. A behaviour which produces fewer than average children cannot be “genetic” and also continue to exist in the population. Obviously, genetically enforced exclusive homosexuality would die out of the population in several generations.

As unlikely as it sounds, surveys show that of persons classifying themselves as exclusively homosexual, about one in three has a child. At that rate, a homosexual gene, or genes, still could not be replaced.

But 15% of male homosexuals are married (Chapter Two). Wouldn’t this preserve any homosexual gene or genes? No. Their number of children is only about typical of heterosexuals, so at 15% there aren’t enough children produced. Even including those who are divorced there aren’t enough children produced overall to replace the putative gay gene or genes. Therefore, any homosexual gene or genes would still slowly but surely breed out of the population.

Sociobiologists, almost the only group of academics who argue seriously that all human behaviour is preordained by genes, have great difficulty accounting for the persistence of SSA in the population. They try to argue that genes causing male SSA would also exist in the sisters of gays, and that the homosexual male would help ensure those genes were passed on by helping his sister and her family—e.g., babysitting, and later helping with money and resources. But these arguments are
Can genes create sexual preferences? 17

unusually weak. On average, surveys show homosexuals tend not to have close relationships with their biological families, and there is no evidence of more altruism among SSA people in cultures examined (Samoa seems to be a lone exception).

Advocates of genetic determination of SSA also argue “homosexual genes” might be preserved in the population if they were carried by women on their X chromosomes, and at the same time conferred on them special advantages in the reproduction stakes. For example these genes might tend to produce a slight physique in men—and a predisposition to homosexuality through the social effects detailed in Chapter Three—but the same genes in women would tend to produce a petite, possibly more feminine woman, more attractive to men. But this is highly speculative and sits uneasily with what little evidence we do have. Male homosexuals are often of strong physique, and mothers of homosexual males are not noticeably ultra-feminine.

A better argument would be that any genes linked with homosexuality might, be associated with less aggressive personalities. Such “sensitive” men can be attractive to women and thus have an advantage in the reproduction stakes, a difference of only a few percent being sufficient to maintain the genes in the population. But if we are arguing in favour of these imagined genes being the cause of SSA, their effects are so weak and indirect that again, we are back in the position of saying that genes do not dictate homosexuality.

Is SSA a fetal development error?

Scientists now know that genes and DNA do not exist in isolation from the environment, but that the environment influences the expression of genes, e.g., the production of the hormone adrenalin depends on threats in the environment interpreted by the brain, and signals sent to the adrenal glands which produces an almost instantaneous response from the cellular DNA. Similarly, but more indirectly, the products of many genes are copied (or not) by cell machinery in response to the body environment, i.e., the balance of other biochemicals in the blood and cells. Production of biochemicals blue-printed by DNA in response to the environment is called epigenetics, and has become an important research field.
One of the mechanisms sounds almost simple. The proteins the DNA wraps itself around are called histones, and they also affect the availability of the genes for copying. The influence of the histones is controlled by (among other modifications) the quantity of acetyl groups attached to them. The more groups attached, the more the gene activity (see p26). Epigenetics is a word that can also be used to describe a fetal pathway of development which is non-standard. These are not mutations, but accidents of development.

Could SSA be a result of an epigenetic development pathway? That seems very unlikely. Figure 5 shows many human conditions which are the result of epigenetic pathways leading to physical abnormalities. Homosexuality is not a physical abnormality. It doesn’t fit the picture. And as we found with mutations the occurrence of SSA is (five times or more) higher than any single occurrence of epigenetic abnormality, and hence is very unlikely to arise from some random or epigenetic developmental disorder before birth.

Left-handedness is often compared with homosexuality. But left handedness, similarly, is far too common, at about 10% occurrence in the population to be a fetal developmental disorder. Rather scientists believe there is a predominant post-birth random factor in its development. (See a fuller discussion in Chapter Nine).

![Figure 5](image-url)

**Figure 5.** Occurrence of pre-natal developmental disorders compared with homosexuality
Born that way?
In this section we show that SSA and OSA only develop well after birth, and compare the time-spread of their first appearance with the time-spread of events known to be under tight genetic control.

Gay activists argue that since they have “felt this way” for as long as they can remember, homosexuality must be genetic.

But 12 published surveys, show that the mean age of first same-sex attraction is 9.4 ±1.1 years for men and 11.1 ± 1.8 for women (errors are standard errors of the mean). This shows that awareness of sexual attraction to the same sex is not a typical gay person's “earliest memory.” Born that way? “Genetic”? Not on this basis.

There is some more evidence from those same surveys that SSA is quite unlike something genetic. Several surveys compare the age of first same-sex attraction with age of puberty. This is interesting because although the environment does influence age of puberty slightly, it is a good example of a genetic event caused by a cascade of gene actions, and its spread over time in the population (e.g., first appearance of pubic hair) is typical of many strongly biological events. The first event is in the brain, a part called the hypothalamus, rather than the gonads and is the production of a small protein (peptide) called (of course) KISS-1!

Probably the best age data come from Hamer et al.16 for 114 male subjects with SSA and these results, rather typical of others published, are in Figure 6 below.

The important point about the graph is that puberty is tightly clustered around age 12, and is thought to be 90% genetically influenced20 but the age of first SSA is very widely and erratically spread. It is not like a tightly enforced genetic clustering in time— something stronger is spreading the results erratically, and we suggest it is random environmental factors. It is possible using a statistical technique called “ANOVA” to approximately calculate that only about 6% of the spread of SSA ages would correspond to genetic influence. From other surveys by sexual anthropologist Whitam and others17 it may be similarly calculated for four different cultures (Brazil, the Philippines, the USA and Peru) that 3-4% of female SSA would be “genetic”—small percentages. We'll see later in the book that a variety of approaches seem to suggest 10% for an indirect genetic contribution to SSA. Opposite sex attraction as calculated from these sources, has maybe 15% genetic influence, but even
there, environmental and random factors are much more important, and “genetic influence” needs to be defined, because it is very indirect.

**Could SSA be a psychological trait?**

SSA fits much more naturally into the category of psychological disturbances and disorders which are common by comparison (Figure 7). This does not prove SSA itself is a disorder. It merely shows that it is in the realm of traits which are less and less “genetic” and physical, and more and more “psychological.”

Gay activism backs whatever current research might be useful in the campaign for gay rights, but the words of one gay activist are probably closer to the truth. The genetic argument was an “expedient lie,” he said.

In the years ahead more genetic links with behaviours will certainly be found, but in no case will these inevitably determine that one is homosexual, or brilliant, or musical, or a reader of *My Genes Made Me Do It!* Whatever you might think about your behaviour, the facts are, your genes did not make you do it. Then the real question becomes; why let them make you do it?
Can genes create sexual preferences?

**Summary**

- No mainstream geneticist is happy with the idea that genes dictate behaviour, particularly homosexual behaviour.
- Genetically dictated behaviour is something that has so far been discovered only in very simple organisms.
- From an understanding of gene structure and function there are no plausible means by which genes could inescapably force SSA or other behaviours on a person. Genes create proteins not preferences.
- No genetically determined human behaviour has yet been found. The most closely genetically-related behaviour yet discovered (aggression in Dutch males) has shown itself remarkably responsive to counselling.
- If SSA were genetically dictated, it would probably have bred itself out of the population in only several generations, and wouldn’t be around today.
- Generally, geneticists settle for some genetic influence of rather undefined degree, most agreeing that many genes (from at least five or six to many hundreds) contribute to any particular human behaviour.

- A genetically dominated SSA caused by such a cluster of genes could not suddenly appear and disappear in families the way it does. It would stay around for many generations. So SSA is not produced by many genes.

- The occurrence of SSA in the population is too frequent to be caused by a chance mutation in a single gene. So a single gene is not responsible for SSA. Nor would many genes all mutate at once.

- SSA occurs too frequently to be caused by a faulty pre-natal developmental process, so it is not innate in that sense either.

- The widespread age-range of first homosexual attraction is very unlike the narrow time-spread of genetically driven phases of human life, e.g., gestation time, puberty, menopause, making homosexuality very unlikely to be genetically driven.

- The histone system which controls genetic expression is strongly affected by the environment, e.g., nurturing, making searches for individual genes responsible for certain behaviours, mostly pointless.

- Same-sex attraction could be about 10% genetically influenced and opposite sex attraction about 15%. But this is weak and indirect, e.g., genes making a man tall don’t also necessarily produce basketball players.

- SSA falls more naturally into the category of a psychological trait.

Transcending your genes

DNA is a measure of what you are? Yes, but depending on what you do, and the choices you make, you may end up merely letting your genes define you, or totally transcending them. The staircase upwards only has its start at the genetic level.
**Animals**

At every stage between the genetic code and the mature organism, all the other influences (anything which is not the gene itself) are continuously interacting in a multitude of ways to create new and higher levels of biochemical interaction and development, each further and further removed from genetic control and less predictable from it. Genes and biochemical processes comprise the first steps. At a higher level, cells interact with each other (e.g., a macrophage cell recognises non-body cells and devours them). At a higher level still, the 250 types of cells in various organs react with one another. Higher still, the animal as a whole reacts to the environment. Probably the apex of animal development is learning from the environment. Learning is perhaps half a dozen levels up from the basic chemistry and almost independent of it.

So the influence of genes is indirect, creating an organism which has huge potential to react and change in response to the environment, but the details of that response are learned. A wild horse primed by its adrenal glands to bolt when it meets loud, fast-moving vehicles can be taught to plod through traffic without fear, and the learning is another environmental influence even more remote from the genes. Did their genes predict there would be men to train them? Of course not. So, even animals become beings which transcend their DNA because we can teach them. Monkeys can be taught a simple sign language for limited communication. Were the details of that language predictable from their DNA? No, it came from completely outside them; humans invented it and taught them.

**Humans**

Geneticists G.S. Omenn and A.G. Motulsky, when they talked about the difficulties of predicting behaviour from gene structure, said, “The hopelessness of understanding behaviour from simple analytical approaches can be compared to the hopelessness of seeking linguistic insights by a chemical analysis of a book.”

Even a mature animal cannot be entirely predicted from its genes. What of humans? Everyone has unique fingerprints, not predictable in detail from their genes. At the level of organ function genetic control is even more remote. Any genetic recipe for heart rate can go no further than prescribing a potential to respond to the environment.
The human brain is the most complex object known, even more complex than our galaxy. As one wise woman said, there is plenty of room in there for a soul! Humans are uniquely self-aware and aware of their own brains. They can write symphonies, poems, develop extraordinary concepts, speak inspirational words which move others to dream, to plan, to love and weep, to laugh, to adore. Aren’t we now talking about another dimension, of spirit? Another level? Where is DNA now? Will anyone dare say the spiritual is completely predictable from someone’s genes? Was it completely predictable from our genes at birth that we, the writers would type, in English and into a Microsoft program this sentence we are typing now? Of course not.

We start our lives forced to climb the extraordinary ladder of our genes. But we make and design the ladders we climb in our environments. Why let our genes dictate to us? Why stay at the animal level? Why not transcend our genes? Isn’t that the essence of being human? We are the ones who can take the first steps beyond them.

References

Can genes create sexual preferences?


Homosexual numbers show nurture prevails

In the eighties and early nineties, it was widely held that homosexuals were about one in ten of the population. The strongest proponents of the “one-in-ten” figure were gay activists who used it in the campaign for gay rights. Hard on the heels of the “one-in-ten” theory came the “gay is inborn” theory. The two worked together to accomplish considerable changes in attitudes of legislatures, churches, and society in general. If it can be shown that a group of people making up such a large proportion of the population is being discriminated against for something it can do very little about (like skin colour), then people will tend to accept it needs special protections.

But the one-in-ten figure is a myth, though that is still not widely appreciated. There is no significant disagreement among modern sexologists over this issue now—the early numbers (derived from the mid-century surveys of Kinsey) are far too high. We shall see that a study of the true percentage of homosexuality gives strong support to an environmentally-induced homosexuality. We shall also see that one of the largest single groups for whom the 10% figure may be true is clergy in the mainline Christian denominations.
The Kinsey surveys

So how did the “one-in-ten” myth begin? In 1948 and 1953, sex researcher Alfred Kinsey published two volumes called Sexual Behavior in the Human Male and Sexual Behavior in the Human Female. Among Kinsey’s many claims was this one: 13% of men and 7% of women in his study were more or less homosexual for “at least three years between the ages of 16 and 55.” Kinsey said the figures represented measurements of “psychologic response” and/or “homosexual experience”—that is, homosexual fantasy and same-sex contact to orgasm. The claim received huge media exposure.

Bruce Voeller, an associate professor at Rockefeller University and a non-practising homosexual, added the 13% and the 7% together and concluded that “an average of 10% of the population could be designated as Gay…As a scientist I could see how handy it was to use the 10% figure,” he said. Voeller, thereafter, became openly gay and was a founder of the modern gay activist movement. He used the figure to drive the campaign for recognition and acceptance.

As I became a national Gay leader I insisted to other Gay leaders that we needed to bring the message(s)… home to the media, to judges and legislators, to ministers and rabbis, to psychiatrists….I campaigned with Gay groups across the country for the Kinsey-based finding that “We are everywhere.” This slogan became a National Gay Taskforce leitmotiv. And the issues became key parts of (our) national, political, educational and legislative programs….After years of our educating those who inform the public and make its laws, the concept that 10% of the population is gay has become a generally accepted “fact”… the 10% figure is regularly utilized by scholars, by the press, and in government statistics. As with so many pieces of knowledge (and myth), repeated telling made it so.

The problem was that Kinsey’s figures were about four times too high.

What was wrong with Kinsey’s work?

- It did not use random sampling, which mostly post-dated him.
Kinsey had an ideological agenda. Paul Robinson, a historian and one of Kinsey’s biographers, remarks “Kinsey assigned [prominence] to masturbation and homosexuality, both of which were objects of his partiality...[He had a] tendency to conceive of the ideal sexual universe according to the homoerotic model” K5 Kinsey was bisexual and was “a cryptoreformer spending his every waking hour attempting to change the sexual mores...of the United States,” although he maintained his only motive was scientific objectivity.32 In this he was simply a profound liar. He was also a “masochist, who as he grew older pursued extreme sexuality ... by the late nineteen forties his risk-taking was becoming compulsive.”31

His research methods were probably unethical. Media commentators Reisman and Fink4 challenge the research methods that obtained claimed orgasms from hundreds of children and infants. The data are therefore quite suspect. Some of the best statistical investigators in the world—Cochran, Mosteller, Tukey—commenting on the Male and Female Reports, agreed that the procedures adopted by Kinsey and his team inflated the homosexual figures.

Modern surveys
By 2010, more than thirty surveys of homosexual occurrence were based on genuinely representative samples, mostly from Western countries.

![Surveys and Dates](image)

**Figure 8.** The percentage of bisexuality and exclusive homosexuality among western adult males
Homosexual numbers show nurture prevails (see Figures 8 to 11). The results are nowhere near 10%; they are about 2-3% including bisexuality. Included are recent Dutch figures, which are atypically high, but make almost no difference to the mean or spread of results.

The middle line in all four figures represents the mean, and the two outside lines the standard deviations, which include about two thirds of the points. Individual points have error bars which are one standard error, as estimated from the sample size.

**Figure 9.** The percentage of bisexuality and exclusive lesbianism among adult females in the West

**Figure 10.** The percentage of exclusive male adult homosexuality in (mostly) Western nations
The surveys are randomised within the study countries, and record by sexual contact people who have always been exclusively homosexual or those exclusively homosexual in activity in the twelve months before the survey. This is a rather restrictive definition, but there is little disagreement about what it represents. It is also fair, because few people identifying as homosexuals are celibate in any given year. It therefore would make little difference if the criterion was self-identification instead. Bisexuality results also used a twelve month criterion. Many studies were omitted because they were of specialised groups, were not randomised, or because the type of data in the figures could not be extracted from them.

See footnote for literature sources for Figures 8-11.

So from about 1990 to 2010 about 1% of the adult male population was exclusively homosexual, and about 0.6% of the adult female population was exclusively lesbian at any given time—a grand mean of 0.8% of the total adult population. If bisexuality is included the figure rises to 2.9±2.0% for men and 1.8±1.3% for women (the errors are standard
deviations). Around 2.4% of the total adult population is homosexual, lesbian, or bisexual. The homosexual percentage is nowhere near one in ten of the population.

**Implications for the nature/nurture debate**

The percentage of homosexuality has important implications for the nature/nature debate.

As we showed in the last chapter (Figure 2), homosexual occurrence is too high, even at only 1%, to be caused by genetic mutation. Most conditions caused by mutation each affect only about 0.025% of the population. At 2.4% the chances of a genetically driven homosexuality are even remoter. Homosexuality fits much more naturally into that group of human behaviours which are predominantly psychological in nature.

Surveys of some high-density gay areas, such as parts of San Francisco, do come up with figures about equivalent to Kinsey’s figure of 10%, so we might conclude that his research might be about right for some parts of some large metropolitan areas.

Since the year 2000, surveys have been done less by interested scientists, and more by census authorities in many countries, including Australia, Canada, and the United States. These surveys are now becoming quite predictable in their results, which are changing little. The results are consistent with those above, but often used the different criterion of self-identification, rather than behaviour.

Researchers at La Trobe University, Australia however, think that the responses of women may need further interpretation. A surprising proportion of women they have interviewed decline to be labelled straight, homosexual, bisexual, or asexual and since many also refused the term “unlabelled” it is not clear what that leaves! Perhaps they change their response according to the situation and have no fixed orientation. Others have commented that some women move about on the sexual continuum in a way that men would never do. Perhaps these categories are not the best way to survey women?
Modern survey data scatter suggest minimal genetic contribution

There is another important feature of the data above (Figures 8-11). It is all very scattered compared with the mean. This is true for the exclusively homosexual data, which, more than bisexuality could be expected to show strong genetic influence. The data was international and included the USA, the UK, France, Netherlands, Australia, Norway, Finland, New Zealand. If SSA is genetically dictated, it should be the same regardless of country, culture or social condition. How scattered would data be if they were from a trait we know is mostly genetically fixed? Figure 12 shows what the scatter is like for adult male height in many countries; (data from Wikipedia in mid 2010). Height is about 90% genetically influenced.

![Figure 12. Mean male height for more than 50 countries (alphabetical order). About the same relative scale as Figure 9](image)

We can see that the data from a genetic trait are very much more tightly bunched than the exclusive homosexual data (Figures 10,11) in spite of the wide variety of cultures. SSA doesn’t look very “genetic” at all. However perhaps the way the sensitive SSA questions were asked could vary from survey to survey and increase the scatter. We think
Homosexual numbers show nurture prevails

this is probably not enough to give the 10-fold range in the scatter for exclusive SSA. The data scatter itself therefore seems to argue against genetic fixity.

Do bisexuals really exist?

Recently academics have questioned whether bisexuals really exist. It’s true that usually SSA or OSA predominates and exact equality of attraction is rare. But it’s also true that when given the choice, many people will opt for bisexual as a category, or identity, and by the standard of being active with both sexes in the past year, they are clearly bisexual. Many say they get different fulfilment from each sex, and the experiences are quite different. Bisexual people do exist.

It is also true that many of those who have same-sex contact actually are married and identify as heterosexual. Some surveys call them “mostly heterosexual.” They are not part of the visible gay community, do not identify with it, and may actively dislike that lifestyle. In surveys which ask for self-identification they may say they are heterosexual. Of course this could have the effect of understating numbers of homosexuals—though this is not a problem if the criterion as above is actually sexual contact, or the alternative criterion of attraction is used.

The surveys of bisexual percentages come up with an interesting statistic. Of all homosexually active males, about 15% are married.8-11,46 A 1970 Kinsey Institute survey of females showed about 45% of lesbians had been heterosexually married, and about 45% were currently married.12 These are important statistics because they suggest that a significant amount of bisexuality is, in fact, homosexual behaviour by men and women in heterosexual relationships. We could probably say that most bisexuals are, in fact, homosexuals and lesbians who are or have been married or in de facto heterosexual relationships. But even the figure for bisexuality isn’t anything near Kinsey’s 10%.

SSA increases show genetic contribution is not fixed

A published paper33 drew on systematic US public surveys since 1988, showing the percentage of people having same-sex partners in the preceding year. This has significantly increased, as shown in Figures 13 and 14, for both men and women. However the number of exclusively homosexual men and women did not change significantly. The author
thought changes were homosexual experimentation by the previously exclusively heterosexual, in today’s more tolerant social climate. Other surveys in the United Kingdom gave conflicting results, but suggested

**Figure 13.** Percentage of males with same sex partners

**Figure 14.** Percentage of females with same sex partners
an increase from about 1% to 2.8% in five years between 1990 to 2000. There is no doubt a permissive society encourages greater experimentation. But this merely emphasises that most of today’s homosexuality cannot be genetically driven.

Dutch researchers recently compared their occurrence data for 1989 and 2008. Bisexuality increased for men from 6.2% to 7.9% and for women from 1% to 5.5%. The results are very high and suggest a lot of experimentation. Similarly in Australia from 2001-2012 female percentages increased. The irony is that Kinsey’s wrong data led to greater permissiveness in the West and became a self-fulfilling prophecy. However, this shows again that SSA changes with social setting.

Drop in SSA with age shows genetic contribution is not fixed
Homosexuality is not fixed, in fact it is far less stable than heterosexuality. Although the Kinsey surveys of 1948 and 1953 greatly exaggerated homosexual and bisexual numbers, they showed one interesting trend, also borne out by subsequent studies—a steady decline in homosexual fantasy and activity with increasing age compared with heterosexuals (see Figures 15 and 16). In other words, homosexual orientation and

Figure 15. Kinsey et al. Change in homosexuality with age in males.
Class 6: exclusively homosexual, Class 5: predominantly homosexual, Class 4: mostly homosexual, Class 3: equally homosexual and heterosexual
behaviour is not a static condition. This has significant implications for arguments that homosexuality is genetically determined. Whatever is genetically determined is by definition, unable to change within a generation.

Later results (Figures 17 and 18) from the large and excellent Chicago-based Laumann study, also show a strong decrease in homosexual behaviour, this time about four-fold (from age 35 to age 55), with a corresponding drop in those who identify themselves as homosexual or bisexual.

Could it be that the older “homosexual” people interviewed simply had not been so active? In that case why did they not retain their homosexual identity? Do the graphs merely show a huge increase in “young” homosexuality in Western society in the last twenty years? What sociological experts call a cohort effect? No, because Kinsey’s much earlier data show the same fall-off with age.

Kinsey was pre-AIDS and the decline cannot have been due to deaths but change in behaviour and fantasy. The Laumann study (Figures 17 and 18), when they used the SS Activity criterion, “If you haven’t had sex with someone of the same gender in the past year, you
are not homosexual,” could potentially have misled. In the gay community, due to the emphasis on youth and appearance, it becomes harder to gain unpaid casual partners beyond middle age. Many have celibacy forced on them. This would account for a significant part of the declines

Figure 17. Laumann et al.7 Changes with age in males

Figure 18. Laumann et al.7 Changes in homosexuality with age in women
in sexual activity his team recorded with age. However SS Attraction and Identity also show age decreases. Other surveys with different criteria also find the same decline, and a California public data set called CHIS showed the effect was not accounted for by SSA people shifting to “inactive” so it does seem to be real.\textsuperscript{10}

For some gays SSA is an extremely fundamental part of their identity. It is just possible that when desire, opportunity and fantasy fade, some gays no longer call themselves gay so are not detected by surveys, even the biased ones of Kinsey.

The conclusion was that heterosexuality absorbed most of these homosexuals.

We could sum up OSA/SSA differences like this: SSA tends to be much more intense and preoccupying, but overall, peaks and declines more steeply with age as well. OSA is a relatively sedate affair in comparison and much more readily tends to plateau and express itself to relatively old age.

Wherever the changed homosexual/bisexual behaviour goes—whether toward the heterosexual end of the Kinsey Scale (consistent with other research findings) or into inactivity—the change is considerable, and at odds with a genetically dictated condition stable throughout the life-span. We will look at spontaneous change in much more detail in Chapter Twelve.

**Urbanisation strongly influences SSA development**

The large Laumann study\textsuperscript{7} asked where people had been brought up during ages 14 to 16 and whether they had any male homosexual partners during the last year. The percentages depended on the degree of urbanization; 1.2% of the males surveyed who had been raised in rural areas reported having homosexual partners during the last year; 2.5% who had been raised in medium-sized towns reported having homosexual partners, and 4.4% who had been raised in large cities reported being active homosexuals/ bisexuals (Figure 19).

For women, the percentages were 0.7%, 1.3% and 1.6%, respectively. In other words, where you were brought up is quite an important factor in whether you end up having homosexual partners. For the sake of argument (Figure 20) let us imagine that the incidence of male homosexuality in rural areas (1.2%) is all due to genetic influence.
Homosexual numbers show nurture prevails

If that were the case, geneticists would also expect 1.2% of the male population brought up in “big cities” to have a genetically based homosexuality, meaning that the homosexuality of the balance (3.2%) \([4.4 \text{ minus } 1.2]\) would be exclusively due to social factors. This means that

**Figure 19.** Laumann et al.’ Homosexuality is dependent on adolescent domicile, ages 14-16

If that were the case, geneticists would also expect 1.2% of the male population brought up in “big cities” to have a genetically based homosexuality, meaning that the homosexuality of the balance (3.2%) \([4.4 \text{ minus } 1.2]\) would be exclusively due to social factors. This means that

**Figure 20.** Contrast between city and country domicile, males only
the environmental factor (3.2%) is far more important than the alleged genetic factor (1.2%). For women the environmental factor (0.9% [1.6% minus 0.7%]) is slightly more important than the supposed genetic influence (0.7%).

In several other chapters we argue that it is entirely plausible that 90% of homosexuality is accounted for by environmental factors. This very approximate comparison from the Chicago study supports that.

Similarly Frisch and Hviid in a study of 2 million Danes found that those who were born in cities were more likely to be in registered homosexual domestic partnerships than those born in the country.44

SSA in the clergy—the real 10% case?

SSA is fracturing churches worldwide. What started out with the appointment of openly gay bishops and clergy in the 1990s has turned, in the last several decades into a revelation of occurrences of homosexuality in the clergy much higher than in the population at large. Kinsey’s 10% is a current underestimate of the percentage within the clergy in several denominations, particularly the Catholic and Anglican churches.

The information the public has been given about sexual abuse by priests has been sanitised to avoid use of the word “homosexual.” The public has been told about child sexual abuse, and pedophilia, but by far the majority of the hundreds of cases made public have been with post-pubertal boys.51 In other words the sexual abuse crisis in the Roman Catholic Church is about homosexuality, not pedophilia.

The appointment of gay clergy, and the blessing of civil unions is unprecedented in the history of the church and has come as far as it has because of high levels of homosexuality in its leaders and electing bodies and high levels of public tolerance and ignorance.

The Episcopalian church in the United States provoked a major rupture with Third World members of the Anglican communion in 1993 by appointing an openly gay bishop, Gene Robinson, who had divorced his wife and was living with a male partner. In 2010 the denomination elected a lesbian bishop. Malcolm Boyd, a US Episcopal priest said he met more gays in seminary than he ever met in Hollywood.34 An anonymous US Catholic priest35 said, “At no time did I ever live in a community where gays did not make up at least half of the community.” In the year 200036 The Times (UK) reported that AIDS deaths among Anglican
clergy were 10 times higher than the percentage in the whole population. There are reports of very high percentages of gay people in many theological colleges in the UK— a typical figure is 30%. These figures are anecdotal but Heckler-Feltz reported in 2000 that AIDS deaths among US Roman Catholic priests were also about three times higher, though based on a rather incomplete survey. Of live clergy, 15% said they were homosexual and 5% bisexual. See also a Wikipedia article, and the assertion of 80% homosexuality in the Vatican curia.

Incomplete as these figures are, they seem very high compared with those for the general population. Why are people with SSA attracted to being clergy? Some may see it as a refuge—a “safe” place where they hope with God’s help to overcome the condition. Or a place where they can avoid questions about why they don’t have a girl friend or aren’t in a long-term heterosexual relationship. Or a place where they can find others like themselves. Still others may be seeking to move the church from within towards increasing acceptance of homosexuality in its priests. Others may be attracted to the idea of a “serving”, i.e., non-competitive male environment. Kinsey did not find unduly high numbers with SSA in the clergy, so this seems a trend of the last few decades.

Of course there are many anecdotes about SSA within the clergy. The Roman Catholic priest who had a fatal heart attack in a gay sauna was in good company—two fellow priests who happened to be there were able to give him the last rites. Some situations are farcical. After the election of Gene Robinson, a journalist at a press conference asked the spokesman “So if I am heterosexual, divorced, and living unmarried with a partner, I can now be an Anglican bishop?” The spokesman demurred, saying that they would want to look at that situation very closely. The press conference dissolved into laughter and broke up.

Before the Reformation, Luther reported that in Rome one cardinal was considered saintly because he confined his sexual attentions to women, rather than including boys as all the others did. In 2006 however, when the Catholic church was in the process of tightening standards to prevent continuing priestly homosexual activity with young male teenagers, it found Anglican liberal views a barrier to further ecumenical talks. From Roman Catholicism’s current official perspective, one sexually active SSA priest is too many but unofficially and at high echelons homosexual activity continues unabated.
Conclusion

Modern surveys show the homosexual percentage in Western adult populations is much lower than one in ten, except perhaps in particular groups such as Roman Catholic and Anglican clergy. About 1% of adult males are exclusively homosexual and about 0.6% of adult women are exclusively lesbian. The figure for bisexuality and exclusive homosexuality combined, rises to about 2.9% for males and 1.8% for females, an average of 2.4% of the total adult population. Much of the bisexual component could comprise homosexuals and lesbians who are or have been married, but, even then, the figure falls far short of Kinsey’s 10%. The figure in the West however is rising because increasing tolerance encourages greater sexual experimentation. But this may be superficial social and sexual activity, passing with time, rather than expression of a structured-in orientation.

Both Kinsey’s figures and modern surveys when interpreted show the genetic contribution to SSA is minor and the environmental contribution is much greater.

People move away from homosexual identification and behaviour with age, whereas heterosexuals do not (meaning homosexuality is not determined.) The data scatter is too high for homosexuality and bisexuality to sit easily in the genetic category, and the location of upbringing strongly influences SSA development, genetic factors being minor.

References

Homosexual numbers show nurture prevails


Are heterosexuals born that way?

Most heterosexuals asked how they became heterosexual would probably shrug and say something like, “I don’t know, it just happened. Maybe I was born that way?” But it’s no mystery how we become heterosexual; the stages of human development toward heterosexuality are well known and documented, and in this chapter we’ll look at the most important ones. Altogether they make a strong case for an environmental rather than a biological basis to sexuality. The research literature also gives good evidence that many people who have a homosexual orientation often had a struggle with a couple of stages that are part of heterosexual development. We will also propose that strongly individual responses, often to random events, are involved in sexual development.

The conclusions of this chapter overturn the theory that there is a prenatal surge of testosterone which permanently and overwhelmingly masculinises the brain. However, that theory will be discussed in its own terms in Chapter Eight.
STAGES OF HETEROSEXUAL DEVELOPMENT

Affection, nurture and bonding

Animals
A female fly lays eggs near food, but she is not around when the young grubs hatch. They have no family life, no mothering, no fathering. The presence of the female fly is not needed; the grubs do not need her affection, but still breed like, well…flies. On the other hand some of the higher animals particularly need early mothering. Affectionate early nurture seems to produce the capacity for affection in offspring—with effects on sexuality.

Researchers who have brought up monkeys completely isolated from other monkeys, giving them only a cloth mother figure, have observed subsequent breakdown in their mating behaviour. When they were frightened, young male monkeys would run to the cloth figure and cling to it as a kind of substitute mother. But when they were mature and were introduced to sexually receptive females, they were confused, clumsy and fumbling in their attempts to mate, and frequently failed to do so when they tried. The researchers concluded that mating is not completely instinctive but partly learned, and depends on the quality of early nurturing. Female monkeys brought up without maternal nurture don’t have such obvious trouble mating, but their behaviour as mothers is alarming. They are brutal and even lethal; “helpless, hopeless and heartless” the researchers observed,¹ a finding they extrapolated to abusive human parents. Early isolation and lack of nurturing fail to create affection in offspring. This affects the mating abilities of male monkeys and makes poor mothers of female monkeys. Much later, researchers discovered that lack of mothering caused marked biochemical changes in the brains of monkeys that lasted for years.²

There is a lot of research about animals, mainly laboratory rats and the effects of removing the mother (or father) for a time. The effects produced in offspring as adults (anxiety, mild depression, worse visio-spatial skills, poorer sexual and parenting skills, and greater drug addiction) could be a human model.

Does it really apply to humans? It will be a long time before we know for sure. But if it does, then the rat data tell us that the brain is
almost genderless at birth and that gender differentiation only develops with time. Interaction with the mother is really important and actually changes and primes our brains and makes biochemical changes in them, different for boys and girls.

For rats the biochemicals and processes involved are known. Young female rats deprived of just one day of maternal grooming, as adults had higher luteinising hormone and progesterone circulating and increased sexual receptivity, but were inferior mothers. For males with the mother absent for one day, one study showed there were degenerative changes as adults in the parts of the brain called the hippocampus and cerebellar cortex. As adults they were much slower to get involved with sex and ejaculated only 2/3 as often as controls.

Rat brains are anatomically the same for males and females at birth even on a microscopic scale. But there some submicroscopic biochemical differences; the maternal grooming causes sex-dependent differences in methylation of the histone proteins, changes in the estrogen and progesterone receptors in the brain, and changes to hormones and cell turnover in the brain organ called the hypothalamus (connected with sexual activity). Maternal deprivation also permanently switches the brain to enhanced “learn” mode (brain plasticity). It is as though the stress sends a signal to the brain that it will be unusually important to learn to cope in this stressed environment. The same authors said that sex differences in the brain are “not an inherent emergent property but are instead largely determined by extrinsic factors,” e.g. maternal grooming. The most critical biochemical change resulting in the pups from the grooming is in the enhanced level of estradiol, a rather versatile sex hormone which triggers further changes that are different in each sex. Careful anatomists say there is one structure in the rat brain that does indeed express biochemical maleness or femaleness and that it is weighted at birth to develop as female or male given the usual grooming. “These data suggest that early social interaction, similar to hormone [effects] may... organize typical sex differences in the brain.”

Breeders and biologists often experience difficulty inducing captive pandas to mate, which may stem from relatively high levels of social interactions with humans in captivity. Some keepers in China and Thailand have shown their pandas videos of “panda porn”— footage with mating pandas in an attempt to teach them to mate. A number
have been successful, even resulting in reproduction. But this is merely one example of the difficulties of captive breeding programmes: far from sexual reproduction being instinctual, innate and automatic, it is heavily dependent on social circumstances. The constant presence of human keepers from birth, frequently handling the animals, disrupts their sex life. We can expect a large learning component in human behaviour as well.

One piece of scientific research on animals adds an interesting perspective to parental and peer influences on later sexual behaviour. Kendrick and colleagues at the Babraham Institute in Cambridge, UK\(^8\) allowed ten ewes to raise goats from birth and ten nanny goats to raise lambs from birth. The fostered kids and lambs grew up in mixed flocks of sheep and goats but the kids fraternised mainly with lambs and adopted their play and grooming habits, and the lambs fraternised mainly with kids. Once mature they ignored their own species and tried to mate 90% of the time with the foster mother species. They kept this up every day during an observation period of three years, and even after years of mixing with their own species, the males did not revert (but females did). If the sexuality of these lower animals was so influenced by learning, human sexuality will be more so.

Humans

What about us? Do we learn to be affectionate from our earliest relationships? It seems we probably do. Environments severely deprived of nurture don’t just make us unable to be affectionate with either sex, they actually kill us.

The thirteenth century chronicler Salimbeni of Parma, Italy, told the story of his contemporary, Frederick II of Germany.\(^9\) Frederick had extensive domains in Sicily and Italy, was Holy Roman Emperor, and was considered perhaps the most enlightened man of his age. He was tolerant toward Jews and Muslims and a patron of the arts and sciences. He was also reportedly “bald, red and short-sighted.”

Frederick II had a theory that there was an original Adamic language, innate to all mankind, but that we did not grow up speaking it because we were exposed to the languages of our countries through our parents. He thought that if children were brought up in isolation they would automatically start speaking this original language. So he took
some children and committed them to the care of nurses, but only for feeding and bathing. There was to be no cuddling, caressing, or speaking. The outcome? The children did not survive long enough to develop any language at all. They all died.\(^\text{10}\) (Frederick’s reaction is not recorded, but he was so short-sighted he should have been redfaced, to put it baldly.)

In 1760, a Spanish bishop recorded: “in an orphanage children become sad, and many of them die because of this sadness”. In those days an orphan child in an orphanage received minimal care and little affection.\(^\text{9}\)

In their attempt to breed a master Aryan race, the Nazis took children born from genetically “ideal” parents and attempted to raise them under controlled conditions to realise their maximum potential. The directors of the program did not give the children normal mothering; they were left to their own devices in an institution for long periods. The experiment was a disaster. Again, some of the children died, and most of the rest developed severe psychological problems, which often left them unable to form normal relationships.

Various childhood researchers concur. Langmeier, well-known for research into the effects of extreme isolation in early childhood, has found children deprived in this way are slow to develop generally, and find it difficult to form normal human relationships of all kinds.\(^\text{9}\) Nielson, et al. looking at offending delinquent adolescents, found numbers of offences correlated with extent of early maternal separation. These children “lack basic human trust and capacity for empathy, and their interpersonal relationships are shallow.”\(^\text{11}\) In a classic paper, Helen Deutsch linked early loss of maternal nurture with lack of affection and inability to form relationships in adulthood.\(^\text{12}\) Beres and Obers (cited in Schwartz, et al.\(^\text{1}\)) remark on the effects of severe deficiency in early maternal nurture. They followed thirty-eight subjects aged sixteen to twenty-eight who had been institutionalised early in life, and remarked that none of them “demonstrated the capacity to make a successful marriage or to parent.” Beres and Obers thought this was primarily an intimacy problem. Another feature of some individuals with attachment problems is a total lack of fantasy. Some find any kind of imagination difficult.
In the 1950s, the World Health Organisation asked British psychoanalyst John Bowlby to research the mental health of homeless children. His response was a monumental book, *Attachment and Loss*, which led to more affectionate child care in institutions. The work also led to hospitals permitting parents to live in, to maintain bonds with hospitalised children. Bowlby found that extreme emotional deprivation in early childhood produced children with very cold personalities who were unable to form lasting relationships. They also craved affection. Later sociological surveys generally confirmed and expanded Bowlby’s work by showing that paternal influence uniquely and independently explained psychosexual development.

Work on 91 institutionalised girls showed that in adult life they had much more frequent mental difficulties and severe parenting difficulties. However, the support of a good spouse and of good living conditions in adult life were powerful protective effects.

A very large survey of 1800 institutionalised orphans as adults published in 1997 showed some fascinating trends. They had achieved better education and finally work income, than the population average. They were twice as happy as the rest of the population and had half the rate of mental illness. This showed that generally the orphanages had done a rather good job; 86% of the study orphans had not wanted to be adopted out of their orphanage! However these adult men and women had a higher divorce rate (29% and 63% respectively) than the general population at the same age. Yes, orphans do suffer—in unexpected ways.

Parental gender expectations and training

There seem to be very few gender differences in temperament of newborns. One study found differences in only 4 out of 34 test items, and comments “similarities between boys and girls are much more the norm than differences related to gender, but even though they are quite subtle, differences do exist in the way newborn infants react and behave in the neonatal period.”

In contrast, affection shown to baby boys (by anyone, but especially the mother) sometimes produces an erection. This undifferentiated response becomes more and more specific with age, eventually being restricted to those of the opposite sex who are potentially sexually
responsive. This process of differentiation is connected with the development of gender identity.

Mothers often deny treating boys and girls differently, but studies show they do. The parents know the gender of the child and from then on treat him or her as a member of that sex—often unconsciously. Boys’ limbs are exercised and stretched far more, and the vocal babblings of girls are imitated far more. Later in infancy, boys are allowed less physical contact and less verbal and eye contact than girls. Boys are more likely to be held facing away from the mother (and father) than toward. The parents are more likely to point something out to a boy than a girl. The mother tends to yield more often to the boy’s demand to feed, whereas the girl is more readily denied and given direction. She has to yield to her mother’s ideas of how much to take and when. When this sort of different behaviour is repeated hundreds of times, it is bound to have an effect. “By the age of thirteen months, there are clear differences between male and female children,” says LaTorre. There is apparently an attempt to “develop independence, adventure and mastery in the boy… The males show much more exploratory and autonomous behaviour.”

Most other people also reflect their gender expectations toward the child. In some experiments, researchers took young babies and pinned opposite-sex names on them: girls names on boys and vice versa. Without knowledge of the experiment, people who were strangers to the babies were brought in to see them. Predictably, they cooed over the “girl” babies saying “Isn’t she pretty?” and over the “boys” said things like, “Looks like he’ll be a good cricket player when he grows up.” A father, watching his young son cut a steak with unsteady knife and fork, remarked approvingly, “That’ll give you big muscles!” Presumably he would never have said it to his young daughter. If a small boy drops his trousers and pees in the back garden, mother probably laughs, but if her daughter takes off her underwear and throws it over the neighbour’s fence, she is probably corrected. Studies again show that the boy is given much more freedom and allowed to do many things the girl is not. His dirtiness and untidiness is tolerated far more than a girl’s.

The growth of gender-awareness

Imitation is one of the child’s main methods of learning. One of a baby’s first milestones is the first smile, at about the age of seven weeks. When
it is not indigestion, it may be an imitation of its mother’s smile. At about five to seven months, a child knows the difference between Daddy and Mummy, and begins to turn to them for comfort and protection rather than strangers. At about the same time, a sense of “self” begins—children begin to realise that mirrors portray themselves as separate beings. ¹⁸

Even at five months, researchers¹⁹ could find little genetic component to temperament as shown in physical activity, social gaze aversion, positive or negative expressivity and self comfort. It was mostly caused by other sources, such as erratic reactions to family environment.

With a subtle test—eye-tracking, i.e., recording how long a child watched gender appropriate toys²⁰,²¹—researchers were able to show that for children of 3–8 months girls preferentially watched dolls and boys watched trucks! Some researchers have found young female monkeys similarly prefer to play with dolls and male monkeys prefer trucks!²² Nobody really knows why. One could guess that there might be a very indirect reason such as fascination with moving objects compared with more static baby-like objects.

At age 12 months girls look at people about twice as much as boys do, showing a female preference for people.²³

But the child only begins to develop a sense of gender at about eighteen months, and then only superficially. Shortly before eighteen months, children can tell men and women and boys and girls apart, even in photos, but mainly on the basis of external appearance, such as length of hair or clothing. At about eighteen months the miracle of speech occurs, and the child starts to learn names of things, and then names of classes of things. It starts to learn the names of body parts, including its own genitalia. It becomes aware that it belongs to a certain class of people—boys or girls. At this time gender-typical play begins²⁴ with girls starting a couple of months earlier than boys.

By the age of three, 65–75% of children correctly identify themselves as a boy or girl, but most do not at age two and a half.

Kohlberg²⁵ observed a boy of two and a half years who went round the family circle saying “I’m boy,” “Daddy boy,” “Mommy boy,” “Joey [a brother] boy.” After correction he dropped his mother from the list, but still became confused about the gender of those outside the family. Kindergarten age children already know from pictures of toys what a boy would like to play with and what a girl would. They can also identify
the sex of dolls correctly. They will not be persuaded to change these opinions, even with the offer of a reward! But they are still not clear what male or female really is, and categories and their properties are still very fluid and fuzzy at ages three to six. Before the age of six, children tend to believe in a form of magic; they believe a car could change into a truck under the right circumstances, or a boy into a girl. The famous psychologist, Piaget, and his followers demonstrated this. He found most four-year-olds thought a girl could be a boy if she changed into boy clothes, cut her hair like a boy, and played boy games. Another example is given by Kohlberg:

“The following comments were made by Jimmy, just turning four, to his four and a half year old friend Johnny—

Johnny: I’m going to be an airplane builder when I grow up.
Jimmy: When I grow up, I’ll be a Mommy.
Johnny: No, you can’t be a Mommy. You have to be a Daddy.
Jimmy: No, I’m going to be a Mommy.
Johnny: No, you’re not a girl, you can’t be a Mommy.
Jimmy: Yes I can.”

By the age of four or five, children tend to make distinctions between adult males and females on the basis of strength or size, and boys in particular attach great significance to these qualities. They think that social power derives from physical power, which in turn comes from physical size. “Children agree earliest and most completely that fathers are bigger and stronger than mothers, next, that they are smarter than mothers, and (by six and beyond) that they have social power and are the boss of the family.” Sex roles are stereotyped on the basis of size, strength, and power at that stage; almost all of a group of 16 four to five year old American children believed only males were policemen, soldiers, firemen, or robbers—categories involving danger and aggression. By the age of five, 97% of children know their gender is fixed and they cannot choose to be either a mommy or a daddy. By the age of six or seven, most are certain a girl cannot become a boy regardless of what she wears. By that age they all believe boys fight more than girls. Why? “Because girls get hurt more easily than boys.” The categories and the belief about the categories have become fixed. But they are not aware of gender difference as genital difference until about five to seven, even when extensively enlightened by parents. They also have considerable
difficulty accepting that the differences are natural and normal. They think that the genitalia of the opposite sex are “funny” or “wrong,” or have been cut off, or that perhaps one will grow more like the other.

Even though adult females are seen as less powerful and competent than males, female stereotypes are still powerful enough to make femininity attractive to young girls. The mother or female teacher is more competent and feminine than the young girl. Femininity is associated with “niceness,” nurture and helpfulness, and superior attractiveness for children aged six to seven. Girls continue to prefer feminine objects and activities at all ages.

Parent-child relationships
Psychologists differ over details of the process, but all concede the importance of attachment to the parent of the same sex (or a surrogate), the start of a dependent relationship, and imitation and modelling off that parent for the formation of a sense of gender identity. The child identifies with what is masculine or feminine in the parent of the same sex and absorbs it in a kind of daily osmosis. In identifying with his father (“I am like Daddy”), the boy makes the shift away from his mother that is essential for development of a masculine personality. For this shift to occur, the father needs to be an attractive and “salient” figure to the child: present, involved, warm, interested. Nicolosi\textsuperscript{26} says a father needs to be dominant and nurturing to be “salient”. Paternal warmth—as perceived by the child or by the mother—has consistently been linked to a boy’s willingness to identify with his father and masculinity.\textsuperscript{25} A “bad” father who creates conflict is worse for the boy’s masculinity than no father at all. An emotionally warm and involved father also has an affirming effect on a girl’s developing gender identity as she models her mother and peers.

Psychologists agree that the girl identifies primarily with her mother throughout childhood. By age four, she is clearly identifying with her mother more than her father. Although her identification with her father increases over the years four to nine, it has the effect of reinforcing her feminine values and feminine identification rather than weakening them. The same effect of mother identification does not occur nearly as strongly for boys. The little girl tends to stay near her mother and is encouraged to imitate her and do “mother” things. She learns and
copies dress, appearance, and behaviour. The boy has a more difficult task than the girl, who retains her primary attachment to her mother. He has to separate himself from his mother and learn to imitate his father. This is quite a conceptual leap, and it is no surprise that boys are significantly slower to mature socially than girls. However it isn’t a strong rejection of the mother but rather continued change and growth. The girl also separates from her mother, but later and in a much more subtle way. Imitations of mother and father are well advanced by age three. Perceptions of parents are also influenced by birth order: it is common for first-borns to think later-borns are given more privileges than they received at the same age. The perception of parental warmth even among identical twins is strongly erratic.27 Chance events affecting one twin and not the other can mean each perceives the parent differently.

A recent New Zealand study shows that parental divorce doubles the risk in children of later SSA. The same study showed, however, that children of solo mothers (many not divorced) were not affected.28

Some researchers divide children into “dandelions” and “orchids”. This whimsical distinction means that dandelion children will flourish anywhere, but orchid children are frequently at odds with the family, school and peers, seemingly destined to a life of trouble. However, in the right circumstances orchids “bloom spectacularly” and outshine the dandelions. This little metaphor illustrates the range of different individual reactions there can be to essentially the same environment.

Sibling relationships

Twin study researchers found weak to moderate genetic effects on masculinity and femininity for pre-schoolers but the influence of older siblings and random events was much stronger.29

A large UK study of 14,000 children called the Avon study, showed clear effects on masculinity of a father present in the home, but only for boys, in fact girls were influenced more by elder brothers than their fathers!30,31 An older brother created more masculinity and less femininity in both boys and girls. If there was an older sister, boys were more feminine but not less masculine.30 So masculinity tends to predominate. Although these effects may be large for individuals, for the group as a whole having an older brother only increased masculinity by a few percent.
Peer group relationships

By ages three and four, boys are showing clear preferences for boy-type activities, toys, and boy friends rather than girl friends, preferences that remain stable or increase with age. For girls, the choice of girl-type activities and toys, and girls as preferred friends, is well established by the same ages, but does not increase. When Koch observed pre-school children, he found 80-90% of friends were of the same sex. It seems quite reasonable, comments Kohlberg, to attribute the same-sex preference of both boys and girls aged three to five to the child’s need to maintain its gender identity. Similarity leads to affiliation—boys and girls play with their own sex because they are like them.

So, by age three, boys and girls are already playing in different ways, and each group is quite distinct. Boys can become quite contemptuous of girls. When three-and-a-half-year old Joey was asked if he wanted any girls at his birthday party, he said, “No, I hate girls, girls are icky!”—a judgment partly informed by his natural growth, partly by his slightly older brother.25

Numerous studies show that boys play in a way which already echoes adult male society: games emphasizing competition and rules and winners and losers. Disputes about rules, or indeed about anything, are common, and a hierarchy is established in which each boy knows his (temporary) place. Boys tend to try to order each other about, reflecting their place in the hierarchy. Boys increasingly define their masculinity in terms of competitive achievement and acceptance in male groups. Girls, on the other hand, value relationships, and, if a game starts to cause disputes, it is usually abandoned. Girls want relationships, whereas boys want to be independent. Girls want to work together in an egalitarian sort of way and try to reach consensus by suggestion rather than orders. Paulk32 says that if a boy is hurt in a game, the game continues and another boy will jump in to take his place. Girls tend to stop and cluster round an injured girl even making access difficult for adults.

In one paper comparing boys’ and girls’ styles of handling a given task, boys used competition 50x as much as the girls, and girls used “taking one’s turn” 20x as much as the boys.33

By the age of eight, roughly 85% of both sexes believe their own sex is best. Boys who cross the line are mercilessly teased. “No-girls-allowed” activities are common to boys, in the attempt, by the boy some
psychologists believe, to consolidate his gender identity following the shift in identification to his father. Boys listen increasingly to what their associates want and believe, rather than to their parents, absorbing the sense of what is acceptably masculine from each other. As LaTorre says, the sexual orientation “soaks in from the outside.” A similar process happens for girls. The peer group has a similar role to that of the same-sex parent. Mixing mainly with their own sex strengthens a child’s sense of being male or female, and the differences between groups deepen.

Boys’ and girls’ groups differ. Boys include friends and acquaintances, but girls are much more choosy, restricting the inner circle to friends only, though these friends change much more frequently than the composition of the boys’ circles.

First attraction

As the differences increase, a natural curiosity develops about the other group, and this leads in a significant minority of cases to sexual investigation and experimentation; by the age of seven and eight more than one half of boys have been sexually exploratory with other boys and more than half with girls, usually without the knowledge of their parents. Only about half the girls were involved in pre-pubertal “sex play” of any kind. In more than two thirds of cases, the experimentation took place only once or twice, suggesting curiosity rather than attraction. There are stirrings of sexual fantasy in a faint pre-echo of puberty. At this age boys, in particular, become more interested in the sexual nature of female adults. Most of this still appears to be curiosity rather than hormonally driven because the mean age for first attraction is close to 10 for both boys and girls, about two years earlier than puberty, but possibly corresponding to the peak age of gender formation of boys’ and girls’ groups at school.

As puberty approaches, peer and parental pressure often leads girls to abandon tomboy appearance and pursuits, and intensify their gender characteristics. According to one study, girls become kinder and more sensitive but boys become braver and more adventurous.

Puberty

The next milestone in heterosexual development is puberty. In boys, the body is flooded with the male hormone, testosterone; in girls,
female hormones, estrogen and progesterone. In boys, the voice deepens, the genitals enlarge, and body hair thickens; in girls, breasts develop and menstruation begins. Both become aware of themselves as sexual creatures. Boys experience their first fully erotic arousal at about age thirteen (unless exposed prematurely to porn), and romantic fantasy begins in girls. In heterosexuality, this new sensation is expressed toward the opposite sex. But puberty does not create a sex drive that overrides existing sexual orientations, preferences, attractions, and emotional attachments. The hormonal surge only eroticises the psychological orientation that already exists. In people with a developing heterosexual orientation, sexual desire is channelled toward the opposite sex.

Even in intersexes, the pubertal surge usually expresses itself according to the gender of upbringing. Intersex people who have male gonads have been sometimes raised from birth as girls because of their ambiguous external genitalia, but at puberty they are flooded with male hormones and have erotic dreams (in a way which a young woman is much less likely to), the equivalent of the male “wet dreams,” but the imagery in their dreams is typical of young women’s dreams, not young men’s.

Sexual orientation is unsteady at the start. In early adolescence, deep emotional involvements with the opposite sex are quite rare, and there is usually a “superficial game-like quality to heterosexual interaction… It is almost like the play behaviour of the child.” Although they are also associating strongly with their same-sex peers, and confirming their own gender, adolescents often doubt their own masculinity or femininity at this stage. Same-sex sexual experimentation is quite high in adolescent boys; 12% reach orgasm with another person of the same sex, but usually only once or twice. Further information about the unsteadiness of adolescent sexual orientation is given in Chapter Twelve.

Falling in love

“Falling in love” rather than childish ‘crushes’ is another stage in the process of becoming fully heterosexual, one that doesn’t appear to be related to puberty, puberty being hormonal, and falling in love social.

Researchers know of some cases of girls falling in love before age twelve, but no cases of boys doing so. Even those children who are precociously sexually mature at very early ages—such as eight—do not fall in love,
although many of them have definite heterosexual fantasy, or dreams leading to orgasm, and may masturbate. In one case reported in 1932, a boy who became sexually mature before the age of four was reported to have made “obvious and distressing sexual advances to adult women with whom he was left alone.” But he did not fall in love.\textsuperscript{39} Falling in love doesn’t seem to be biologically driven; rather, it seems to require a certain age and stage of social development.

Branden\textsuperscript{41} argues that at base, romantic love is based on values as expressed in emotions. If so, values might well not be well developed when young, which could account for the lateness of love.

In the romantic West, much has been written about this mysterious sensation, but “falling in love” is not really very mysterious. A lot is now known about why people in the West are attracted to each other. In his book \textit{Families and How to Survive Them},\textsuperscript{42} Robin Skynner, a family therapist, boils attraction down to three things: social pressures (class, religion, and money), conscious personal reasons like good looks and shared interests, and unconscious attractions commonly called “chemistry”. To demonstrate how chemistry works, Skynner breaks his new classes up into groups while they are still strangers to each other and asks each person to choose “another person from the group who either makes them think of someone in their family or gives them the feeling that they would have filled a “gap in their family.” No one is allowed to speak during the exercise. When they have found each other they are encouraged to see if they can find out why they chose each other, and to talk about their family backgrounds. Then each couple chooses another couple, making foursomes, and then each foursome forms itself into a family of some kind, agreeing with each other about roles. In each case, Skynner reports, people choose others whose families have functioned in very similar ways to their own—for example, difficulty in showing affection, incestuous relationships, absentee fathers, or obligatory cheerfulness. In this group exercise, there are always people who are not chosen. The first time Skynner tried the exercise, this group of leftovers found they had all been fostered, adopted, or brought up in children’s homes. Although Skynner concedes his trainees are deliberately looking for someone making them think of their families, he says we are unconsciously attracted to certain kinds of people in a way that somehow mirrors the way we learned to relate in our families. In other
words, to a significant extent our responses when we “fall in love” have been unconsciously learned. They are not always the best ones.

We also know that falling in love is incredibly specific—a man doesn’t automatically fall in love with the sisters of his girlfriend. It is one person, and even one person of a twin pair sometimes!

Good parental warmth is related to children having fewer sexual partners later, i.e., lack of promiscuity but more specificity of attraction.43

In many non-western cultures, marriages are arranged, and people fall in love after they are married. That’s the way the culture does it, and if the arrangement is a good one, socially and economically, and there is mutual consideration, love usually follows.

A study44 of 445 pairs of twins, most of them identical, found no genetic contribution to the way “people make emotional attachments to each other.” Rather, the study found that spouses were more like their partners in “love attitudes” than twins were to each other.

If heterosexuality were genetic, one would expect an indiscriminate attraction to the opposite sex across the board. But (excluding incest, which falls in a different category) this is not the case. Young men do not want to marry their sisters, unless they have been separated from them during their upbringing.35 Studies in Israeli kibbutzim, in which unrelated children are raised together from a very early age while parents work, show they do not find each other erotically interesting in adolescence, though there are no restrictions on romantic involvement between kibbutzniks. In one study, all the young people without exception married outside the group they had grown up with.39

An influential study by Bem45 argues that what is “exotic becomes erotic”. In other words, a large part of what drives sexual attraction is the mystery of the other sex which has developed separately for years in childhood. Although this idea has been attacked by various researchers as inadequate, there is a general agreement that the exotic is one factor feeding into the erotic.

Marriage

A large Danish study found that a factor leading to heterosexual marriages for their children was youngish parents with a small age difference, in a stable relationship and an above-average number of siblings. Men with an unknown father were 20% less likely to marry.46
It seems marriage is often a vote that the family created by one’s parents is worth trying to copy.

Masculinity/Femininity not essentially sex-linked
The development of masculinity and femininity ends up very far removed from biology. An intensive statistical study of adults shows that masculine and feminine traits no longer show a sharp two-category, male/female division. Nor do other “psychological” gender related traits. On the other hand, physical traits such as waist/hip ratio show a much sharper division. This suggests sexual orientation mostly does not come from being male or female.40

Cultural conditioning
Sexual attraction and behaviour also depend on the conventions of a particular culture. In Wild Swans,47 an account of three generations of women in a Chinese family, Jung Chang writes of the custom of foot-binding. “My grandmother was a beauty…but her greatest assets were her bound feet, called in Chinese ‘three inch golden lilies.” Not only was the sight of women hobbling on tiny feet considered erotic, men would also get excited playing with bound feet, which were always hidden in embroidered silk shoes.

When Jung Chang’s great grandfather was seeking a suitor for his daughter, he planned the first meeting so that this daughter’s “tiny feet” would be seen to advantage in their “embroidered satin shoes.”

The custom has clear cultural origins. It began about a thousand years earlier when a Chinese emperor bound the feet of his concubines to stop them from running away. But they became erotic symbols—in spite of the fact that bones were broken and deformed in the binding process and that the dead skin stank when the bandages were removed.

The attraction of Victorian men to women’s ankles was another “cultural” erotic response. So is the reaction of males in some Moslem cultures to a naked female arm.

It is common for members of one culture to not be particularly erotically attracted to members of another, at least initially. It takes time to appreciate the social conventions of what is erotic in a particular culture and how well a person fulfils them.
Highly individual and random factors

People also develop their sexual orientation and preferences through chance incidents—random circumstances unique to the individual that are in some way associated with sexual arousal. Once the behaviour starts it tends to be repeated, and gradually become habitual. According to Gebhard of the Kinsey Institute, unusual behaviours and preferences can often be traced back to one-off incidents of this nature. He gives two examples. A young teenage boy experienced strong sexual arousal when he was wrestling with an older girl who was stronger than he was and on top of him. He later developed an attraction to large, muscular, dominant females, tried to include wrestling in love play, and became a bit masochistic. In another case, a boy broke his arm, which, because of the circumstances, had to be set without anaesthetic. It was extremely painful. While this was being done the doctor’s nurse clasped him close to comfort him. He became sexually aroused and later developed a fetish for brunette hair styles the same as the nurse’s. His sexual behaviour also became somewhat sadomasochistic. Gebhard places considerable emphasis on the role of chance circumstances in the development of sexuality. He comments about data “which show to an almost frightening degree the power of chance operating through variables in the immediate situation.”

We will see in Chapter Ten that twin studies also show very individualistic reactions are predominant in the factors leading to sexual orientation. These reactions are mostly to people and (often) to unusual circumstances that become charged with significance for the individual in some way, rather than to common everyday routines and experiences in a family.

Habit formation and addiction

According to Gebhard, any kind of heterosexual activity started soon after puberty almost invariably continues from then on. In other words, what we start doing we tend to keep on doing unless the negative consequences outweigh the perceived benefits. We form a habit. If the habit becomes a way of meeting emotional needs, it can become addictive.
Genetic contribution

A study by Hershberger\(^{88}\) and another by Whitehead\(^{89}\) concluded from three different approaches that the genetic contribution to heterosexuality was about 15%—surprisingly low.

Summary—the development of heterosexuality

No-one appears to be born heterosexual. Rather, heterosexual attraction is learned, developing over a period of time in response to certain environmental factors, in particular:

- Good maternal nurture from the earliest stages and through the first few years: nursing, feeding, loving, touching, talking, closeness, eye contact, and care of physical needs. This develops the ability to experience or show affection both to the opposite sex or to the same sex.
- Identification with and imitation of the parent of the same sex (or other close same-sex models).
- Acceptance by and identification with same-sex peer groups including elder brothers or sisters.
- Identification in a boy with what is culturally “masculine” and in a girl with what is culturally “feminine” (gender conformity).
- The day-in-day-out treatment of boys and girls, as boys or girls respectively.
- The biologically-programmed hormonal rush of puberty. This adds sexual drive to whatever prevailing psychological gender identity is already present. That is, it reinforces existing gender orientation but doesn’t change it.
- Falling in love. This appears to be unrelated to genes or puberty; it is something environmentally conditioned that requires a minimum chronological and social age.
- Culturally prescribed sexual behaviours, like arousal over women’s bound feet.
- Personal sexual preferences and behaviours that can be traced back to early sexual arousal in unique circumstances.

If anything was going to be programmed into the DNA, you would think heterosexuality would be. The urge to survive and reproduce ought
to be one of the most basic in the species. But heterosexuality including falling in love, seems to be a psycho-social learning process spread over many years. And for many heterosexuals the desire for a satisfying family life has come from their own experience of a good-enough family.

**HOMOSEXUALITY**

If heterosexuality is learned, what about homosexuality?

Some people have seen domestic animals mounting the same-sex of their own species and concluded homosexuality is intrinsic to the natural world and so intrinsic to humans. But such animal behaviour is more often linked to, e.g. battles for dominance in a herd or over territory, ownership of females or olfactory confusion, than to normal behaviour.

In this section we survey some of the many influences known, with the strong caveat that they do not apply to more than a small minority of people in the whole population. That is, each individual factor does not cause homosexuality in the vast majority of people, but for those who are homosexual, it has been found to be significant. Some homosexuals will identify very strongly with one factor, but not others. Where a number of these influences have occurred homosexuality is more likely to develop. But everyone has their own story.

**Relationships with parents and peer groups**

The psychological literature on homosexuality clearly reveals breakdowns in learning processes critical to the development of heterosexuality.

Adoption may be a factor. Although really definitive studies are lacking, the percentage of SSA people adopted seems to be about 6%, double the US national average. \(^{32,49,50,51,52,53}\) This suggests possible disruption of usual parent-child bonding processes leading to heterosexuality.

Family relationships matter. Frisch and Hviid \(^{46}\) in their survey of factors which led to Danish “homosexual marriages” found that lack of a father or a mother, made that outcome about 20% more likely. However having older siblings decreased the probability about 13% for each elder sibling. Younger siblings each decreased the probability about 9-13% for men and women. \(^{53}\) Similar patterns were found for U.S. adolescents. \(^{54}\)

Rather than bonding and identifying with same-sex parents, imitating and role-modelling, numerous studies of homosexuals show early breaches, negative relationships, and resistance to identification and
modelling. In one comprehensive study of homosexuality, 84% of homosexual men said their fathers were indifferent and uninvolved compared with 10% of heterosexual men, and that only 10% of homosexual men identified with their fathers in childhood, compared with two thirds of heterosexual men. Dickson and Byrd found a similar numerical difference and it is quite a big effect. This factor is confirmed in recent research. However it only accounted for 3% of total effects for the whole population, i.e., only 3% of a total population became homosexual as a result, but it was an issue for a large percentage of homosexual men. For those already vulnerable in some other way the effect would be much higher than 3%.

Rather than boys playing with boys and girls with girls, studies show pre-homosexual children have few friends of the same sex and are rejected by same-sex peer groups. They show boys who played with girls, didn’t like male sports, and wanted to be around women more than men. Poor relationships with peer groups are even more common in the backgrounds of male homosexuals than poor relationships with fathers.

Numerous empirical studies have shown that homosexual women have poorer relationships with their mothers than heterosexual women. Saghir and Robins found only 23% of homosexual women reported positive relationships with their mothers and identification with them, compared with 85% of heterosexual women.

Bell et al. comment that, in both boys and girls, a negative relationship with the same-sex parent reduces the desire to identify with that parent. Children with reduced identification are more likely to develop “gender non-conformity” (“sissiness” in boys and “tomboyism” in girls; the sense of feeling “different” from their peers). This is what we find in male and female homosexuality. Although this effect, “childhood gender non-conformity,” has been considered an excellent predictor of later homosexuality this conclusion was based on clinical samples, and one large recent random general population survey finds the effect is only weak—10-12% of gender non-conformists becoming homosexual adults.

However, it is worth noting that gender non-conforming clients in the clinical samples had parents with very high mental disorder levels and these rather feminine, insecure boys (a result of poor parental
bonding and modelling) can attract paedophile interest (early male sexual abuse is often a significant factor in the lives of homosexuals). So, although twin studies claim moderate to strong genetic origins for “childhood gender non-conformity,” social reasons can also be significant.

Sex researcher, Bell, also remarks that severe childhood gender non-conformity can be rebellion against gender norms.

Nicolosi remarks that “the masculine qualities conveyed in the healthy father-son relationship are confidence and independence, assertiveness and a sense of personal power.” A boy who has not bonded well with his father and has only a weak identification with him is not developing a sense of masculine identity and will not fit well into childhood male peer groups. Male homosexuals characteristically say they were rejected by childhood male peer groups because they were “weak, unmasculine, unacceptable.” That’s when the name-calling starts: “sissy,” “gay.” Bullying becomes common. Saghir and Robins found 67% of homosexuals were called sissy or effeminate by others (compared with 3% of heterosexual men), and that 79% of these men in childhood and early adolescence had no male friends, played mostly with girls, and rarely or never played sports. One study reported about the adolescent experiences of homosexual men “…sexually explicit feedback (from heterosexual peers) with critical implications occurred commonly among the homosexual men, which they interpreted as implying an insufficient masculinity.”

An interesting study in Taiwan found that lack of maternal care and high mother/father over-protection (not letting boys develop resilience) explained 62% of the homosexuality in male military recruits. This is an extraordinarily high influence, and probably reflects the strong role of the family in Taiwanese society.

It does show how hugely important parental factors can be in some cultures, and presumably in some individuals in the West.

A similar pattern is seen in lesbianism. Young girls resistant to mother identification and modelling do not fit well into female peer groups. In Saghir and Robins’ group, 70% of homosexual women were “tomboys” as children, compared with 16% of heterosexual women. They had no girl playmates (unlike pre-heterosexual girls), played mostly with boys, and were active in team sports. Most rejected playing with dolls
and showed no interest in domestic role-modelling. Sixty three percent wished they were boys or men, compared with only 7% of heterosexual women. The attitude persists into adulthood. One of the two findings that differentiated lesbian women from heterosexual women was the feeling in lesbian women that they were less feminine and more masculine.

They express disinterest in feminine accessories and fashion, prefer “sporty” and tailored clothes, and shun make-up and hairdos. They see their social and domestic roles as being incompatible with those of other women. They behave more competitively and are oriented toward career and accomplishments with little interest in raising children or in domestic pursuits.”

Sexual activity and sexual abuse

Several major studies have highlighted more childhood and adolescent homosexual activity in pre-homosexual children and adolescents. Van Wyk and Geist, looking at a sample of 7669 white male and female Americans, say both lesbians and homosexuals were more likely to have had intense pre-pubertal sexual contact with boys or men. They draw a link between male sexual abuse and later lesbianism, but also say that most lesbians learned to masturbate by being masturbated by a female. It appears that these women as growing girls had retreated from distressing male sexual contact at the same time as they had also experienced female sexual contact. By contrast, young pre-homosexual males appear not so much to be in flight from female sexual contact, as to find satisfaction in male sexual contact. Male homosexuals were more likely than heterosexual men to have been masturbated by other men or boys, they comment, and “once arousal to the particular type of stimulus occurs, it tends quite rapidly to form a pattern.”

Finkelhor found young men sexually abused by older males were about four times more likely to engage in homosexual activity as adults. Nichols reported male sexual abuse of lesbians was twice as high as in heterosexual women. Gundlach and Reiss reported a similar figure. Peters and Cantrell (cited elsewhere) found more than two thirds of lesbians reported being forced into sexual experiences with males after the age of twelve, compared with only 28% of heterosexuals.
The best review of the effects of childhood sexual abuse\textsuperscript{69} concludes that 12-37\% of SSA adults experienced this, but only 4-16\% of OSA adults.

Wilson and Widom\textsuperscript{70} followed sexually abused children into adulthood for 30 years and concluded that over their lifetimes men who had been sexually abused were 6.75 times as likely to be involved later with same-sex sexual partners. This is a very large effect. The effect on women was not significant. However for men the sexual activity was mostly not in the last year. The same six-fold effect of sexual abuse was observed elsewhere.\textsuperscript{71}

So sexual abuse appears to be a factor in the development of homosexuality. Ex-gay groups (Chapter Twelve) suggest that when a boy’s relationships with father and peer group are unhappy, childhood and adolescent sexual intimacy with another man leads to a later association of sex with male interest, affection, and acceptance. One former homosexual, Michael Saia,\textsuperscript{72} says homosexual men are not looking for sex when they have their first sexual encounter. He says they are looking for acceptance, understanding, companionship, strength, security, and a sense of completeness. Sex becomes the way to get it.

“I was starved of affection,” said Bob.

I didn’t like the sex at first, I just wanted someone to really love me. I told myself, OK, if this is what I have to do to get the touch, I’ll do it. Then it got to where I liked it. So…

(personal communication)

Lesbianism, on the other hand, is primarily emotional rather than sexual. Lesbianism is a relationship in which two women’s strongest emotions, affections and sexual feelings are directed toward each other.

One researcher in developmental psychology, Elizabeth Moberly, whose conclusions have been widely accepted by the international ex-gay movement sees sexual abuse as a secondary contributor to homosexuality.\textsuperscript{73} She posits the main cause as early “defensive detachment” from the parent of the same sex that interferes critically with the identification process that produces a sense of gender in children. This breach between a child and the same-sex parent (which, she says, could happen for any number of reasons, and is as often a result of childhood misperception of parents’ actions as of parental neglect or abuse), structures
itself into the relationship and leaves the child with a deep need for
the same-sex love, affection, and gender identity that it has rejected
or which has not been provided, Moberly says. Difficulties in attach-
ment and identification lead to a sense of not belonging in same-sex
peer groups and from then on homosexual development follows a fairly
predictable course: a drive for same-sex affection, affirmation, accept-
ance, and sense of gender identity; masturbation and/or fantasy around
a certain admired same-sex figure; a sexual encounter; the beginning
of habitual responses; self-identification as homosexual; “coming out;”
finding partners; the homosexual lifestyle, and for some, gay activism.
Most people with homo-emotional needs and homosexual responses,
however, do not “come out” to friends and family or live a visibly homo-
sexual or activist life-style.

In one of the largest studies of a homosexual population, Bell, et al.
said homosexuality could not be traced back to “a single psychological
or social root."59 However, they gave the highest values to a constellation
of factors: negative relationship with the parent of the same sex, “child-
hood gender non conformity,” and adolescent homosexual arousal and
activity. And these factors together were statistically significant. (This
study is further reviewed in Chapter Eleven.)

Puberty occurs at the same age as for heterosexuals.74 This tends
to discount many possible innate biological causes.

Homosexual identity as an adolescent is quite erratic. A survey of
many adolescents75 found that 3.4% reported gay/lesbian or bisexual
(GLB) identity (another 3.4% were unsure), 9.0% reported same-gender
attraction, and 4.0% same-gender sexual behaviour. However there
was no consistent pattern of overlap between the three measures, and
no single measure effectively defined this GLB population. The question
about attraction identified 71%; identity identified 52%; and behaviour
only 31%. This is in great contrast to adults for whom the three meas-
ures coincide almost entirely. It probably means that there is consider-
able adolescent experimentation without necessarily a great deal of
attraction. Expression of homosexual orientation is not stable until the
end of adolescence.

So, if heterosexuality results from a learning process that involves
relationships with parents, siblings and peer groups, puberty, sexual
encounters, highly individual experiences, and repeated behaviours, homosexuality follows a similar path.

The adult SSA male is almost always quite securely biologically male, as the SSA female is biologically female. The insecurity is inward: psychologically a male feels insufficiently masculine. Many feel they are perpetual outsiders regardless of success. They value masculinity hence they don't like effeminacy in other males—gay or straight. A large worldwide multicultural study found that according to standard masculinity tests SSA adults were less masculine on average than heterosexuals, and lesbians were more masculine on average than heterosexuals—although there was a huge overlap between the SSA subjects and heterosexuals. Another statistically significant difference was that SSA males were much more likely to treat others as objects (i.e., sex objects) than their heterosexual counterparts.

Some bisexuals seek heterosexual partners except when tired or depressed when they seek homosexual ones. This shows the malleability of bisexual orientation.

We repeat that most of the factors we outline in this chapter are weak influences on average in the total population, but for selected individuals (i.e., those who later become SSA) they may be critical. This means there is no single, unique path to SSA. Rosario et al. identified at least five pathways to SSA after study, and wrote “it may not follow a single pattern but may follow a variety of pathways”. One study on SSA concluded there was “support for the multidimensional model of identity development and exploration.” Nor is any individual factor overwhelming by itself. In fact a fair summary is that for any given factor the majority of a population will not develop SSA; several factors must act together. This gives rise to an aphorism: There's many a way to SSA.

Summary of homosexual development

For a variety of reasons the heterosexual model is not followed. Reasons include sexual abuse (by men), and a variety of ruptures with same-sex role models. Sometimes this is the father or mother, sometimes peers, probably including siblings. Quite a common consequence is being or feeling less masculine (males) or feminine (females) than others in the same-sex peer group. This can lead to rejection by peers (even other peers who are SSA) leading to feelings of being different, gender
non-conformity and a growing drive to make up the sensed deficit through a strong connection with an individual of the same sex, which becomes eroticised—essentially SSA. However individual reactions and stories predominate. Males feeling inadequately masculine, can envy heterosexual males and this can be confused with erotic feelings. SSA women frequently reject femininity but envy it less.

Bisexuality
In contrast, we observe that bisexual people find different needs met with each sex. For females, intimacy with females is very important and perhaps sexual contact with safe, non-threatening males (perhaps gay). For males the physical contact with males may be important and the relational aspects with females, perhaps including family.

Increasingly research is concentrating on “mostly heterosexual” people, who although overwhelmingly heterosexual, experience a slight attraction to the same sex as well. They tend to suffer mental health deficits, such as depression, at rates comparable to bisexuals and those exclusively homosexual.88

No sexual orientation
A few percent of the population, though physically normal, appear never to have learned a sexual orientation. Leiblum says

Some patients often show a chronic lifelong lack of sexual interest…Often we are unable to identify evidence of psychic inhibition of libido in such individuals but rather seem to be dealing with a permanent state of “asexuality.” Sexual stirrings or urges seem not to occur instead of being blocked or repressed.84

An interest group of the asexual (an interest group founded on a lack of interest seems rather paradoxical!)85 were not distressed by their asexuality, nor did they have a higher than normal degree of mental disorder. Masturbation was not different from population occurrence, so sexuality was present. They were rather socially withdrawn but functioned well.

In another study on asexuality (18 males and 75 females),85 although their sense of gender identity was well entrenched, some were
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aesthetically attracted rather than sexually attracted and 11/93 were attracted (but not sexually) to both sexes. Many felt they had “always been this way” and there was no obvious choice involved. Cuddling was about the limit of sexual activity.

One researcher described the unusual situation of a married couple with complete lack of sexual interest, who had known each other since childhood and discovered their common indifference. They appear to have married for companionship. When interviewed, they had lived together twenty years and slept in each other’s arms, but there was no genital contact at all. There was no physical abnormality. They were quite content. This may not be a complete lack of sexual orientation, but it had no erotic expression.

So it seems sexual orientation itself is not an inevitable consequence of genital development.

Conclusion

Heterosexuals tend to take their heterosexuality for granted as if it just happens. But it seems to develop slowly and steadily over years—about two decades—through fairly clearly known and accepted processes. Psychologists are in broad agreement about the general stages of heterosexual development and unanimous about one thing: heterosexual orientation is not genetically determined. They will say it is overwhelmingly learned, i.e., environmentally influenced. Most will also say genetics has a part to play, but only a very minor one.

Homosexuals in contrast frequently have difficulty with several of the developmental stages leading to heterosexuality, particularly attachment to and gender identification with the same-sex parent and good-enough connection with same-sex peers, leading to needs for same-sex affection and affirmation that become eroticised. Once the pattern of sexual gratification starts, a habit begins, becomes ingrained, and then often addictive. Rates of male sexual abuse are higher in homosexuals and lesbians than in heterosexuals, and this is a factor. If heterosexuality is learned, then homosexuality is, too. But there are many chance factors involved.

So, what role might genetics play in homosexuality? Probably about the same role it plays in the pregnancy of a fifteen year old girl. You could argue that if she is born with the combination of genes that make her
attractive in her culture (and therefore subject to more sexual pressure from interested males than she would be if she were ugly), then she is genetically predisposed to become pregnant at age fifteen. In homosexuality, it would seem that any biological trait that adds to a person's sense of “gender non-conformity” (one of the strongest predictors of later homosexuality) could be said to genetically predispose him or her to a homosexual orientation.

But did your genes make you heterosexual or homosexual? No, it seems you learned it over many years.

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How strong are instincts?

People will sometimes argue that if a behaviour isn’t genetic, it is so deeply part of them it might as well be. They usually mean that the behaviour is long-term, thoroughly embedded, and seems to be quite resistant to efforts to change it. If people wanted to argue that homosexuality was like a powerful human instinct, what might that mean? Would it mean it was unchangeable?

We all have some strong instincts; if a car tries to run us over, we dodge, and faster than we might imagine! Survival is probably our strongest instinct, the maternal instinct could be next, and the infant’s instinct to suckle, eat, and sleep is a close third.

We have an instinctive fear of loud noises and fast movement of a dangerous object toward us: our body goes into the fight/flight reaction and we either attack or run for our lives! We have a blinking reflex when something comes near our eyes; digestive reflexes; a pain reflex, e.g. instantaneous removal of a hand from a flame. Sleep seems to be a reflex when we are very tired. The contractions of childbirth are a reflex. We have a knee-jerk reaction when we’re hit just below the kneecap. Even male ejaculation is a reflex—it can be triggered by an electric shock. Then there is the sneezing reflex, yawning…you can add to the list.
Can we train our instincts?

Waft enough dust or pepper into someone’s nose, and almost everyone will sneeze. We sneeze instinctively. Or do we? It’s true that we sneeze in response to the reaction between the dust and our nose, but there is a pause during which we can stop or go on. A finger hard under the nose may stop a sneeze; looking at bright light may encourage it; sleeping stops it (we don’t sneeze while we sleep!). Doctors can stop labour contractions with drugs; we can stifle a yawn. Some reflexes can be trained, and trained surprisingly far. It is natural to blink when something is put in your eye, but if you wear contact lenses you can learn to control that and (usually) not blink until the lens is in the eye.

We can train many of our most basic instincts. We can train ourselves to ignore hunger pangs, and fast for religious or other reasons. When we have gone without food for a few days, we are not taken over by reflexes which force us to drop everything and concentrate all our attentions on getting food—indeed, after a few days the hunger pangs tend to disappear altogether and reappear only after the traditional forty days fast, when the body is at its last extremity. Considering we’ll die quite soon if we don’t eat, it’s amazing how weak the influence of hunger on behaviour is.

Similarly, although we will die or go mad if deprived of sleep for weeks, the sleep reflex is not overwhelming. Adolescents can keep themselves awake for an all-night event!

As a baby grows, it slowly learns to lose its fear of heights, at least enough to climb trees, hills, and in extreme cases mountains and overhanging rock faces. The rock climber may even enjoy the tension and fear! Blondin walking a tightrope over the Niagara Falls; Houdini the escape artist bound in chains, locked in a casket and dropped underwater; both had brought their survival instincts under control and revelled in the risk. Soldiers trained in mock battle conditions, senses assaulted by the loud shock of nearby explosions, learn to overcome their fear of death and obey orders. On the real battle field their training holds up—they fight rather than fly. So even the fear of death can be controlled.

Maternal instinct

The mothering instinct is among the most powerful instincts. In the animal kingdom, timid ewes will charge humans and dogs if their lambs
are threatened. Most mothers will protect their young. You would expect the mothering instinct in man to be more deeply programmed than in any species, because the newborn baby is unusually defenseless at birth. Its brain is so undeveloped that it will die if it is not mothered for the first few years. Mothers are equipped to conceive, carry, and suckle their young. They appear to be the natural nurturers.

Fathers don’t appear to have the same instinct to nurture. Surveys usually show that they spend only about one third of the time with their children that mothers do. Are human males biologically programmed to be poor nurturers, much more instinctively geared to fight aggressively outside the home to provide food for their families? Are we like the rats? The female rat constantly attentive to her young, licking, feeding, and guarding them, and looking after the nest structure; the male rat a menace, aggressively biting, and even eating young rats! Is this evidence for strong instinctive differences between male and female?

If that is the case, then it can certainly be reprogrammed. In an unusual experiment, biologist Jay Rosenblatt took several-day-old rats and put them in with virgin females. The females showed no mothering instincts and of course could not nurse the pups, so the pups tended to languish. Rosenblatt replaced the pups each day, and by the sixth day there was an enormous change in the behaviour of the virgin females. They began to look after the pups, licking them, retrieving them, and even more astonishingly, lying down as though trying to nurse them. Even though they were not primed by the hormonal changes of pregnancy, the presence of the pups alone was sufficient to trigger the maternal behaviour.

Rosenblatt tried exactly the same thing with adult male rats. After six days, the males started behaving just like the virgin females: licking the pups, retrieving them when they strayed, and even lying down as though trying to nurse them! In other words, maternal “instincts” were evoked by the presence of the pups in male rats, sometimes known to eat their infant offspring.

In rhesus monkeys the typical indifference of male monkeys towards infants can be broken down to the extent that they will show “maternalistic feelings as tender and solicitous as any shown by a rhesus mother” to any infant who needs care.
There is a celebrated instance in which a wild bitch died five weeks after giving birth, and the remaining five adult male dogs raised the nine pups themselves. In about 40% of primates, males care for the young. Sometimes they snatch the infant from the mother apparently for the sheer pleasure of carrying it about. Among the marmoset and tamarin monkeys, it is hard to say which is the primary caregiver.

Obviously, male behaviour is not firmly and instinctively imprinted in lower animals. It can be radically changed. The old rule applies: if lower animals, whose behaviour is much more biologically programmed than ours, can retrain natural instincts, then human beings can to a much greater degree. The modern woman who insists that men are quite capable of mothering and nurturing children appears to have science on her side; fathers are certainly able to increase the quality time they already spend with their children. Certainly “house-husbands” have brought up very young children. With glass bottles and rubber teats, a father can even nurse a child! There have even been a few rare cases of older men who (probably through some hormonal disturbance) were able to breast-feed young children. Similar hormonally disturbed cases, some induced by hormone treatment to fight tumours, are reported reasonably frequently.

Nor is maternal behaviour an over-riding instinct in human females. Some human mothers abandon their babies at birth. Hundreds of thousands of babies are aborted each year. Some women are poor mothers; some men make good ones. It seems the mothering instinct can be developed or neglected in a woman, and evoked in a man. If this is so what might be possible for same-sex attraction?

Irises

We never usually think about adapting to bright light—our irises adapt automatically and we don’t think about it. How would we go about training that reflex even if we wanted to? Norman Doidge describes that this change has happened in one ethnic group even though they have not set out to do it deliberately. The Sea Gypsies are a tribe who make their living mostly from the sea by diving. They live in the Burmese archipelago. Remarkably they can see at depth in the sea without goggles, by adjusting the irises in their eyes—probably producing the same effect as a pin-hole camera with its greater depth of field. Swedish researchers who
found this were initially most surprised because the reflex was thought to be unchangeable. But they were ultimately able to teach Swedish children to do much the same. Training can change brain circuits—what do you have that you think is a reflex but needs changing?

Sexuality
The urge to reproduce—to ensure the survival of the species—is a powerful instinct. But, like the survival instinct and the maternal instinct, it is not an overwhelming reflex. In fact, it can be controlled with training, as many in religious orders know. A significant minority (about 10%) of the general population has no wish to reproduce at all. So the urge to perpetuate the species is obviously not an overriding drive.

Actually, our sexual instincts often have to be rather vigorously prodded before they’ll move into the driver’s seat. The ejaculation reflex only takes over when a certain threshold of stimulation is passed, and usually quite a bit of stimulation is needed. We might not want to stop, but we can. Our instincts do not control us. An interesting proverb says, “Blessed is the man who controls his spirit [the drives which move him in various ways]. He is better than the man who captures a castle” (Pro 16:32). What makes our sexuality appear so powerful is all the training it gets. We are encouraged to express the sexual side of our natures. So, even though our urge or need for sexual expression might end up feeling irresistible, it’s really no more than an over-developed instinct, demand-fed hundreds of times for decades.

Homosexuality
Homosexuals cannot reproduce, so homosexual activity cannot be considered an instinct to perpetuate the species. If it could be called an instinct, it is no less malleable than any other of the powerful instincts that man experiences, which, we have seen, are subject to a huge degree to man’s will and other environmental influences.

Addiction
Addiction is not an instinct, but can become something very close to an instinct. The surfaces of body cells are chemically configured in such a way that they resemble a lock waiting for the right key to turn in it. The chemistry of certain drugs is like the key that turns perfectly in the
receptors of cells in certain organs of the body, and, after a while, the reaction becomes a part of cell life, creating a chemical dependency which the body feels as a need. If pleasurable sensations accompany the process and this “hooks” into some way into emotional relief, then an addictive cycle begins, minimal at the start but increasing in strength until it seems almost impossible to control. Is addictive behaviour an uncontrollable compulsion? Has the cell physiology made us do it? No, we helped it hundreds of times. But it’s possible to reverse the process and rediscover the old normalcy (or find a new one).

Conclusion

We can learn to bring our instincts under control, or we can allow our instincts to control us. Instincts develop because they are fed. No behaviour takes us over without years of encouragement. If we have spent all our lives cultivating a certain behaviour by thousands of repeated actions and responses, then it will eventually seem like a powerful urge—so powerful that it seems irresistible, or even genetically programmed. But nothing is unchangeable. If we can lose our fear of death with training, and even enjoy the risks, if fathers can become “mothers,” then sexual reflexes can also be trained. It may take a few years to reverse the training we have given them, but it can be done.

We are created to be voluntary animals, not involuntary ones. On these grounds alone, it makes no sense at all to maintain we are doing something we just can’t help doing. Somehow, we have trained ourselves into the habit. Though not without difficulty, we can just as effectively train ourselves out of it, if we really want to. But we will need the help of others and of a Higher Power.

Homosexuality, if some want to call it an instinct, is no different from any other instinct.

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What produces the sexual identity of intersexes?

A study of people with ambiguous genitalia gives unusual support to the prime role of the environment and upbringing in shaping human sexuality. The majority of intersexes (people of ambiguous gender appearance) who have come to the attention of researchers have opted for the gender of upbringing rather than their chromosomal gender—even in the face of emerging contrary biological characteristics. Only a small minority would greatly prefer to change.

Sometimes babies are born with such ambiguous genitalia that medical staff do not know whether the child is a boy or a girl. Until about the 1980s, parents instructed to raise these children in one gender or other often found them developing physically (usually with the onset of puberty) contrary to the gender of upbringing. But, when these children were given the option of corrective surgery and hormonal intervention at puberty, 90% of those whose cases have been researched opted for the gender in which they had been raised, rather than their biological gender, even in the face of quite contrary physical characteristics. In many cases—though not without difficulty—these children grew up to develop gender behaviours consistent with their gender of choice/
What produces the sexual identity of intersexes?  

upbringing, rather than their biological gender. They felt attraction, experienced erotic arousal, fell in love in ways characteristic of their chosen gender, married, and raised children.

Today diagnosis is much more sophisticated and medical options much wider. The situation varies a little from country to country, showing social conditioning is important. Many more elect to change from female to male than the reverse because they think being male is better, but overall, lumping all intersex conditions together, about 90% still choose to remain in the gender of upbringing.

A lesson in biology

Almost everyone, including homosexuals and lesbians is born chromosomally female (XX) or chromosomally male (XY). When a male sperm carrying 23 chromosomes unites with the female ovum, also carrying 23 chromosomes, the fertilised egg quickly becomes a 46 chromosome cell of 23 pairs, one of each pair from the father, one from the mother. All the chromosomes carry the genetic material that gives us our biological characteristics, but the 23rd pair is the sex chromosomes, usually comprised of one X chromosome inherited from the mother and an X or Y chromosome inherited from the father. An XX combination in the fertilised egg produces a female, and an XY combination produces a male. Sometimes these standard combinations do not happen, and rare combinations result from uneven cell division or for reasons that are still not very clear to researchers. One of the X chromosomes can be “lost,” leaving only a single X. These fertilised X cells still grow normally, but produce individuals who are very short (4 ½-5ft:137 cm+) and physically female, but have no ovaries and are infertile, a condition called Turner’s syndrome. Some fertilised cells can end up XXX, resulting in women with a normal female body, but diminished fertility, and sometimes mental retardation. Males can be XYY, with male body type, reduced fertility, and increased height; XXY or XXXY (Klinefelter’s syndrome) both cause male body type, but with unusually small penises, shrunken testes, and varying but low production of the male hormone, testosterone, so that at puberty they become only moderately masculine and have scant body hair. The percentage of homosexuals among people with Klinefelter’s syndrome is about typical for the general population (contrary to a commonly circulating myth), but about half of those with
the syndrome have no interest in any type of sex (they are quite prone
to sexual anxiety), partly due to their physical attributes.

Such varied sexuality means a person’s chromosomal pattern is not
forcing any particular sexuality upon them.¹

There are all sorts of rare combinations of X and Y, and some people
have a mosaic; e.g., XXY in one cell and XY in another. But, in general,
if people have an XY or XX combination of some kind they will develop
physically as male or female respectively.²

**Overwhelming effects of rearing?**

John Money, Anke Ehrhardt, and John and Joan Hampson, at the Johns
Hopkins Medical School in Baltimore, Maryland, spent a lifetime stud-
ying unusual sexual conditions and intersexes. What they found in the
sixties about the role of upbringing in the formation of gender identity
and sexual orientation led them to the conclusion that the influence of
upbringing and rearing was so overwhelming that it was as if a new-born
child was a blank slate, written upon only by the influence of upbring-
ing and socialisation.³ Ultimately this has proved to be too extreme a
conclusion.

In a summary of all cases of intersexes that had come to their atten-
tion (particularly the work of the Hampsons), Money and Ehrhardt said
about 90% chose to remain in their gender of upbringing in spite of
contrary biology; that is, despite some or any of the following: contrary
chromosomes, gonads, hormonal sex, internal sexual organs or external
genital appearance.³ They remark that of that 10% who changed their
gender, almost all of them made a female to male change. Although the
90% established a gender identity consistent with their sex of choice,
they did not do it without “difficulty, embarrassment, and shame”. This
represented the situation until the end of the seventies.

**Boy raised as a girl**

We now consider a well-known case which flatly contradicted the assur-
ance of the Hampsons and Money, but turns out to be rather misleading
because it happens only in a minority of cases.

One of Money’s cases² was a boy, one of normal identical twin
boys, biologically male in every respect, who suffered a surgical mishap
during circumcision by electrocautery at the age of seven months. His
What produces the sexual identity of intersexes?

penis was cut off flush with the abdominal wall. After months of agonising, the parents decided, when the boy was seventeen months old, to raise him as a girl, and doctors performed the first stages of feminising surgery. The child was called Joan, wore girl’s clothing and hairstyle, and the parents were regularly counselled how to raise her under the circumstances. The identical twin brother was raised as a boy. John Money touted this as a perfect case showing the malleability of gender because the reports seemed to show the “girl” was adapting well to the change. Her mother made a special effort to keep her in dresses because she was initially resistant to them and preferred jeans. By the age of four she had a clear preference for dresses over slacks, wore bracelets and hair-ribbons, and took pride in her long hair. But Joan was tomboyish, had a lot of energy, and was often the dominant one in a girls’ group. Her mother tried to teach her to be more ladylike. Further treatment was planned after puberty.

When the girl was about 13, she was interviewed by the British Broadcasting Corporation (BBC) and three psychiatrists, who concluded her gender identity was insecure. She refused to talk about sex. When asked to draw a human figure she drew a man because “women are too difficult.” She complained that men had it good in life and women didn’t. She had found it difficult to be accepted in her group of girls because she was not very attractive, and because her rather clumsy gait had gained her the nickname “cave-woman.” She thought she would rather like to be a mechanic. The BBC panel thought that the transformation had been rather shaky, perhaps even inadvisable. It seemed the attempt to environmentally over-ride the basic biology was a failure and “Joan” became the subject of a scholarly fight between Money and other researchers who believed Joan should never have been brought up as a girl. Some of them took the debacle as evidence that gender identity was so fixed at birth, that efforts to change it were futile. One sex researcher in Hawaii, Milton Diamond, argues for a “prenatal (biological) organization,” a “built-in bias with which a person interacts with his environment,” but an extraordinary flexibility to adjust to an erroneously imposed gender.

Money was accused of suppressing some of the evidence he had accumulated that adaptation to the new sex was much less than perfect. It all came to a head in early 1997 when it was revealed that at the time
of the BBC interview the cat was already out of the bag; Joan had found out three years before that she was really a boy. A year before the program she had rejected hormone treatment for feminisation. (No wonder she looked somewhat masculine.) A year after the program she began a two year program of penis reconstruction and began to call herself John. Eventually he married a woman several years his senior and adopted her children. Sadly, several years later he committed suicide, just as his co-twin had, some years before, so this complicated story may be further complicated by some mental illness.

A confusing picture, but one that shows, nevertheless, that gender is not written into our genes or gonads. It is malleable and responds strongly to environmental signals. Before the “cat was out of the bag,” the boy was behaving to a large degree like the girl he was being raised to be. Afterwards he decided to co-operate with his then known genetic biology rather than suppress it medically, and he also began the corresponding psychological gender shift.

This tragic story is well known, and even been the subject of a book, but it is less well known that Bradley et al.\textsuperscript{5} reported a (non-twin) case in which the same accident happened much earlier in life. The boy, brought up as female, clearly identified as female even after many years, reaching young adulthood, but said her sexual orientation was bisexual. So this story is almost the opposite of the one above—sometimes reassignment can work.

There is a later collection of data like this—by Meyer-Bahlburg et al.\textsuperscript{7} in which they managed to find no less than seven boys whose early accidents had forced the amputation of their penises. They were raised as female, and 69% stayed that way. One was not happy in her gender assignment (“gender dysphoria”). Telling the person the medical details of the accident did make a difference—half of those told the medical facts before puberty decided to change to male.

Rather similarly,\textsuperscript{7} of 16 boys born through a prenatal biological accident without penises, brought up as female, 12 or 75% chose to remain that way though two were rather unhappy with the assignment. Of another 17 with the same condition brought up as males, all remained that way. This shows a theme already apparent —there is a preference in most societies to remain or become male. The authors\textsuperscript{7} also said
that there was no good evidence that the prenatal surge of testosterone masculinises the brain. The authors concluded:

The data do not support a theory of full biological determination of gender identity development by prenatal hormones and/or genetic factors, and one must conclude that gender assignment and the concomitant social factors have a major influence on gender outcome. On the other hand a number of female-raised individuals did change gender to male and others developed a possible gender-dysphoria, which indicates that gender assignment does not dictate outcome either.

The critical word is “dictate.” But what is surprising is how successful upbringing in a contradictory gender actually was.

These data show that the case of the twin boy (Joan/John) was not typical, though the media exposure suggested it was. Most will stay in the gender they were brought up in, male or female. But cases like Joan/John’s are very rare. What happens on average with the more common conditions? Do intersex people stay in the sex of upbringing? We will try and summarise. First we describe one of the most common.

Adrenogenital syndrome in females (Congenital adrenal hyperplasia)

This condition, which affects female fetuses, is the result of a genetic defect; the adrenal glands do not produce their proper hormone, cortisone. Instead, they release a precursor product, which acts as a male hormone, an androgen. This enters the bloodstream of the female fetus too late to masculinise the internal reproductive system, which is already female, but in time to masculinise the external genitalia. The result is a chromosomal female with a uterus and two ovaries, but anything from a grossly enlarged clitoris resembling a penis with partially fused labia (resembling testicles) to a fully formed penis and empty scrotum. Because people with this condition continue to produce androgen for the rest of their lives, they must also take doses of the antidote, cortisone, to counteract it—in childhood, to stop an excessively masculine puberty which comes 8 to 10 years too early, but also in adulthood. Although the ovaries continue to secrete normal levels of female hormones, these
are overwhelmed by the high amounts of androgen being produced by the adrenal gland.

The percentage of these women who are brought up female but want to change to male varies with country and research group:

Byne\(^8\) 2-10% changed, Dessens\(^9\) 5% changed, Long\(^10\) 0% (they became indistinguishable from controls by adulthood), Meyer-Balburg\(^11\) 5.2% changed, Reiner\(^12\) 22% changed from female, Slijper\(^13\) 13% changed from female. So, overall, about 10% of these females with adrenogenital syndrome chose to change, but the percentage depends on the research group.

Turner’s Syndrome (single X chromosome)

We met this condition above. It has an interesting bearing on the subject. Because of their lack of ovaries, or non-functional ovaries, all Turner’s Syndrome people take estrogen throughout their lives. Some marry men, and recently some research surveyed how they were functioning sexually. The women were in two groups—those in relationships and those not. Approximately 30% of the study group were involved in a partner relationship, and this group scored within the average range for heterosexual women on fantasy, arousal, experience, orgasm. The authors concluded they had relatively normal overall sexual function, but the majority of unpartnered women reported very low-level sexual functioning.\(^14\)

Since both were on estrogen this hormone seems to be a negligible contributor. The conclusion is that sexual functioning arises overwhelmingly out of the partner relationship.

Males born without gonads

One more intersex study is enlightening. Szarras-Czapnik et al.\(^15\) reported on 10 males born without testes. What was their sexual orientation? The males cannot have had the prenatal testosterone surge which is supposed to make the brain masculine so this is an interesting test. The males were all solidly male and with a heterosexual orientation—7/10 had had sex with a woman. This argues again that at least for males, upbringing is far more important than testosterone.
What produces the sexual identity of intersexes?

Biologically-induced gender change (5α−reductase deficiency)

One special genetic condition seemed initially a possible major exception to the general rule of remaining with the gender of upbringing, and this was the deficiency of an enzyme called 5α−reductase. This deficiency prevents formation of one of the male hormones, dihydrotestosterone, so that the usual prenatal surge of testosterone that differentiates a boy from a girl before birth does not occur and external genitalia are ambiguous. If the condition is not diagnosed and treated, everyone gets a shock at puberty when the testes become detectable and the body becomes masculine. The researchers studied 38 of these cases in the Dominican Republic, particularly 18 who were “unambiguously raised as girls”.

Their findings? At puberty or after, 17 of these children changed to a male gender identity and developed an erotic interest in women. Many became heads of families. The researchers argued that androgens made a “strong and definite contribution to male gender identity.”

But the conclusion is not as straightforward as it seems. Critics of the research argue that men had much greater status and prestige in Dominican society, and that together with sudden masculinisation, a choice to be male could be strongly culturally influenced. Certainly the Dominican study seems to stand alone in the strength of its argument for a hormonal basis to gender identity. Another researcher into 5α−reductase deficiency drew an opposite conclusion. Gilbert Herdt, the most prominent researcher among the Sambia of the eastern high lands of Papua New Guinea, found five cases of 5α−reductase deficiency in his study group. In this case the individuals were raised as girls but on their sudden masculine development at marriageable age (puberty), were treated as a third sex. Although the Sambia are a strongly misogynist culture, there was no attempt—contrasting with the Dominican Republic case—to adopt a male gender, because the culture forbade it; the Sambia believed a boy could only become a man through ingestion of male semen in prescribed regular fellatio in childhood. Based on this cultural prohibition on becoming male Herdt argues that gender identity is therefore culture dependent rather than hormone dependent. Herdt also maintained that only 13 in the Dominican Republic study, not 17,

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§ It is doubtful that the brain is masculinised by the pre-natal testosterone surge but the surge is sufficient to masculinise both internal and external sex organs.
lived unequivocally as men. In an almost identical condition found in the Gaza strip, only 28% changed gender at puberty.\textsuperscript{18}

In the West, of those who have 5α−reductase deficiency, only a small percentage elect to change gender at puberty, and they find it difficult.\textsuperscript{19} Ninety percent are content to remain in the gender of upbringing (female), possibly because the perceived rewards of being a Western woman are greater than in other cultures.

**Androgen Insensitivity Syndrome**

In this condition the developing male fetus is insensitive to testosterone and is born with feminine genitalia. Outside the West this condition may not be detected at birth and the child is raised as a girl, and only referred to medical people when puberty does not arrive. Although the person has a vagina, there is little desire for sex, and it is often painful. The gender identity is firmly female, but now there are about half a dozen cases described where families have chosen to raise the child as male instead, and it seems moderately successful, though medical opinion is very doubtful about its wisdom. This mainly serves as a further illustration of the surprising adaptability even of gender under various cultural conditions.

**Other changes by intersexes later in rearing**

In India,\textsuperscript{20} of 74 intersex patients, all but one stayed with the sex of rearing. In Egypt\textsuperscript{21} 10% changed. Reiner\textsuperscript{12} in a paper from the West found that of 60 raised as female, 43% declared themselves female but 53% changed to male. This was a rather exceptional group in which there was considerable decision to change.

In Bahrain, McCarthy\textsuperscript{22} found all female patients reassigned the male gender accepted that, but no males accepted a change to female! In Russia, according to Lev-Ran,\textsuperscript{23} all adult patients resisted reassignment and wanted to remain the way they had been brought up!

It was noticeable that a Western group with cloacal exstrophy\textsuperscript{7} (in which sexual organs are poorly developed and internal organs such as bladder may protrude) had a large percentage—about 33%—of people who wanted to change from female to male and more who were unhappy.
What produces the sexual identity of intersexes?

Since the congenital adrenal hyperplasia group predominates (among intersexes), the original estimate of those who wished to change up to about 1980 still holds and is around 10%. The influence of upbringing is strong, but less so in the West since the days of the Hampsons.

Should intersex children be allowed to choose?

In the last few decades a strong minority opinion has formed that intersex children should be allowed the maximum choice and puberty should be delayed by medical intervention. A human rights ordinance in San Francisco\textsuperscript{24} sought to avoid early intervention. However, traditional medical opinion argues strongly that the intersex condition is devastatingly embarrassing for school children and that surgical intervention, even with its trauma, is far preferable. Less than 10% of intersex children later disapprove of the early intervention, and in countries such as Vietnam where corrective surgery is not available, intersex adults lament that it wasn’t.\textsuperscript{25} However, one report that studied 38 surgical early interventions found that four were much later at serious risk of Gender Identity Disorder.\textsuperscript{26} It seems the medical specialists can’t win: if there is no early surgical intervention, about 10% want to change later in life; if there is early surgical intervention, a different 10% will want to change.

Summary

So if we ask the question, “What produces the sexual identity of intersex children?” we have to answer that upbringing greatly predominates—even in modern society, and more so in a less liberal society.

That is, about 90% of intersexes on record have elected to continue in the gender in which they were raised, even in the face of strongly contradictory biological and physical characteristics. If the influence of upbringing is so strong that it can over-ride obvious contrary biological predispositions, then it is more powerful than biology in the development of gender identity, at least in most countries. It becomes nonsensical to argue that gender identity in chromosomally normal individuals (like homosexuals) is genetically or biologically enforced. In modern Western society, sexual identity appears to be about 10% genetic and 90% environmental. So it is quite plausible that homosexuality is also 10% genetic and 90% environmental.
References

What produces the sexual identity of intersexes?

What do different cultures tell us about homosexuality?

The huge variety of sexual expressions in different cultures sharing essentially the same genes shows genetic influence is minimal.

In 1994, an Italian-American geneticist, Cavalli-Sforza, published a huge genetic atlas\(^1\) the outcome of a monumental study of the genetic characteristics of different ethnic groups. He found that the human race was remarkably homogeneous, genetically. The more genes his team studied, the more they found all ethnic groups shared them. Cavalli-Sforza eventually studied fifty genes, and found that all ethnic groups had most of them. His conclusion was that, in spite of superficial differences, e.g., skin colour, the different races are essentially the same genetically. Later work shows in fact, that something between 99.7% and 99.9% of the genes in any two unrelated people are the same.\(^2\) **

If all ethnic groups share almost all their genes, we can make two assumptions about any behaviour that is claimed to be genetically produced:

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\(^2\) ** Although there is a lot of variation in DNA coding reflecting different ethnic groups, these variations produce identical genes, so most genes remain the same.
What do different cultures tell us about homosexuality?

- It will be very predictable, very specific and similar all over the globe.
- It will be present at roughly the same percentage in all cultures.

We also know that many genes, usually hundreds, are involved in human behaviours, and that behaviours affected by many genes will change very slowly over very many generations (Chapter One). That is, they will be very stable for centuries, with only minimal changes from generation to generation. This is true not only in families, but also in cultures.

But if we look at homosexuality, we find none of the characteristics of genetic properties.

- There is a huge variety of homosexual practices between cultures and even within them.
- The prevalence of homosexuality has varied considerably in different cultures. In some cultures, it has been unknown; in others, it has been obligatory for all males.
- There have been, and are, rapid changes in homosexual behaviour, not only over personal lifetimes but also in cultures. Not only that, but entire types of homosexuality have disappeared over the course of just a few centuries.

In fact, anthropologists have found such huge variations in heterosexual and homosexual practice from culture to culture, and such sudden changes in sexual practice and orientation, even over a single generation, that they mostly want to say that all sexual behaviour is learned. In the words of one writer J. Rostand, “In the secret coming together of two human bodies, all society is the third presence.”

Let’s first take a brief look at heterosexuality which has lots of sexual variety.

**Variations in heterosexual customs**

In 1952, two anthropological researchers, Ford and Beach, reported the results of a project organised by Yale University, that surveyed 190 different cultures in a very large cross-cultural study. There was a wide range of heterosexual activity. There was no breast stimulation in six cultures, no kissing in nine, in two others sexual excitement was correlated with
scratching or biting, in one urination was part of foreplay, in another
guest sex was practised (i.e., it was good hospitality to offer your wife
to a visitor). Among the Lepchas, all young girls were sexually experi-
enced by eleven or twelve, and even as young as eight. Bestiality occurred
only erratically in cultures; in some it was unknown; in others, it was
tolerated.

In a survey of preliterate cultures in 1971, Paul Gebhard of the
Kinsey Institute and member of the original Kinsey research team noted
that fetishism, voyeurism, exhibitionism, and well-developed sadomas-
ochism were very rare or absent, appearing only in more “advanced”
societies.

What is sexually appealing in females depends on the culture.
In Arabic culture, a fat woman is beautiful. In ours, a slim but well-
rounded figure may be considered desirable. A broad pelvis is attractive
in some cultures, a narrow one in others. In some cultures, the shape
of the mouth is particularly sexy. In our culture, firm breasts are erotic,
in others pendulous breasts, in others again the breasts are not erotic at
all. In Japanese culture, there is a much greater erotic attraction to the
nape of the neck and to older partners than in ours.

Even a superficial look at heterosexuality reveals a range of prac-
tices too broad to be genetically determined or strongly influenced.

Variations in homosexuality

We have established that a genetically induced homosexuality would
tend to be fairly uniform in expression throughout the world. But
neglecting minor variants two entirely different types of behaviour
col-existed historically—the Greek model, and a little known Melanesian
model—and three co-exist today, the Greek model (secretly practised),
the Melanesian model, and the Western model. The variety of practices
outside these models, and even within the Western model, are also quite
at odds with a genetically prescribed homosexuality.

The Greek Model

At the height of the Greek culture, according to the social custom, an
older married man was expected to take a younger boy as a kind of
squire and have sexual relations with him. Today, the West would call
him a bisexual pederast. The older man would act as a mentor to the
young boy and train him in manhood. He would even find the young boy a bride when he reached marriageable age. Then he would find another boy and start the process again. As described by one scholar:

This sort of Greek male’s ideal picture of himself was that he serviced his wife, had a sexual friendship with his mistress, and did his national duty by teaching younger men how to behave with bravery and honor—which more or less frequently involved buggering them in an idealistic manner. It was only the boy he “loved.”

In the Greek model, a boy starts out exclusively homosexual in his relationship to his bisexual mentor, and then is strongly encouraged to become bisexual at maturity.

In Greek culture, homosexuality between adults—as we have it in the West today—was considered despicable (mainly for the receptive partner). One classical writer, talking of the mature male who was also receptive, said, “we class those who enjoy the passive part as belonging to the lowest depth of vice and allow them not the least degree of confidence or respect or friendship.” Boys were not denigrated for being receptive—it was appropriate to their status.

The Greek model was found in early imperial Greece, medieval Persia, and at various times in China and Byzantium. It was found in the Sudan, in feudal Japan among the Samurai, and in the Libyan desert, where, fifty years ago males “talked about their masculine love affairs as openly as they discussed their love of women.” The Mameluke rulers of Egypt imported young boys from the Asian steppes. The Aztecs and Mayans also subscribed to the Greek model. According to one account from the early 1900s, Arabic speakers in North Morocco believed young boys would not learn the Koran properly unless they had sexual relations with their teachers. Sexual activity with boys or slaves was sometimes regarded as a right among those with power and status. Amongst the Big Nambas in Vanuatu, a father actively sought ‘guardians’ for his sons who would mentor them and have sexual relationships with them.

The Melanesian Model

The Melanesian model is not well known in the West. In it, men pass through three compulsory and sequential stages: passive exclusive
homosexuality, active exclusive homosexuality, and exclusive adult heterosexuality. Many of the cultures practising it were in Papua New Guinea, and perhaps the best-known group was called the Sambia (a pseudonym).

The Sambia believed that boys were naturally girl-like and would not develop manly qualities and sexual maturity unless they ingested semen. The culture required adolescents to be fellated regularly (often daily) by young boys after they were taken from their mothers at about age seven. When the boys reached the initiation rite at puberty, they then had to repeat the process with younger boys as their social duty. They continued to do this throughout adolescence, until they reached marriageable age. Then they had to stop all homosexual activity, become exclusively heterosexual, and marry. Any man who still wished to engage in homosexual activity with those of his own age or younger was considered aberrant, a “rubbish man.” (About 5% continued with the practice.) However two such radical shifts in behaviour in one lifetime would not be possible if homosexuality were genetically-mandated. One missionary familiar with the New Guinean tribal cultures (Don Richardson) suggests the prescribed homosexual behaviour among youth might have been insisted upon by polygynous older men to keep youths away from the young girls they wanted as their own wives. Many anthropologists believe an extraordinary fear of contamination from women in this culture may have contributed to the practice (i.e., marriage was considered highly dangerous). Whatever the cause, anthropologists agree that it was culturally mandated.

The Melanesian model was found mostly in southern Papua New Guinea, and in the islands to the northeast. Overall, some 10-20% of Papua New Guinea cultures fell into this category. Sometimes the sexual expression was anal, sometimes oral. In some places, a youth was not permitted to fellate his friend, but could fellate his potential enemy. In others, boys were “grown” by friends within a group. In the Marind, an older youth who practised pederasty on a younger boy had to later marry that boy’s sister, a practice also followed by the Etoro, Kiwai, and Keraki, except that in the latter two groups, sodomy was practised rather than fellatio.
The Western Model

The Western male homosexual model is comparatively recent and is quite different from either the Greek or Melanesian models, which institutionalised pederasty. The Western model is characterised by exclusive homosexuality between adults, usually of approximately equal status, and an insistence that the behaviour is intrinsic. It is also highly politicised.

The first appearances of the Western model appear to have been adult homosexual networks in cities in France in the fifteenth and sixteenth centuries; for lesbians, some records date from the late 1700’s. Mollyhouses in England, in the 1700s, appear to be another pre-echo of modern homosexuality. These appear to have been essentially “adults only” houses of male prostitution, in which the receptive partners were very feminine in appearance. Homosexual relations between adults do occur in the historical record before that time, but the new element in the Western model is the relative absence of bisexuality and pederasty. Historically, exclusive homosexuality was rare compared with bisexuality.

Greenberg, a well-known researcher of social contexts of sexuality, comments that modern western homosexuality implies that “erotic attraction originates in a relatively stable, more or less exclusive attribute of the individual,” whereas in Western history or in non-Western forms of homosexuality, “distinctions of age… and social status loom larger.” Modern lesbians, however, are uneasy about calling homosexuality intrinsic, politicised lesbians preferring, in their commitment to the empowerment of women, to see lesbianism as a choice. In the Western model, a person identifies himself as “homosexual,” though the word was coined only in the late 1800s.

The Western model tends to encourage promiscuity in males (though AIDS has partially restrained this). A small subset of the male culture encourages a “monogamous” relationship with another adult, though usually with substantial amounts of “recreational sex” on the side. Bisexuality is often viewed as latent homosexuality; there is strong pressure to make a choice to be exclusively homosexual. Though sexual interest in pre-pubertal children is very minor there is significant interest in young post-pubertal teenagers, as far as is possible in Western countries, which universally proscribe it. Lesbianism has, until recently, placed considerably higher emphasis on sexual faithfulness among
partners, though there is a recent new emphasis on sexual pleasure for its own sake. But even among the modern gay community, sexual expression varies from country to country; anal intercourse is more popular in some than others.

The modern homosexual movement is so unusual that some authors have talked about “the uniqueness and particularity of the modern structuring of homosexuality into a gay world compared to pre-capitalist forms.” For instance, in some cities, such as San Francisco, gays have created urban ghettos—entire suburbs in which gays live and provide a full range of gay professional, social, and sexual services.

Rotello, a gay man, in a thought-provoking survey argues that the Western model essentially originated between World War II and about 1970. He mentions that before the war it was medically considered that same-sex relations were safer than opposite-sex relationships with prostitutes—rates of sexually transmitted disease were higher in the latter. Now, it has reversed. He says, “Few groups in history appear to have changed their overall sexual behaviour as rapidly and profoundly as homosexual American men in the decades before AIDS”. He describes it as “a culture of unprecedented sexual extremism”. Although he says “many have less than total control over what they are doing” he is not arguing this is genetically mandated, but implying it is a cultural shift.

The Western model is, therefore, nearly unique historically. Its appearance has been too sudden, its evolution too swift, and spread too considerable to have been genetically produced. Its low occurrence in some cultures, such as Arabic-speaking cultures (which more usually reflect the Greek model), is also inconsistent with a genetically prescribed condition. The lesson of history and culture is that cultural homosexuality is self-taught.

Summary
These three coincident homosexual streams, each very different from the other, in a context in which humankind shares more than 99% of its genes, means homosexuality does not conform to any genetically prescribed model. In a genetic model, homosexual behaviours would be practically identical.

Not only are there quite different models—the Greek, Melanesian, and Western—co-existing today, but there are a myriad of other
homosexual customs and practices, not the behavioural uniformity associated with a genetically dictated homosexuality.

More permutations...

For other cultural variations see the references\textsuperscript{[7,9,10,11,12]}. Many of these are not just variations in individuals but in whole people-groups. They are minor models, but extremely varied. They included the Berdache, a kind of third sex among USA native Americans. Lesbian variations were much less common and Ford and Beach\textsuperscript{3} recorded only 17 cultures in which that behaviour was known at all, and the behaviours were all quite different.

*Cultures without homosexuality*

If homosexuality were significantly influenced, let alone dictated by genes, it would appear in every culture, but in 29 of 79 cultures surveyed by Ford and Beach in 1952,\textsuperscript{3} homosexuality was rare or absent. It was very rare in the Siriono, even though there were no prohibitions on homosexual relationships in that culture. The researcher observed only one man displaying slight homosexual traits but apparently not sexually involved with another man. Homosexuality appears to be historically rare among Orthodox Jews,\textsuperscript{13} so much so that learned rabbis, the interpreters of Jewish law, usually allowed men to sleep in the same bed, because likelihood of sexual contact was considered negligible. Kinsey also found very low homosexual incidence among Orthodox Jews.\textsuperscript{14}

Some anthropologists have questioned Ford and Beach’s findings, believing that irregular sexual intimacy is not something foreign researchers can easily get information about. One sexual anthropologist, Whitam,\textsuperscript{15} thought homosexuality must be genetically enforced because he found it practised in some isolated groups in South America and East Asia who knew nothing of the practice elsewhere.

But evidence from other remote tribes in New Guinea—all genetically related—suggests differently. This evidence comes from missionaries who commonly spend decades living in one culture, far more than almost any anthropologist. The anthropologist will argue that the sexual practices will never be admitted to missionaries; on the other hand it could be argued that missionaries will be unusually sensitive to practices transgressing Christian teaching. Overall they can be considered
as reliable witnesses. For example, in contrast to groups like the Sambia in the New Guinea highlands, where homosexuality was compulsory, only about 2-3% of Western Dani (also in the New Guinea highlands) practiced it. However, in another group of Dani who were closely genetically related, adult homosexuality was totally unknown. Missionaries report that when they were translating the Bible into Dani for this group, their tribal assistants, who knew their own culture intimately, were nonplussed by references to homosexuality in Romans 1; they did not understand the concept.

Another missionary, with the same group for 25 years, overheard many jests and sexually ribald exchanges among the men, but never a single mention of homosexuality in all that time. One man of our acquaintance grew up in a Dani group for many years, and knew the culture and language far better than any anthropologist or his parents. He told us there was no adult homosexuality in this sub-tribe, adding that he would definitely have known about it if there had been.

When Dani went to help with missionary work among the Sambia, they were astounded at some of the homosexual practices they encountered for the first time. Although it is always difficult for a foreigner to be completely sure whether a rare and stigmatised behaviour exists, it is certainly true if three such different experiences of homosexuality can occur in groups of people so closely related genetically, 100% genetic determinism of homosexuality is an impossibility.

Careful recent work\textsuperscript{20} confirms there is a significant number of societies completely without homosexuality and quite a strong relationship to hierarchy, viz., there is more homosexuality in more hierarchical societies. This suggests male-on-male sex in these cultures is more about dominance and submission than genes.

\textit{Sudden changes}

We have mentioned that human behaviours associated with many genes change slowly over many generations or centuries. But history shows us that homosexual practice has disappeared quite suddenly—in some cases over a couple of generations—as the culture has changed. For example, there were many berdaches among the North American Crows in 1840, but by 1900 only one was left. Among the Potowatami, there was a dramatic decrease in berdaches between 1870 and 1930. The
transvestite Koniags of Kodiak Island disappeared between 1800 and 1850. The “men turned women” (manang bali) of Borneo were common in 1850, rare in 1911, and are now unknown. The Samurai pederastic practices vanished long ago. Among the Aymara (South American), the homosexuality, lesbianism, and transvestism recorded in historical times has now disappeared. Tahitian mahus are far less common now than in the late eighteenth century. Anthropologists, in somewhat irritated fashion, attribute many of the changes to Christian influence. In some cases, homosexuality disappeared so rapidly that accurate information on homosexual practices was hard to collect. The customs of the Sambia vanished, under missionary teaching, about 1984. Even at the height of the Sambian pederastic culture, the sudden change required of men of marriageable age from homosexuality to heterosexuality argued against its being genetically innate, and in favour of a substantial cultural basis to homosexual orientation and practice.

But change was not always missionary-mediated. Men’s houses, besides being homosexual hot-houses, were also venues for planning war raids. In some cases, the government stepped in and simply closed the houses down, sometimes jailing offenders. This worked; it also completely disrupted and contributed to the disappearance of pederastic activity in a few years.

The Greek model (cultural pederasty), after becoming popular in Rome, disappeared slowly with time as the culture absorbed several ascetic philosophies. There was a further decline after the Christianisation of the Roman Empire. But even this change over a few centuries was probably too sudden for a genetically dominated behaviour. The sudden rise and disappearance of lesbian practices, such as the Pearl River communities in China and the “Mummies and Babies” movements in southern Africa, were incompatible with any genetic model.

Even within the modern gay scene, there have been changes in practice, which have been far too swift for anything genetically induced. Fisting (insertion of the hand into the rectum) was virtually unknown in the forties and fifties, but a large minority of gays (at least in San Francisco) have now experienced it at least once, and the practice has spread to lesbians with both anal and vaginal expression. Feminine mannerisms have decreased among male homosexuals, and a recent trend has been an exaggerated maleness.
Another trend has been a huge increase in homosexual experimentation by heterosexuals documented, for example, in the Netherlands and in New Zealand (women).

By 2010 the Western Model, in spite of catastrophes like AIDS, seemed fairly well established, and had persuaded itself it was innate.

Summary

When Greenberg comments that “it is reasonable to suppose that if a bunch of Melanesian infants were to be transported in infancy to the United States and adopted, few would seek out the pederastic relationships into which they’re inducted in New Guinea,” he summarises the essence of this chapter. If sexual behaviour were genetically driven, the Melanesian infants would seek out pederastic relationships in their new culture.

The diversity in homosexual activity in different cultures also argues against genetic enforcement. If homo sexuality were genetically mandated, the type of homosexual behaviour would be tightly defined by the genes involved and almost uniform in all cultures. If we want to argue genetic homosexuality, Vines report that the human race shares more than 99.7% of its genes, means that of the 22,500 human genes in the human genome, between 23 and 70 genes would have to account for all the variation in homosexual practice that exists globally, in addition to all other non-sexual differences. This is highly unlikely—probably impossible.

If homosexuality were genetic in origin, it would appear at about the same percentage in all cultures. But this is clearly not so. Among the genetically related tribes of the New Guinea Highlands, homosexuality was simultaneously practiced as mandatory pederasty among the Sambia, was unknown in another group even as a concept, and practised by 2-3% of a closely related group. A significant number of cultures appear not to have practised homosexuality at all.

The rate of change of homosexual practice also argues against genetic causation. Slight changes in practice would appear over 1000 years if there were some strong genetic pressure for it, but not the extensive decline of whole models over several centuries (e.g. the Greek model), not the entire disappearance of homosexuality from some cultures over several generations, and certainly not the very sudden
30-year rise of the modern Western model, with characteristics so different from its predecessors, and its own swiftly changing practices. The Western model is the least likely to be ‘genetic.’

The expression of homoerotic desire does not seem to be genetically imprinted. Sexuality appears to have an overwhelmingly cultural component, ebbing and flowing with changes in cultural values and expectations. Certain sexual expressions may be historical phenomena which flourish for a time because of particular circumstances, and then cease, e.g. Pearl River lesbianism which ceased in 1935. Pederastic homosexuality can be culturally mandated, as among the Sambia, or culturally proscribed, as in the West.

When anthropologists survey the evidence, they are, to a surprising degree, united in the belief that behaviours such as homosexuality and lesbianism are not produced genetically, but by social conditions. If they tried to put a figure on the genetic content of homosexuality, most of them would probably argue for something near zero.

Did their genes make them do it? Not according to the anthropologists.

References


7

Pre-natal hormones? Stress? Immune attack?

Hormones
Many people have wondered if homosexuality is caused by exposure in the womb to unusual levels of male or female hormones. The theory is that if a male embryo is exposed to lower than normal levels of male hormones, or a female embryo to excess male hormones, the child may grow up homosexual. Such exposure to sex hormones may make lower animals bisexual. In this chapter we argue any such effect is small.

In normal development, it takes a natural surge of testosterone in the embryo to turn the female reproductive tracts into male sex organs. You could say that the default sexuality in the womb is female, and that, without the testosterone surge the embryo would remain female.

Treatments for medical conditions during pregnancy and certain rare hormonal conditions in humans have given researchers opportunity to study the effect of high or low levels of male and female hormones on the embryo in the womb and on later sexual orientation. We will look particularly at two of them.

Also, see Chapter Eight for detail of supposed effects on the brain.
Exposure to diethylstilbestrol

Between about 1940 and 1970, diethylstilbestrol, an artificial female sex hormone, was given to pregnant mothers at risk of miscarriage. (It is no longer administered because of increased risks of genital cancer in daughters and sons of these women.) The doses of diethylstilbestrol given to women in the study were very high: 5-250 mg. per day. In much later research, the children of these women were queried in detail about their sexual orientation in the previous eighteen months: fantasies, romantic/sexual daydreams, and many other detailed tests. In two studies, there was slightly more lesbianism than in the controls (a normal comparison group), but two earlier studies found no difference in sexual orientation. A fifth study, the latest and most definitive showed no difference. So, the girls were exposed to levels of female hormone far in excess of anything a fetus would naturally be exposed to, and, even at those very high levels, no effect was found.

A study of twenty boys, exposed to diethylstilbestrol in the womb, showed that none had homosexual tendencies (though one of the non-exposed controls did). This suggests that pre-natal exposure to this hormone does not lead to homosexuality in men.

Adrenogenital syndrome

When girls are exposed to male hormones in the womb, one outcome is adrenogenital syndrome. You met adrenogenital syndrome in Chapter Five. To recapitulate briefly: in the development of a female fetus, the adrenal glands normally produce a hormone called cortisol which is involved in control of protein and carbohydrate metabolism. In adrenogenital syndrome, because of an enzyme deficiency, an androgen is produced instead. Girls exposed in the uterus to this hormone (at nine times the usual concentration) develop unusually large clitorises (more like miniature phalluses), and, if the condition is untreated, can grow up looking very masculine. These days, females with this condition are given life-long drug treatment to counteract the masculinising effect of the continuing androgen production. However, 40 years ago, girls with this condition were sometimes left untreated, and researchers have studied them to find effects on sexual orientation.

Earlier studies showed no effect on sexual orientation, but one study by sex researchers Money, Schwartz, and Lewis came up with a
large group (37%) who were bisexual, but not lesbian, which seemed to show a large influence on sexual orientation. However, a survey of diabetic patients matched with the girls for age and hospital experience came up with identical levels of bisexuality. Unless we argue that diabetes also causes bisexuality, it would appear that common environmental factors in the two groups might have been responsible. These girls were frequently hospitalised and subjected to much medical scrutiny and interviewed about their sexuality. The Money, Schwartz, and Lewis study has also been criticised for poor interviewing techniques, which over-estimated the bisexuality of the respondents. For example, girls with this syndrome who are untreated are acutely embarrassed about themselves and often unwilling to talk about sex at all. In this study, they did not feel feminine and did not have boyfriends. Some of this may have been interpreted as bisexuality. In Chapter Three, we mentioned gender nonconformity as one of the strongest predictors of future homosexuality. These girls felt very different from their peers. They were particularly conscious of their excessive hairiness, which they said was the one thing they would like changed more than anything, even their deep voices.

Environmental factors appear to override hormonal influences, according to another study; no correlation was found between masculine behaviour in girls with adrenogenital syndrome and increased physical masculinisation. In still another study of the condition, lesbianism seemed to be associated with poor vaginal function, in which the girls doubted their femininity.

A Swedish paper found some effect on sexual orientation. Non-heterosexual orientation was reported by 20% of the sample which was significantly different from controls. Meyer-Bahlburg and others conclude that there is definitely some effect, but it is rather modest. In a quite thorough investigation, 31% of women had crushes on other women (but so did 14% of the controls), 9% expressed love for other women which was just significantly different from the controls, 11% had actually had sex with other women, but this was not significantly different from the controls. “Most women were heterosexual, but the rates of bisexual and homosexual orientation were increased above controls not only in women with classical CAH, but also in those women with a non-classic form of the syndrome, and the effect correlated with the degree of prenatal androgenization.” Boys can also have this condition.
They are simply exposed to more male hormones than usual. This might be expected to completely eliminate homosexuality. But, in a sample of thirty, one experienced homosexual attraction.\textsuperscript{11} This level (3\%) is not significantly different from the occurrence of homosexuality in the normal population. The sample is too small to say much more, except that exposure in the uterus to excess masculinising hormone clearly does not eliminate homosexual orientation in males.

These results disproved the theory of pre-natal exposure to excess hormones as an infallible cause of homosexuality. Exposure to excess androgen had no effect on boys, and a modest effect on girls. The girls were exposed in the womb to one of the strongest doses of male hormones known in the scientific record, but a minority became bisexual or lesbian. What, then, can possibly be producing lesbianism in females experiencing normal conditions in the womb? Not exposure to pre-natal hormones, it seems.

In a lesser known 1974 study of 18 young women in Soviet Russia who had adrenogenital syndrome, none showed the slightest trace of lesbianism or lesbian erotic fantasy.\textsuperscript{12} The author attributed this to stricter mores in the Soviet Union. Regardless, it seems the result is sensitive to social setting.

Subsequent papers confirmed more masculine-type play as children, and somewhat less heterosexual interests, but remarkably, in view of the high level of male hormone exposure, in one study of 250 girls, 95\% had no problems with female gender identity.\textsuperscript{46} One conclusion would be that the effects of the hormones were remarkably small.

\textit{Finger ratios and sexual orientation}

In 2000 Williams et al.\textsuperscript{13} Californian researchers, published results which seemed to confirm hormonal influence on sexual orientation. They measured finger length ratios at a gay and lesbian fair, and found the ratio of index/ring finger was significantly more “masculine” in lesbians. Since people are born with these ratios, this seemed evidence that pre-natal hormones, mainly testosterone, were powerfully influencing sexual orientation.

Digit ratios could be measured using a photocopier—an easy laboratory test!—so an explosion of confirmatory studies followed, and were extended to males, but the results for the men turned out to collapse in a
mess of contradictory papers, (one contributing factor to finger lengths for the men was ethnicity), and as at 2010 only the lesbian results are firm enough to comment on.

We must emphasise that the connection between the finger length and lesbianism is actually weak. Van Anders and Hampson\textsuperscript{14} could only explain 6-9\% of the variance (i.e., explain 6-9\% of the lesbianism using finger lengths). Put simply, that is a very weak effect. Also, heterosexuals with the same finger ratios outnumbered lesbians 60 to 1.\textsuperscript{13}

In a rather tour-de-force experiment, Lutchmaya et al.\textsuperscript{15} measured the fetal hormone levels directly in the amniotic fluid of pregnant women and then much later, after birth, measured the digit ratios in the children. This did not look at sexual orientation of course—too early for that—and they found a relationship between the hormone ratios and the digit ratios, but again rather modest. However this result was only just statistically significant and it needs replication. Seventy three percent of the explanation for the digit ratios was not the hormones.

Twin researchers Paul et al.\textsuperscript{16} did a study to find the extent of genetic influence (as opposed to hormonal influence) on the finger-length ratio and concluded that 66\% of the effects were genetic. This is above average, moderately strong, but much stronger than the effect of hormones. The conclusion then is that there is some genetic feature which influences this ratio and that is predominant. Hormone effects are secondary at best, according to the authors.

McFadden\textsuperscript{17} found that the women’s finger length ratios did not correlate with other supposed markers of prenatal hormone exposure, called otoacoustic emissions, fluctuating asymmetry and visuo-spatial expertise. It rather seems whatever the explanation for the effect, it is not very likely to be hormones.

The enticing idea that prenatal hormones are fixing one’s sexual orientation in stone proves only to be a quite weak effect.

\textit{Other pre-natal hormone effects}

Knickmeyer\textsuperscript{18} used the same system as Lutchmaya et al., waited until the children were born, and observed their play. They found no link between pre-natal hormone levels and children’s play whether gender-typical or atypical.
A more recent paper\textsuperscript{19} also looking at sex hormones in amniotic fluid, similarly waited until the children were born and observed at 13 months the tendencies to play with gender-typical or atypical toys. This could be taken as a rough indication of future SSA. Though there were very clear gender-linked preferences for gender appropriate toys, this was totally unrelated to previous sex-hormone levels—except for progesterone, which makes no biological sense and which the authors themselves rejected. However there was a link with family structure—a large number of elder brothers suppressed masculine preference in boys. There was also a strong influence of more elder sisters—they promoted more feminine play in boys. A similar effect was seen for girls—an excess of elder sisters was linked to less feminine play. But these are social effects and much stronger than any prenatal sex-hormone effects. These social effects are not completely consistent with those in Chapter Three and more work is needed.

So prenatal sex hormone exposure did not even affect gender-typical play very much.

\textit{Adult exposure to sex hormones}

Do sex hormone drugs given to adults have any effect on sexual orientation?

It was long believed that homosexuals had lower levels of testosterone (male hormone), or higher levels of estrogen (female hormone) in their bodies, and that lesbians had higher levels of testosterone and lower estrogen levels. The corrective step appeared to be administration of counter-balancing doses of whatever hormone was necessary. But it didn’t work. Male homosexuals given male hormones only became more sexually active, not more heterosexual. So doctors experimented with doses of estrogen in the thirties to see if they stimulated androgen feedback responses. The father of computer science, Alan Turing, arrested for homosexual activities, was required to take estrogen. It had no apparent effect.\textsuperscript{20} Courts ordering men to undergo hormonal treatment to change their orientation eventually stopped the practice as it became clear it was ineffective.

In the literature, as reviewed by New York hormone expert Meyer-Bahlburg, three studies suggested testosterone levels were lower in male homosexuals, twenty found levels in homosexuals were the same
as in heterosexuals, and two found elevated levels in homosexuals.\textsuperscript{21} Another reviewer of the biomedical literature, from the Netherlands, Louis Gooren, remarks, “Not only have the best designed studies failed to find differences in hormone levels between homosexuals and heterosexuals, but...the scientific principles of endocrinology do not make that plausible.”\textsuperscript{3} Nor, he commented has it ever “been reported that sexual orientation underwent a shift induced by the change of levels of androgens and estrogens.”

On the other hand, there is plenty of evidence that hormonal therapy raises or inhibits existing sex drive. Rates of sexual fantasy and orgasm more than tripled in one group of men being treated with androgen for very low levels of testosterone.\textsuperscript{22} This is one of the strongest effects on record for heightened libido. A similar test of women on estrogen replacement therapy showed about a 20\% increase in libido compared with controls.\textsuperscript{23} When they are given to combat advanced breast cancer androgens also increase libido in women.\textsuperscript{24} Some drugs decrease libido. Oral contraceptives tend to lower sex drive by about 30\%, according to one study.\textsuperscript{25} But, even in those cases, habits and mental attitudes can overrule. Even with chemical castration recommended for some sex offenders, some criminal sexual behaviour persisted because of mental habits that had been established. In one classic study, in which men were treated with estrogens and anti-androgens,\textsuperscript{26} some criminal sexual behaviour continued even though sexual activity dropped to about 25\% of normal, and interest to about 60\%. Even physical castration has equivocal effects for many offenders. For some, sexual fantasy and performance decrease quite rapidly: in one study of 2500 sex offenders, repeated offences fell from 50\% to 3.5\%—but a small minority continued to be as sexually active as ever.\textsuperscript{27} It is still possible for castrated men, paraplegics, or eunuchs to have mental orgasms.\textsuperscript{28} But generally apathy sets in.

As one reviewer of the literature on hormones and libido comments, The available literature suggests that humans have not escaped completely from the endocrinological control of sexual behavior and that humans are similar in certain ways to the other mammals. On the other hand it is also obvious that social learning plays an extremely important role in human sexual behavior.\textsuperscript{24}
The placebo effect
Hormonal effects are often small compared with the effects of mental attitudes. People who think a treatment is going to work often show improvement even though the treatment is proven ineffective. This is called the placebo effect. Placebos are inactive substances, without physical curative effects, which are often used in drug trials. For this reason, double-blind trials are now the rule when drugs are being tested: neither the patient nor the researcher knows who received the placebo and who received the prescribed drug until afterwards. Studies of the effect of drugs on libido are subject to a strong placebo effect—people who believe the treatment will raise libido often show increased sex drive, suggesting that state of mind is one of the most powerful influences on human sexuality.

One researcher of the effect of hormones on libido (Brown-Sequard, in Paris) was notorious many years ago for insisting that a preparation of monkey testicles had revolutionised his sex life. Only much later did researchers learn that the testicles had been accidentally prepared in such a way that any sex hormones had been thoroughly eradicated. The effect was all in the mind. “Very many suggested effects on libido are anecdotal, and doubtful, and may arise from increases in general well-being,” says one researcher in the field.29

Maternal stress
In rats, researchers have found a link between maternal stress and demasculinising effects in the sexual behaviour of male offspring. The mother’s stress leads to a delayed testosterone surge in male rats. An East German researcher, Dorner, claimed to have found a similar stress effect in humans during the Second World War. If mothers underwent a lot of stress, he found no heterosexuality in their young offspring, 25% bisexuality, and 35% homosexuality. The remainder were too young to know what their preferences were.10

These were spectacular results, but the study appears to be maverick. Other studies on rats could not find the effect, and stress in human mothers delays the testosterone surge much less markedly than in rats. Dorner has also been criticised for not interviewing the mothers.30 Three other studies on humans did not find any effect.30 A later and more sophisticated study, although it found no correlations with
stress for boys, did find an unsurprising relatively strong correlation between homosexual fantasy and childhood gender non-conformity\textsuperscript{30} (see Chapter Three). Curiously, in this study, there was a moderate correlation for girls between maternal stress and lesbianism, which made no sense to the authors. Girls are not exposed to a pre-natal testosterone surge, so a delayed surge makes no sense in this context.

The latest and biggest survey\textsuperscript{31} basically concludes that there is a weak effect for boys and a more significant effect for girls. A similar survey for the stressful effects of an historic Dutch famine could find no effects.\textsuperscript{32} In no case can the effects be described as overwhelming, which is why it has been so hard to establish. It is another minor factor in the development of homosexuality for a few people.

The Maternal Immune Hypothesis
— the “anti-boy” antibody

Another popular recent theory to explain homosexuality is the “maternal immune hypothesis”. It argues that an immune attack on the fetus by the mother predisposes to SSA.

This section will conclude that the hypothesis is much too speculative.

The maternal immune hypothesis\textsuperscript{33} is that a male fetus may cause an immune reaction in the mother, rather similar to the development of Rhesus sensitivity in an Rh negative mother with an Rh positive baby. In this syndrome the first child is untouched, but the mother has an immune reaction, and any subsequent Rh positive children are severely attacked by the mother’s antibodies, and may suffer neurological damage. The SSA hypothesis is that the mother reacts to the maleness of the first boy and creates antibodies that—like other maternal antibodies—penetrate the placenta and enter any subsequent male fetus, attacking developing brain tissue, particularly male-specific brain sites. Some researchers think lower birth weight is another result of this hypothesis.\textsuperscript{34} The new-born boy is supposed to be predisposed to SSA. However this hypothesis does not try to explain SSA in a first-born and can be calculated to explain only 17\% or less of total SSA.\textsuperscript{35}
According to the theory, the antibodies in the mother increase with each male child, raising the likelihood of SSA with each subsequent birth.

There are several major problems with the theory:

*One:* immunological attack by the mother probably creates more frequent schizophrenia or autism but neither was found in people with SSA when surveyed.

*Two:* the original finding of an excess of older brothers in men with SSA is now looking increasingly doubtful. Many large samples cannot find the effect.

*Three:* if the attack is against male-specific targets then the testes should also be attacked since there are a lot more male-specific targets there. Attack on the testes would result in impairment of fertility in males with SSA. One would be likely to detect increases in four conditions which usually group together—poor semen quality, hypospadias (somewhat feminine deformation of the penis), and cryptorchidism (undescended testes). These three conditions are usually summed up in the following inclusive category: testicular dysgenesis (the testes do not develop). These conditions have many causes, and birth weight is also low. However none of them is known to be associated with homosexuality.

In fact, individuals with hypospadias have slightly increased psychological levels of masculinity. This is interesting because for hypospadias, levels of testosterone are low right through pregnancy to the post-natal period. Is it really possible that the testes in the fetus under supposed immune attack by the mother can still produce levels of testosterone high enough to avoid hypospadias, but low enough to produce SSA? This doesn’t make sense. Orchitis (inflammation of the testes) would be a symptom of generalised immune attack on maleness but neonatal orchitis is much less common in males than homosexuality is.

Work with large samples of adolescents shows there is no difference in age of puberty between SSA and OSA people. But one would expect a later puberty if the functions of the testes are impaired by maternal immune attack.

An attack on “maleness” should particularly affect development of male genitalia in any fetus which is later SSA-prone. But the opposite
has been found. From the data gathered by Kinsey, penile lengths were statistically 0.8 cm longer for males with SSA than males with OSA.\textsuperscript{38}

The biggest unanswered question is: if there is no attack on the testes which have the largest congregation of male-specific targets, why would there be on the brain? The best interpretation is that no such attack takes place.

\textbf{Four:} People with SSA do not show evidence of impaired brain function which would result from maternal attack on the male brain.

Attack on fetal male brain neurology has also been supposed from previous studies to manifest itself in learning difficulties, but in reading and writing rather than arithmetic.\textsuperscript{39} However the known better verbal fluency in males with SSA\textsuperscript{40} and the fact that they are not known for learning difficulties, argues that homosexuality is not a result of any supposed anti-male immune attack.

The most definitive study to date (Flannery and Liderman, 1994),\textsuperscript{41} with a sample of 17,283 mother and son pairs, tested whether enhanced autoimmunity in the mother (a possible measure of attack on the fetus) was associated later in the child with cerebral palsy, mental retardation, seizures, articulation disorder, reading or arithmetic disability, verbal or performance aptitude deficits and ADHD. After controlling for birth factors, enhanced autoimmunity did not correlate with the above neurological problems. This large survey contradicted earlier surveys with poorer control, which gave rise to the idea of such a link (Gualtieri and Hicks, 1985).\textsuperscript{42} Later work shows that the immune reaction was connected with later schizophrenia, autism, and depression, but not changed sexuality.\textsuperscript{49} It seems we can add homosexuality to the list of conditions not related to maternal immune attack. A much more thorough criticism of the maternal immune hypothesis is given elsewhere.\textsuperscript{43}

We have to conclude that there are several layers of hypothesis moving the maternal immune hypothesis from the “speculative” to the “very speculative” and there is evidence against each.

\textbf{Summary}

Although there are some pre-natal hormonal effects on sexual behaviour for lower animals, there is not convincing evidence for such an effect on sexual orientation in humans. The studies examining the effects of high doses of female hormones to pregnant women are particularly
informative because these are very high doses and any hormonal effects on sexual orientation should show up clearly. But the result is a dubious effect on women and no effects on men. Any effects on sexual orientation appear to be better explained in terms of gender non-conformity—a psychological construct. Sex hormones do increase or lower sex drive, but that appears to be about all.

The maternal immune hypothesis seems very speculative, and needs much more evidence before it is taken more seriously.

We leave the last word to several researchers in the field. James summarises the evidence for effects of prenatal hormone exposure on subsequent sexual orientation as “weak”.

In summary, the evidence from prenatal endocrine disorders and from the offspring of hormone-treated pregnancies suggests that hormones may contribute to, but do not actually determine, the course of sexual orientation in individuals with an abnormal sex steroid history during prenatal life.

“At this time, the literature does not support a causal link between hormones and homosexuality.”

Also, “In clinical practice numerous patients are encountered with gross abnormalities of their hormonal profiles. As a rule this does not impact on their gender identity or sexual orientation.”

So, not only your genes didn’t make you do it, it seems your hormones didn’t either. In sexual orientation, the strongest stimulation appears to come from the mind and the environment.

References

Pre-natal hormones? Stress? Immune attack?


Pre-natal hormones? Stress? Immune attack?

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Are brains “gay”?  

You have a sexual brain. Right?  
Women’s brains are innately different from men’s brains. Right?  
Homosexual brains are innately different from heterosexual brains.  
Right?  
Transgendered brains are innately different from heterosexual brains. Right?  
You’re born with these brains, and can’t change them. Right?  
Well—we don’t think so. It’s the subject of a continuing scientific scrap. But this chapter shows that the brain is surprisingly unsexy, and there’s little argument about it. The clearest conclusion from this chapter is that the brain is plastic, changeable, and that you are able to change your brain and your sexual feelings, though this may sometimes take considerable effort.

We’ll try and trace the thinking of scientists about this.

X and Y chromosomes produce sharp gender differences
The X and Y chromosomes are very different. The X chromosome is very long and complex, the Y chromosome is short and simple! You would expect this huge difference to be as strongly reflected in the brain as it is in physical differences between male and female. Figure 21 shows this
clear differentiation in male and female bodies, particularly the genitalia. Intersex conditions are rare.

The old organizational/activational hypothesis

From the mid 1930’s scientists were able to chemically isolate sex hormones for the first time and it was clear they had effects on sexual activity. They also found that in male fetuses but not female, there was a testosterone surge prenatally, at about week 8-24, and it was supposed that this created a male brain, different from a female brain. After WWII the effects of the sex hormones were becoming well known—the mature sexual behaviours of laboratory animals could be altered by injecting sex hormones in young ones. Phoenix et al. summarised findings by creating the Organizational/Activational Hypothesis in the late ‘50s. According to this hypothesis, the brain was irreversibly masculinised by the prenatal testosterone surge, but sexuality was not expressed in childhood and only become obvious at puberty—when there was a kind of activation. There was also an activation at puberty for females.

This idea had immense influence on the research that followed, and at least a thousand papers directly quoted the work. Although the theory

Figure 21. Diagrammatic illustration of intersex frequency (data from Wikipedia, Intersex)
wasn’t established well by the experiments, most scientists believed that
the whole brain was affected by these surges—i.e., was sexy. The theory
still is important today, but the research findings summarised in this
chapter are slowly undermining and supplanting it.

At this time it was thought that the brain was quite statically organ-
ised—for example, a calculating part, always stayed a calculating part—
and any positive change was slow and difficult.

It had long been known from dissections that at birth boy’s brains
tended to be about 5% larger than girl’s brains. This is still well estab-
lished and not controversial. Even then the brain structures appeared
to be very similar, only becoming more dimorphic (differentiated) with
age. Any differences seemed a matter of degree, rather than reflecting
the independent paths of Figure 21, i.e., there was a lot of overlap.

Brain anatomy not strongly gendered

From the mid 1970’s, following many dissection studies on post-mortem
material, researchers started to use a technique called MRI (Magnetic
Resonance Imaging). It became possible to see small details in the brain
and make more detailed comparisons, without exposing the subject
to x-rays. Many researchers then began looking for brain differences
between male and female, homosexual and heterosexual and trans-
gendered people.

There was general agreement that the differences between male
and female structures in the brain at birth were not like the clear-cut
differences between male and female genitalia. Whatever brain struc-
ture was examined there was overlap in size; nearly a complete overlap,
as in Figure 22. This result is well established.

Figure 22 is very different from Figure 21.

Figure 23 shows a similar overlap in adult brains. There is less over-
lap for adults than for infants. In other words most gender differentia-
tion in the brain arises after birth into adulthood, not during a prena-
tal testosterone surge.

Figure 24 shows the brain and parts referred to throughout this
chapter.
Figure 22. Overlap of male and female brain-space volume sizes (ICV) of infant brains.²

Figure 23. Overlap in ICV volumes for adults. The darkest area is the area of overlap
The parts usually thought to be associated with gender/sexuality are the thalamus, amygdala, and hypothalamus. Research in the '70s often found sizes (or groups) of features in adult brains that differed by gender, but this was often not confirmed by repeat surveys using new datasets. When researchers tied to predict the gender of an adult brain based on an apparent set of features they frequently got it wrong. The record high success was 89%.³ There is no argument that predictions from brain features are very fuzzy compared with those from the chromosomes.

By 2016 the situation was not much better. Major studies contradicted each other. The claimed 89% success rate clashed with findings from another large study that could find no differences at all. That latter paper rather controversially concluded,

“...brains do not fall into two classes, one typical of males and the other typical of females, nor are they aligned along a male-female brain continuum. Each brain is a unique mosaic of [male and female] features.”⁵
Another similar paper concluded,

To date there is no consensus whether sexual dimorphism exists, or if such differences are caused by differences in… [instrument] correction methods. ⁶

Another paper,

We do not even know what a female brain is other than that it is not male.⁷

So is there really a gendered brain?

**Gene studies show gender in the brain is very minor**

More evidence accumulated from study of the genes. If there is a very gendered brain one would expect the genes expressed in the brain to be quite different for males and females. And one would expect most of the genes to show the gender effect.

Some researchers studied brains of rats just after birth, during the critical period for gender differentiation.⁸ Concentrating on the small parts of the brain which seemed likely to be involved within the hypothalamus, they found a region called the POA, (the Pre Optic Area) in which only 9% of the genes were differently expressed for male and female. For the medial basal part of the hypothalamus the figure was only 0.9%. Other regions had even fewer differences. This seems to say that almost all of the rat brain is not “sexy” or gendered, and even for the regions which are, only a small number of genes are different for male and female. The rat brain is mostly not gendered, and the division is not sharp. There is almost no such thing as a “male rat-brain” or “female rat-brain” and we need to adjust our thinking.

**Does this hold for humans?**

A parallel to the rat study would require analysing the brains of many recently deceased babies, so is not done. The best we can do is look at the brains of just-dead adults⁹. The work looked at the “sexiness” of other organs too.

**Figure 25** is remarkable because it shows the liver is very strongly gendered, with more than one third of genes differently activated in men and women. But it is a puzzle why is it much more gendered than
the ovary and testis! The leucocytes (blood white cells) are about as
gendered as the hypothalamus—which contrary to expectations was
barely gendered. Genes in the brain, even in the hypothalamus, are not
strongly gendered.

This undermines the whole idea of a sexual brain, and shows our
universal tendency to over-sexualise many things. Yes, the brain is sexy,
but only slightly sexy.

What of the future? Probably in line with the frequent finding of
multiple pathways for development of sexual behaviours we will find
smaller than expected contributions from gendered parts of the brain
but also input from prenatal environment, hormones, birth, post-na-
tal hormones, maternal socialisation, puberty, and sexual experiences.

Given that it is so hard to distinguish a male brain from a female
brain, it is an unproductive exercise to look for differences between
homosexual and heterosexual brains. There have been many papers
on intrinsic homosexual/heterosexual brain differences but there is no
agreement on a clear differentiating feature—even though this type of
research is nearly half a century old. This also holds true for transgen-
dered brains. The adult brains of homosexuals and transgendered people
are reflecting many years of exposure to environmental influences which

Figure 25. The relatively “unsexy” nature of human gene expression in the
adult brain.
almost certainly confuse outcomes. For example, as we shall see, even intense and prolonged thinking about sexual matters changes the brain.

The work comparing homosexual and heterosexual brains has reached a kind of stalemate; a meta review by Byne and Parsons in 1993\(^{10}\) concluded that there was much confusion and little which agreed universally. All agreed however that there was a lot of overlap and differences were nothing like what might have been expected from the sharp differences between X and Y chromosomes in cells.

Reviewing the whole controversial and poorly replicated field, Byne later doubted whether there was even a specifically male organisation of the brain,\(^{11}\) partly because some individual males with extremely low levels of testosterone were completely male and performing sexually as males in spite of it.

Some researchers claim to have found some differences—again not sharp—between transgendered and heterosexual brains, concluding that male to female transsexuals tended to have female brain features. But other researchers have found no differences between male to female transsexual brains and heterosexual male brains. As Byne said 20 years ago (and there has been no change):

No presumed sexually dimorphic cognitive or behavioral brain function has been shown to be independent of learning and experience.\(^{12}\)

**Current thinking on hormone influences in humans**

It is now known that the original early testosterone surge in human males is only the first of four (as it is in rats). There is a second one in the last nine weeks of pregnancy, a third in the first six months after birth,\(^{13}\) and of course the one at puberty.\(^ {14, 15}\) The latter three last much longer than the first one, and may well be predominant influences. If the postnatal surge is blocked in experimental animals the subsequent male behavior is badly affected and this is a current hot topic of research.\(^ {8, 13}\)

The neuroscientists observe that the largest anatomical changes making brains sexually dimorphic (though it takes an expert to tell) are during puberty\(^ {16}\) and the longer the hormonal exposure the greater the differentiation. They believe puberty is only one of the factors in development of male and female and not merely an activation of a previously
existing state as held by the organisational activation hypothesis. As summarised by Kauffman:¹

most identified sex differences in the brain and behavior are produced under the influence of postnatal sex steroid signaling

and

Sex differences in the brain are not an inherent emergent property, but are instead largely determined by extrinsic factors⁷

which being interpreted, means most brain sex differences depend on circumstances after birth, not before. In other words the social environment could be strongly contributing.

Neuroscientists no longer believe that the brain is once-for-all completely organised in a male way during pregnancy, or that brain structure is rigid and unchanging. Authors of about 15 papers in the last decade have independently concurred:

…our current knowledge of sex-based neurobiology has outgrown this simplistic model. Multiple lines of research have contributed to this conclusion.¹⁸

Biochemical male/female differences in young rats depend on environment

We now look at recent research showing that male/female brain differentiation in rats is strongly influenced by the environment, particularly by maternal grooming. This probably has implications for human brain development.

This work on rats by the University of Virginia School of Medicine¹⁹ is important. Researchers couldn't find any male/female biochemical differences in fetal rat brains during all of pregnancy, in the places where they expected them—the amygdala, pre-optic area and hypothalamus. Instead there was a huge male/female difference (30%) in the cortex and hippocampus a few days before birth, as measured by epigenetic markers (see Chapter One). The differences in the cortex and hippocampus seem to lead to male or female processing and memory differences—let's
call them different thinking styles. But many of the markers dropped back to the same levels in both males and females in the first six days after birth, i.e., the difference dropped from 30% to zero. This doesn’t seem to reflect a permanent differentiation between male and female brain structures. However, the 30% male/female difference in a few other markers remained different in males and females after birth.

So, there are some real biochemical differences in rat brains between male and female.

But these pale into insignificance compared with effects on sexuality caused by the environment—especially maternal grooming which we look at now.

Maternal interaction and grooming
Maternal interaction with the newborn rats has a profound effect on the structure of the brain and later full heterosexual orientation. Even rats need their mothers! If rats are deliberately brought up with mothers absent, in an echo of the devastating effects of complete maternal deprivation on children described in Chapter Three, neither rat sex develops full heterosexual orientation but behaves in stunted male and female ways and their brains are observably anatomically and biochemically different from maternally groomed rats. The absence of the mother has led to brain changes.

With this clue from the rats it is probably not surprising that institutionalised children (who have had no mothering) have difficulties in later opposite-sex relationships (see Chapter Three).

The early prenatal, the late prenatal, the early postnatal and pubertal testosterone/estrogen surges were not enough on their own to fully sexually program the rats. They also needed maternal presence and grooming. Similarly hormonal surges were not enough on their own to fully gender-program the brains of institutionalised children who had no mothering. (Though later nurture can help reverse early damage (see Chapter One).

Earwigs brought up in isolation are unable to provide good maternal care and male fruitflies bought up alone, show various behaviour disturbances including a notable increase in same-sex behavior.
Brain development points to strong environment input

When a baby is born, its brain is only one third of the size of the adult brain, and many of the neural connections are only established in the first three years through the stimulation and exercise which babies receive. This proceeds with extraordinary intensity; after only one year the brain is already 70% of adult size. At the point of peak formation of neural paths this corresponds to two million fresh connections every second.

This leads to two other brief arguments in favour of an environmentally-based sexuality.

One: If only about one third of the neurons in the adult brain are present at birth, and the form and structure of the remaining 66% that develop depend heavily on learning, experience, exercise and behaviour, then we might conclude that about one third of brain structure is biologically fixed and two thirds is the result of environmental interaction. We could further argue that because the child experiences so little in the womb in comparison with the bombardment of stimuli he or she begins to receive after birth, the environmental contribution to brain microstructure is in fact, even at a conservative estimate, much closer to 90%. (This roughly approximates the 90% environmental and 10% biological contributions to sexuality proposed throughout this book.)

Two: The DNA in all 23 pairs of chromosomes in a single fertilised cell is three billion rungs long (See Chapter One), but there are 200,000 billion synapses or neuron junctions in the brain. Even if each rung coded for one junction (which it doesn’t, see Chapter One) all the rungs together could only specify about one junction in 66,000! The rest would have to rely on cues from the wider environment. DNA can only specify a negligibly small fraction of neuronal details.

Brain plasticity

It is fair to say the brain, but particularly the immature brain, is like a computer which is constantly reprogramming itself, but including genuinely random actions as well. Particularly in children, neurons fire at random, and if that neural path is reinforced through experiences the path becomes fairly permanent, though not set in concrete. If it is not reinforced, the path becomes hard to excite, and eventually its neurons get pruned. Extensive stimulation is needed or pathways do not develop,
and some periods are more important for certain kinds of stimulation than others. For example, if a child is deprived of light to the eyes in a critical early period, it develops childhood cataracts and becomes blind. If an adult is deprived of light for a few weeks, no such damage happens.23

Similarly, if a Japanese child does not hear the difference between “l” and “r” sounds in speech they will find it hard as an adult to hear any difference, or to pronounce those letters differently, but even so, enough concentrated practice will slowly change that.

The size of the brain does not change after age five but lots of internal structural change occurs in both sexes.24

The maturation of the brain happens in many cycles of neuronal growth and pruning. The last of these cycles is in the early twenties, and cycles can vary from a few months to several years.25

For each growth cycle, experiences reinforce some of the neuronal pathways and the rest get pruned. One consequence of this is the important lesson, Don’t take too much notice of assertions about sexual orientation in adolescence. Change is still happening. For any adolescent reading this—don’t prematurely label yourself as SSA, you will probably change! Changes in adolescence are described in detail in Chapter Twelve.

Changes also take place in the adult brain, particularly with training. Monkey experiments have shown that artificial exercise of three digits on the hand increases the area of the brain associated with those fingers and decreases the other regions proportionately.23

Violinists have a grossly enlarged area of the brain devoted to the fingers of their left hands which routinely get much use. Non-jugglers who learn a juggling routine for three months produce observable small changes in the small-scale structure of the brain, and these changes can also be reversed if juggling stops.26

Importantly, mental rehearsal of some physical skills can be almost as effective as the real thing. Thinking about something changes your brain. One of several examples is internet addiction. It does not involve new physical skills but mainly brain activity, however it causes detectable changes in the grey matter of the brain.27 Now consider: how many times do most people think about sexual activities? How much brain change would you expect? Breedlove23 showed that sexual experience altered neuronal size in rats by 15-20%. Sex, probably even thinking
about sex, alters the brain. As does addictive viewing of internet porn\textsuperscript{28} which has more effect on brain structure and activity than actual sex.

London taxi drivers have an enlarged area of the brain dealing with navigation. Is this innate? No. London bus drivers on set routes did not have this enlarged area, and after retirement of the taxi drivers, the brain area involved diminished.\textsuperscript{29} Taxi-drivers were not born that way, but developed the brain area through huge amounts of navigation and learning, and only maintained it through constant use.

Childbirth changes the brain. Neuroimaging shows the brain in mothers is younger than for childless peers, but it is not clear whether this “baby brain” complaint of new mothers, results from playing with children, or birth trauma, but one might have expected an older brain in mothers from all the stresses!\textsuperscript{4}

One amazing story from Berlin describes a patient with a brain tumour who was operated on progressively over 18 months. Researchers were able to show that the main centres controlling limb movement migrated within the brain through self-reprogramming, so that although the surgery removed some of the former control sites the patient was able to maintain movement skills.\textsuperscript{30}

We change our brains at the micro-level through the way we exercise, and anything we do repetitively especially if associated with pleasure, e.g., sexual activity. So, even if researchers eventually do find real differences in the brains of homosexual people compared with heterosexual, they could well be the result of their homosexual activity, not the cause of it.

There is now a lot of clear evidence that environmental factors alter the brain. Early stress in rats causes many visible changes in their brains.\textsuperscript{31} Huge stress creating Post Traumatic Stress Disorder in humans, causes changes in the brain part called the frontal-limbic system.\textsuperscript{32} Another researcher finds that stress and maltreatment in childhood cause changes in the corpus callosum, left neocortex, hippocampus, and amygdala.\textsuperscript{33} Most of these changes are atrophy of the affected parts.

Perhaps most relevant to the present subject (though it needs replication) is the discovery that sexual abuse of girls causes age-specific brain changes. If it is at ages 9-10 the change is to the corpus callosum, if at 14-16 the frontal cortex is affected.\textsuperscript{35}

Sexual experience affects the brain—no surprise!
The brain that changes itself
We strongly recommend the book by Doidge: *The Brain that Changes Itself.* This remarkable but very accessible work describes the overthrow of 20th century beliefs about the unchanging nature of the brain. The brain can change a huge amount, very encouraging news to anyone who is stuck in any habitual behaviour.

Doidge gives numerous illustrations of the brain’s plasticity. One is about people who get intense pain in phantom limbs which “remain” after amputation. There is no longer any physical reason for the pain, except within the brain itself. About half the patients were able to get relief from, e.g., cramp in a phantom limb, merely by intensely imagining over a long time that the imaginary limb was in a different position. In other words imagination changed the brain’s perception of pain. He describes how intense exercises targeting weakly performing areas of the brain can make differences which seem almost miraculous, and how any vigorous training causes changes in the observed microstructure of the brain. The level of training needed to make the changes was tiring and extended.

Doidge emphasised the neurological principle coined by well-known neuropsychologist, Hebb: *Neurons which fire together wire together.* By deduction, in human sexuality, this means that if something non-sexual is often associated with sexual arousal it will tend to become part of it. In brain maps genital response regions lie alongside the response region for feet, and Doidge wonders if this might relate to sexual fetishes involving feet. (And could it explain the Victorian ankle fetish?) It also becomes reasonable to deduce that, e.g., if intense emotional focus on someone of the same sex is triggered together with sexual excitement, and frequently repeated it could become homosexuality.

Brain plasticity means it is not unreasonable to expect that homosexuals could become more heterosexual.

Doidge shows that although various skills and behaviours are organised in distinct brain regions the micro-details (or “brainmap”) are dynamic and changing on a day-to-day basis. If one part of the brain is suddenly not used, the areas around it quickly start to recruit these unused brain pathways for other purposes, reprogram them and use them, e.g., parts of the brain involved in the functioning of a lost limb can be re-purposed; parts of the brain used in a now-discarded
skill can be recruited for another very different skill. Doidge sums up
the extraordinary plasticity of the brain with the words, *Use it or Lose
it.* (Or we could say, use it and grow it.)

Sometimes the loss seems permanent—a childhood language can
get completely lost, though “fossil” inaccessible or forgotten language
has been found in the brain, and the person has no conscious memory
of it. Sometimes it is partial—a musician may find it hard to retrieve
accurately a difficult musical piece after some years. But it will return
quickly if practised again. Some development windows in early life may
even re-open in adulthood given the right circumstances.37

Even if part of the brain is strongly associated with a particular
sexuality it should be possible to change it. Stopping a sexual activity
and avoiding sexual stimulation, while giving oneself to another absorb-
ing brain activity for months, e.g., thoroughly mastering a musical
instrument, would lead to a diminishing of the intensity of that sexual
response. Months is about the time-scale of first significant change. That
can be true for learning a musical instrument too! But detectable struc-
tural change for some activities can happen in as little as two hours.38

It could be expected—though this is not mentioned by Doidge—
that any brain structures associated with sexual activity would be much
changed in those very elderly people for whom such activity has long
ceased. MRI scans already show declines in brain activation in response
to erotic stimuli in middle age compared to younger ages.39

Doidge’s conclusion about sexuality is that human libido is not a
hard-wired invariable biological urge, but can be curiously fickle, easily
altered by our psychology and the history of our sexual encounters
and “It’s a use-it-or-lose-it brain, even where sexual desire and love are
concerned.” This would apply both to same-sex attraction and oppo-
site-sex attraction.

If we train hard enough, an activity can become automatic and we
pay it less conscious attention. Details of driving, throwing a ball, read-
ing, even tying shoelaces, don’t and often can’t demand full attention.
Martial arts experts strive to reach this level of automatic response,
because there is no time in a fight to work out the best counter-attack.
It is also particularly true of playing a musical instrument. Many of the
basic techniques like chords, scales and arpeggios, are so deeply learned
that we don’t think about the details and indeed can’t if the music is fast. Doidge says this degree of training alters brains so much that after death the brain of a musician is uniquely different from other brains.

Studies show that we make decisions, e.g., to move an arm, a fraction of a second before they are conscious. We have delegated even some of our decision-making to unconscious levels. This does not mean free-will is an illusion, but that we have trained ourselves to the point that the response is ingrained and automatic; part of us is now a well-functioning machine.

In the same way it can seem that sexual orientation is so deeply embedded that it is innate. But, really, it is no more innate than any complex skill we have spent a long time developing. From what we now know about the brain, it is possible to reprogram it by changing our thinking, fantasies and behaviour. It may take several years of intense effort but our sexuality is not dictated by our brains—instead our brains can begin to reflect changes in the way we live.

Summary

Scientists have not been able to find clear structural differences between the brains of boys and girls at birth except size. At that stage of life their properties and functions and behaviours overlap almost entirely. Male and female behaviours—let alone homosexuality and heterosexuality—are not hard-wired into the brain at birth.

Mother-child interactions after birth influence the brain structure and future sexual orientation. This means early hormonal effects on the brain are far from inevitable. In fact, only one third of the brain is formed in a new-born child; the rest is developed through learning and experience (environmental input).

There is strong evidence that very little of the brain is actually “sexy,” and, e.g., the liver is much more so. Male/female differences are generally much smaller than expected.

Many early attempts (in the nineties) to find male/female, heterosexual/homosexual differences in adult brains based on size of structures gave contradictory results. Where differences appeared to exist, further studies failed to reproduce them, or they could be attributed to influences in the environment. There is no well-established method for correctly
differentiating adult male and female brains, let alone homosexual or transgendered brains.

We can be confident that any replicable male/female differences found in adult brains (or between “homosexual” and “heterosexual” brains), will have been shaped largely by learning and behaviour.

What learning and experiences do to the brain is not set in concrete. The dynamic nature of brain connections, means that new neuronal pathways can be formed and old ones reshaped. Intensive exercise, training or imagination changes the brain microstructure.

We are not victims of our biology or the experiences which shape the detail of our brain. Anatomy is not destiny; change is always possible. The brain is plastic and is in a constant state of change. Indeed the question is rather: what change is not possible?

We are not saying that the structure of the brain you were born with has no effect. It has. It can be profound. But that structure can also be profoundly changed, and we don’t yet know the limits. They are probably sky-high.

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In 1993, the West was told that a scientist had discovered a “gay gene”—a gene causing homosexuality. The details were confusing for non-scientists, but the headline stuck. For Mr and Ms Average Citizen, it seemed that homosexuality might be genetic.

Actually there was no “gay gene.” Even the scientist referred to, a gay man, Dean Hamer of the United States National Institutes of Health, never claimed to have found a gene determining homosexuality. “We have not found the gene—which we don’t think exists—for sexual orientation,” he said. However, he claimed to have found evidence that some male homosexuality was passed through female members of a family. More specifically, he claimed to have found a linkage between homosexuality in males and a small stretch of the DNA on the X-chromosome.

This chapter will look at these studies, but as discussed in Chapters One and Eight, scientists now believe that thousands of genes may be involved in almost any trait and that gene expression depends on environmental events and even social interactions. Gene patterns may be a recipe for tissues and bodies, but don’t dictate behaviours. Though much research has tried to find specific SSA genes, none have yet been conclusively found. Any connections are very weak, indirect, not specifically
sexual and we’ll see that a very large 2019 study shows an alarming amount of early work was simply wrong.

**Gene linkage studies**

Hamer’s work falls into a category of research called “gene linkage studies.” There was a surge of research in this field in the late twentieth century but because thorough “whole genome” scans are now the norm, gene linkage studies are becoming rather passé. A whole genome scan means all the genes are examined; a gene linkage can only look at a few at a time.

The first most spectacular linkage study, was the discovery, early in 1993, of a gene responsible for Huntington’s disease. The gene had already been tracked down to chromosome 4, but it took six teams of workers at ten different institutions ten years to find whereabouts on chromosome 4. Over the succeeding decade, researchers also identified genes causing cystic fibrosis, muscular dystrophy, and other diseases.

From 1990 to 1993 biologists had astonishing success mapping the human genome (on schedule and within budget!) and analyses are still being published. In one five year period near the end of the nineties, the genes corresponding to 1450 physical conditions were identified and their precise location on various chromosomes found. Inspired by these successes, some scientists began talking optimistically of uncovering the genetic basis to human behaviours in the same way. This is what Hamer tried to do, and what other scientists, called behavioural geneticists, had attempted to do before him, but with scant success.

*What happens in Gene Linkage studies?*

In linkage studies for behaviour, researchers look for an extended family with an unusually high incidence of some behaviour, such as bipolar disorder, and then take samples of tissue from all available members and analyse the DNA, looking for segments in common using sets of tiny, synthesised DNA segments, called “markers”—an identical set for each person. These tiny markers are configured in such a way that they attach in a lock and key fashion to any stretches of DNA that mirror the markers; they usually contain a small range of genes. Searching for one gene in 22,000 is worse than looking for a contact lens in a swimming pool, but, in this way, segments of DNA (also containing “irrelevant”
The “discovery” of the “gay gene”

The “discovery” of the “gay gene” (genes) can be found in different people. If the same sequence is associated consistently with a given trait, then researchers assume the marker lies close to the gene that codes for it, along with the other irrelevant genes. At that point, the researchers believe they have found a linkage.

The strength of linkage analysis is in studying physical diseases that have distinct symptoms and are caused by a single dominant gene. When they attempt to link behaviours to a single gene, they run into a volley of scientific scepticism, for several reasons.

First, no mainstream geneticist believes that behaviour is linked to one single gene (see Chapter One). “It’s very rare to find genes that have a specific effect,” says Harvard biologist Balaban. Second, in the word of one writer for Science, “the field of behavioural genetics is littered with apparent [gene linkage] discoveries that were later called into question or retracted.” It was only in the first decade of the 21st century that gene linkage studies became more reliable. Unfortunately the supposed SSA—genetic link was publicised before that time. And, as mentioned, the most recent studies have moved beyond linkage studies to very detailed scans of the entire genome.

In the next section we survey gene linkage studies that have tried to identify genes linked to schizophrenia, to put in perspective what is needed for success in gene linkage studies.

About the time Hamer sought to associate SSA with a section of the X-chromosome, linkage studies were scientifically dubious, but seemed worth pursuing although similar gene linkage studies on schizophrenia and alcoholism had given rather contradictory results.

Schizophrenia

Gene linkage studies on schizophrenia blossomed with the completion of the human genome project. Using markers, many regions were found on various chromosomes which correlated strongly with schizophrenia, and studies on fresh family lineages and families from other ethnicities often confirmed them, though there were puzzling lacks of confirmation from time to time.

However the results for some regions of the DNA seemed so convincing that scientists began looking for specific genes within them. By August 2005, at least 25 chromosome regions were thought to be involved, and an equal number of genes on them were being investigated.
Of these there was strong evidence for involvement of 4 genes and “promising but not compelling evidence” for a fifth. Some of the results were described as “very robust.” This was a good consensus to emerge from a welter of initially inconsistent gene linkage studies. The work had progressed so far that some researchers started to experiment with drugs which interacted with the products of the genes known to be involved, in the hope of reversing or at least reducing the progress of schizophrenia.

But this confidence proved to be completely ill-founded. By mid-2010 “whole genome” scanning had thrown the gene linkage results into embarrassing disarray. In “whole genome” scanning—rather than using markers which result in rough screening only—all the genes are scanned in extraordinary detail, nucleotide by nucleotide. Nucleotides are subunits of DNA. There are hundreds of nucleotides in a single gene, each made up of a nitrogen base, a sugar and phosphate.

Enormous multicenter efforts scanned the entire genomes of 7662 subjects and 29053 controls in one study alone; a second involved 3322 subjects and 3587 controls, and a third involved 8008 subjects and 19077 controls but altogether they could not confirm any of the previous gene-linkage work, only labelling them promising. The detailed saga is recounted elsewhere. This was embarrassing because so much previous work now seemed premature. One million gene variants were examined, involving most common variations of DNA nucleotides. They found absolutely unequivocal evidence of a connection to variants in a gene on chromosome 6 linked to immunity, and to three other completely new genes, two called transcription factors (TCF4 and ZNF804A, the latter a “zinc finger” protein because of its composition and shape) and the last, called neurogranin, but, disconcertingly, no one had previously suspected them of being involved. The transcription factors were used by the nucleus to read the DNA sequence and neurogranin is a brain-specific protein connected with biochemical control of calcium. Like the fruit-fly case we described in Chapter One, why these genes should be important in schizophrenia is not at all obvious, and links will be very indirect.

Schizophrenia is certainly reliant on multiple genes, because four genes were found and others suspected: but these significant genes found only account for 3% of schizophrenia. The effect is weak. This is a vivid illustration of how difficult this field is.
Hamer’s Study—SSA

Compared with the scale and outcomes of the schizophrenia project above, early efforts which attempted to link genes with SSA now seem embarrassingly small, very naive and hyper-optimistic. Moreover, Chapter Ten shows the genetic contribution to SSA calculated another way is relatively low, lowering the prospects of success from gene studies.

However: To find the homosexual gene or genes, Hamer and his colleagues first recruited 76 men, who identified themselves as predominantly or exclusively homosexual. They found 13.5% of their brothers to be gay, much higher than the 1% occurrence of exclusive homosexuality in the general male population, and also found a higher level of homosexuality in maternal uncles and the sons of maternal aunts. They then recruited 38 families in which there were two homosexual brothers, suspecting this would show more clearly the effect of homosexuality and Hamer searched for a linkage on the X (female) chromosome.2

Hamer claimed to have found a “statistically significant correlation” between the homosexual orientation and a genetic sequence on the tip of the long arm of the X chromosome, an area called “Xq28”. Hamer published his paper in Science, in July 1993, and immediately became a controversial figure in the scientific community. Numerous letters to the journal Nature were mostly critical.

In the meantime, Hamer11 and colleagues replicated their study using a new population. This time, the results were less impressive—only just statistically significant, but the replication was promising and reassuring.

Hamer's study on the “gay gene” was then contradicted in a gene linkage study12 published in Western Ontario, headed by researcher Rice. Rice found no trace of an association between homosexuality and the genetic region Hamer and his team had pinpointed. Even when the results from all the Hamer and Rice studies were combined, there was no significant association. Hamer argued that the Rice team result was inadequate because they did not select homosexual men with an excess of maternal homosexuality.

Then a “whole genome” study13 appeared from the National Institutes of Health in Maryland, with collaborators from several parts of the US. It was much larger than any preceding gene linkage study.
The first author was called Mustanski, and Hamer was included in the author list, though not leading the study.

According to the results in the paper, no part of the entire genome was statistically significantly linked with SSA. One peak on Chromosome 7 (region 7q36) approached statistical significance but the result did not survive replication by a 2014 study.

Then, using a different method, the Rice team\textsuperscript{10} could not replicate the Mustanski results. So, more conflict!

In mid 2014 a Chicago researcher called Sanders headed a team which published\textsuperscript{8} the result of investigating the genetic links yet again, working on a sample of 409 SSA brothers. They found more confirming confirmation of the Xq28 linkage, but only suggested specific genes which might be involved. Their comment is worth citing, “We also emphasize that genetic contributions are far from determinant but instead represent a part of the trait’s multifactorial causation both genetic and environmental.” Translation: genes as a whole are a minor contribution; there are many factors involved.

Much earlier Hamer’s group attempted an SSA-gene linkage study on lesbians but did not find a link between parts of the X-chromosome and the presence of lesbianism in families.

A 2015 Chinese study showed a connection between a gene called COMT and sexual orientation,\textsuperscript{7} but calculation shows the effect size is weak.

\textbf{The large 2019 genome/SSA study}

In 2019 the results of a very large study appeared in \textit{Science},\textsuperscript{14} one of the top scientific journals, which claimed discovery of five genes connected to SSA. They paid careful attention to statistical validity and the gene discovery is probably correct, but their definition of SSA is surprisingly poor, and the connection doesn’t mean very much. Perhaps they will publish better material in future.

Where can you get the tens of thousands people needed for such a gene/SSA study? Today it comes from places many readers will have patronised—the half dozen companies analysing DNA for private clients. Most results came from the UK Biobank company; nearly 409,000 volunteers had agreed to a survey on sexual matters. Results added in from
other related companies and surveys increased this to 477,000—nearly half a million. We'll call this the Biobank study.

More than twenty authors are listed: from the USA, Sweden, Denmark, Netherlands, UK, Australia and a combined research group from the USA 23-and-Me genome company. This is Big Science.

The most serious problem is that researchers divided the group into two classes: those who never had a same-sex partner, and those who had at least one. Previous surveys describe this as a mediocre classification.

Even Kinsey in the late 1940's talked about those who had merely incidental SSA experience: one or two experiences and nothing thereafter. That's the present case. Researchers know very well that many of these sorts of encounters are exploratory or even sexual abuse, and not a continuing sexual orientation. In fact, in this study, they comprise most of those with some same-sex attraction. Laumann et al. (Chapter Two) found 7% of men had reported one or more same-sex partners but those active at the time of his survey were only 2.9% and exclusive SSA men were about 1%. Most had not persevered. In the same way Laumann et al. found 4% of women had one or more same-sex partners, but those active at survey time were only 1.8%. This means the Biobank study is mostly about sexual explorers. It's dubious practice to label them all “homosexual”.

The researchers warned there were two qualitatively different classes of people—those slightly non-heterosexual, and those exclusively homosexual. The volunteers overall have a rather weak same-sex drive. Why didn't researchers concentrate on those with a strong drive? Well, that would probably have reduced their sample size by a factor of 10, which would make the results much less clear. So they faced a trade-off between mediocre sample description or mediocre statistical test power.

“Getting your DNA done” is quite popular and perhaps the survey will be repeated when there are ten times as many people available and a large active SSA group, but let's see what was possible even with the available sample and doubtful sexual classification.

The researchers present the Biobank results first, and for men they found a connection between four genes and some SSA experience. Embarrassingly, these genes had never been implicated in nearly a dozen similar preceding studies probably involving several million dollars of effort. All the previous work was useless because samples were too small,
but this was realised clearly only in the last five years or so. Even more embarrassingly, the controversy about the genes on the X-chromosome, particularly the XQ28 region was pointless—none of the four genes Biobank researchers found were on the X-chromosome.

For the very first time researchers found three genes correlated with SSA in women, and two of these were also found in men. No previous work had found any gene connections for women. There was some overlap then, between genes for men and women and SSA, but overlap between men and women for most unrelated traits in other studies was much higher. Could SSA be partly different in men and women? Quite reasonable.

When the researchers checked the results using much smaller samples from other sources, and a total of 15000 individuals, they confirmed three of the results, which is a good test of reliability, but the Biobank large sample results were far more reliable.

Two of the genes were connected to smell sensors. Could this be SSA related? But previous studies could also point to vague connections between their spurious genes and various functions and were wrong. So even present alleged connections should be treated rather sceptically.

At this point you may be thinking, “Well, there may not be one unique gene, but a handful. OK, so a small cluster of genes are responsible for SSA? And they have a powerful effect?”

No, they don’t! The researchers were able to calculate the strength of any effect, and an individual with one of the four genes is at most 0.4% more likely to be SSA. Yes, almost negligible. But it is typical of what gene researchers find, which is why they conclude that many, many genes influence traits, each with a very small effect strength. For the Biobank study, the researchers were able to show that the minute influences were spread fairly evenly among all the chromosomes, again confirming there were very many genes and on all the chromosomes.

But what was the sum of all these many small influences? The researchers were able to calculate a range depending on various assumptions and it was 8-25%. In the paper they imply a typical estimate of the total influence strength would be 10%—as derived elsewhere in this book. If 0% is no influence, and 100% is a dictatorship, then 50% would be a medium influence, but 10% is quite weak—and obviously quite indirect.
If there was a strong physical effect on SSA, you’d expect special genes concentrated in parts of the body, maybe within the brain or in the sex organs. The researchers tested individual tissues for other genes suspected of some correlation with SSA but didn’t find it, in fact they found very few correlations with other physical traits (an exception was a finger length ratio in women).

The researchers identified openness to new experiences and risk behaviour in their group of people who had at least one same sex encounter in the last year—though this was not a genetic test. But it again raises the issue of whether their study was of people with SSA or of sexual explorers.

That could also account for the partial gene similarities (overlap) between the men and women—i.e. the common factor is openness to new experience.

So, the Biobank Study, though impressive in its reach and resources, is limited in reaching conclusions about genetic effects on SSA.

**Summary**

The authors of the paper also strongly emphasise a DNA test for gayness is not possible. The scientific community realises that “our genes do not make us do it”. Hamer has always believed that. To give him the last word: “There will never be a test that will say for certain whether a child will be gay. We know that for certain.” This means as clearly as anyone could state, that no-one is born gay.

Those who believe that homosexuality has psychological and sociological explanations have no difficulty with the possibility of genetic linkages to homosexuality. They would argue that any genetic link to a physical characteristic that might heighten a person’s sense of gender non-conformity (a strong known predictor of later homosexuality), could be held to be a contributing factor to later homosexuality. In a boy these might be, e.g. genes related to slightness of build or poor physical co-ordination (making a boy poor at sports). In a girl they might be factors like atypical physical strength, shape, height, or weight, or a more masculine finger-length ratio. Links? Yes, but weak and indirect.
References

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

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*†† 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.
Twin studies—the strongest evidence

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, examining the important Australian Bailey et al. (2000) 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](image)

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.

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.

‡‡ In 2002 Bearman and Brueckner4 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](Taking_from_PubMed.png)

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

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. 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.” 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.* 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\textsuperscript{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.\textsuperscript{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.”\textsuperscript{21}
Twin studies—the strongest evidence

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.

Figure 29. Factors thought by gay and lesbian people to have had some causal connection to their SSA
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 found a result of 18-26% which is much lower than one would expect. SSA should be about the same or lower. Whitehead found a similarly low result from considerations about the timing of puberty.

For homosexuality, the last half dozen twin studies (1998-2013) 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 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. The degree of genetic programming must be very low for SSA compared with puberty.

The technical details of these studies are discussed elsewhere 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.” 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, depression, altruism, religiosity, fundamentalism, psychological inpatient care, fear of the unknown, perhaps alcoholism, and most interestingly homophobia! 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.

“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
Twin studies—the strongest evidence

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. 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.

References


Path Analysis: Social factors do lead to homosexuality

Another method that has been used in debate about the origins of homosexuality is a statistical tool called path analysis. As you might expect, path analysis tries to identify the most common path or paths leading to a particular condition, e.g. cancer. Path analysis produces a diagram, e.g., Figures 30 and 31, that visually demonstrates the network of causes and attempts to assign a relative importance to each cause. The method works best when there are a relatively small number of causes—so does not appear an ideal tool for the study of homosexuality. We’ll see that in fact it fails to find a few predominant causes but does succeed in showing a multitude of causes, or paths.

Two major studies of homosexuality have been attempted using this method: one by a team, Bell, Weinberg and Hammersmith, using data gathered in 1969-70¹ (published in 1981), and another by Van Wyk and Geist published in 1984,² using male and female data collected by Kinsey in the forties of last century, but corrected for bias.
Path Analysis: Social factors do lead to homosexuality

Figure 30. Path Analysis, Male Homosexuality
Figure 31. Path Analysis, female homosexuality
Study one

The 1981 study is particularly important because it has been consistently misinterpreted. The usual claim is that it disproves any social cause for homosexuality. This is both completely right and completely wrong at the same time! What it shows is that social causes as a whole are significant, but a social factor which may be important to one individual will not be important to the majority with SSA, i.e., there are a multitude of paths, each very important to the individuals concerned, but not important for all. However a few common themes still emerge. The work also confirms that chance (random events and reactions) is very important. An in-depth critique is available elsewhere.9

Bell et al. designed a 175-page questionnaire intended to test current sociological and psychological theories about the causes of homosexuality and provide information about the categories, called “variables,” that appear in capital letters in Figures 30 and 31. (We will call them factors.) For example, the question, “During the time you were growing up how afraid were you of your father? Very much, somewhat, very little, not at all,” provided information for the factor, negative relationship with father, in Figure 30. Some questions were open-ended questions, such as “How did you feel about dating?” They tried to cover all popular psychological theories about what caused homosexuality. When all the answers were in, the team combined many answers into much fewer major factors and used a complex statistical procedure to see which of the different variables were most common, attempting to link them into a causal pathway.

Some paths showed up more strongly than others, but even the strongest variable was rather mediocre as a predictor; child gender non-conformity (“sissiness” rather than modern Gender Identity Disorder) for boys was the strongest single variable. But on a scale of 0 to 100, it measured only 12% as a direct contributor to homosexuality. This means that few sissy boys become homosexual as a direct consequence of gender non-conformity alone. However when combined with other indirect paths its role in the cumulative effect is much higher.

The authors concluded: “What we seem to have identified… is a pattern of feeling and reactions within the child that cannot be traced back to a single social or psychological root; indeed homosexuality may arise from a biological precursor.”
Critics of psychological theories of homosexuality interpreted the study to have proved there is no social or familial basis to homosexuality. They then tended to emphasise and research biological causes—with little success as we have discovered 30 years later.

**Study two**

The second study, by Van Wyk and Geist, was limited to the questions Kinsey asked. But Van Wyk and Geist had Kinsey’s huge sample to work with: 3526 females and 4143 males, and Kinsey’s questioning had been wide ranging, so any common features could be expected to emerge. Their path analysis put an emphasis on early sexual experiences and put “gender related” and “familial” (family-related) variables second and third, respectively, on the list of influences. But no single variable scored higher than 10%, and most variables scored significantly lower—around 3.6%. On a scale of 0 to 100, poor relationship with father accounted for about 3.9%. For females, family related effects were found to total less than 1%. Just like the Bell et al. study, this study showed any particular path was important to only a small number of people. However, Van Wyk and Geist commented on their study:

> The degree of similarity between the results of this study and that of Bell et al…is striking. In each case sexual experience variables accounted for the most…[adult homosexuality] followed by gender-related variables and family-related variables in that order.²

The research community was puzzled by the results of the two studies, because social factors did not clearly predict adult SSA. They thought the clinical psychologists with their vivid case studies must simply be wrong. The studies were fuel to those who rejected an environmental explanation and sought a genetic or biological one. So what was going on? Let’s look in more detail at the studies.

**Bell et al**

**Male homosexuality**

Bell et al. actually discovered a number of paths to male homosexuality (Figure 30), and the three most common lend support to psychological
theories suggested in Chapter Three (cold father, negative relationship with father, negative identification with father, childhood gender non-conformity, homosexual arousal in childhood or first homosexual experience in adolescence). Childhood gender non-conformity was made up of three factors: how much boys disliked typical boys’ activities, how much they enjoyed typical girls’ activities, and how “masculine” or “feminine” adult homosexuals said they had felt growing up.

Bell et al. comment,

Childhood gender non-conformity turns out to be a very strong predictor of adult sexual preference among the males in our sample. With total effects of 0.61 (on a scale of 0 to 1) it ranks first in importance among our 15 developmental variables and appears to influence a variety of explicitly sexual variables; in fact it has a direct connection to every single variable following it on the path model.

They go on to outline the path, remarking that boys who did not conform to the childhood gender stereotype were more likely to feel sexually different, either in childhood or adolescence; more likely to experience homosexual arousal in childhood or adolescence; somewhat more likely to have some kind of homosexual genital activities in childhood; and more extensive involvement in homosexual activities in adolescence. “Each of these [factors] in turn makes adult homosexuality more probable.”

**Lesbianism**

In women, the effect was similar (Figure 31): the most common path linked the factors unpleasant mother, hostile rejecting mother, negative identification with mother, childhood gender non-conformity, adolescent homosexual involvement, and adult homosexuality. Again, say Bell et al., childhood gender non-conformity was the second strongest predictor overall (53%), though it was less likely to develop among those girls who reported “much identification with Mother,” and was particularly strong for homosexual women (48%) who had masculine pastimes in childhood.

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§§ These percentages do not appear in Figures 34 and 35. However they are derived from them but by a statistical procedure too complex to go into here.
These results were significant
Bell and Weinberg found 76% of adult homosexuality could be explained by their paths. However, they interpreted this result as simple “tracking” from adolescent to adult homosexuality, i.e., a pattern started in adolescence continuing into adulthood, and dismissed it. Similarly they found “tracking” from childhood SSA to adolescent SSA. As a result, when they summarised their results for men and women, they said that none of the factors linked into significant paths. But their “tracking” explanation has been shown, at least since the 1994 study by Laumann et al., to be incorrect. The vast majority of SSA adolescents will not become SSA adults, and adolescent SSA itself is extraordinarily unstable (see Chapter Twelve). We calculate that even allowing for tracking, these social factors all taken together still account for about 30-40% of adult homosexuality, but there is no single predominant path. Rather than concluding therefore that social factors were not important, they should rather have commented that social factors are important, but no one factor is important to all. Both are true—social factors as a whole are significant, but no social factor by itself is significant for the majority. A fairer critical interpretation of their results is that the most common paths for male homosexuality and for lesbianism (described above) are among the most significant of the network of paths discovered, and will be very important for many individuals.

Why weren’t they more significant?
If you look at Figures 30 and 31, Childhood Gender Non-conformity, you will notice a vertical arrow: 0.88 in Figure 30, and 0.87 in Figure 31. Put a little simplistically, this is the amount of gender non-conformity in their sample that Bell et al. found their model was unable to explain. The figure actually translates to 77% (the authors explain this figure is calculated by squaring the figure on the vertical arrow). That is, it was not clear what led to childhood gender non-conformity in 77% of cases. The vertical arrow appears against most of the variables and the unexplained causes are high. Some calculated percentages of causes not explained were: Homosexual Genital Activities in Childhood (Figure 30) 92%; female Childhood Gender Non-conformity (tomboyishness) 76%; female Adolescent Homosexual Involvement 58%. This could mean either, or both, of three things—wrong questions were
asked, or a large number of individual paths were involved, or a lot of chance was involved.

The right questions and unique factors

One reason could be that the researchers did not ask the right questions. Three general factors which have since proved important are

1. Where were you brought up, large city, town, or country? (See Chapter 3, Figure 19). This relationship for men is “marked and strong.”
2. The presence in the original home of adult mental problems.
3. A family member in prison.

These were individually statistically significant. See also Chapter 3.

Individual and unique factors, which couldn’t easily have been elicited even by 175 pages of questions, contribute to the variables. If different questions had been asked, or if respondents had been able to offer their own opinions as to why they grew up homosexual, their responses could well have strengthened particular existing pathways, or unique experiences themselves might have emerged as one of the most significant pathways to homosexuality.

It is the nature of path analysis to eliminate those factors that do not apply to everyone in the sample in the simple attempt to find common factors. This is what Bell et al. did. But the net effect, as Van Wyk and Geist comment, is that “idiosyncratic and unique sexual and non-sexual experiences” as contributors to homosexuality are ruled out. Think of your own idiosyncratic and unique sexual and non-sexual experiences and judge whether or not you felt different as a result of those experiences.

The following people believe certain experiences, which they remember very clearly, were critical in the development of their later homosexual attraction. John mentions the “traumatic and unforgettable” day his father told him (at the age of five) and his sister that he and his mother were divorcing, and he would have to live for the rest of his life with his mother. Lorna said she realised, as a child, that her mother could not be trusted, but that her father “had it good” while her mother had to work all the time, so she didn’t want to be a woman. Roberta mentions a frequently repeated story of the death of her father’s first wife in childbirth that filled her with fear of being a woman. Then she was raped by her boyfriend. Steven talks about his father favouring an
older brother who was good at sports while he wasn’t. James mentions a rejection of his male genitalia at very young age, after he observed violent sexual abuse of his mother by his father. Jane recalls frequent sexual contact with her father who was not in other respects hostile to her. None of these falls easily into the variables in Figures 30 and 31. These days internet porn would probably have to be added to the list.

Van Wyk and Geist
This path analysis was not looking for causes of homosexuality. It was an attempt to eliminate the bias in Kinsey’s sample to see how it affected homosexual occurrence and distribution through the Kinsey classes. Kinsey himself was not concerned to find causes of homosexuality, so his questions were not geared that way. So Van Wyk and Geist’s contribution from the Kinsey sample to the debate on the development of homosexuality is incidental rather than deliberate.

Nevertheless, what did they find? They found that “intense sexual experiences and feelings of arousal and pleasure or discomfort associated with those experiences [were] the strongest precursors of sexual orientation.” All variables considered, they found higher levels of homosexuality among males in the Kinsey sample who reported

- poorer teenage relationships with their fathers,
- had more girl companions at age 10, fewer male companions at ages 10 and 16, avoided sports participation,
- learned of homosexuality by experience, learned to masturbate by being masturbated by a male,
- had intense pre-pubertal sexual contact with boys or men,
- had neither heterosexual contact nor petting to orgasm by age 18, found thought or sight of males, (but not females) arousing by age 18, had homosexual contact by age 15…and had higher first year homosexual behavior activity.

For women, they found more homosexuality among those who

- had few girl companions at age 10 and few male companions at 16, had learned to masturbate by being masturbated by a female,
- had intense pre-pubertal sexual contact with boys or men, found thought or sight of females, but not
males, arousing by age 18, had homosexual contact by age 18, and higher first-year homosexual behavior frequency.

All these factors together accounted for 36% of adult female homosexuality and 78% of adult male homosexuality (including the significant link from adolescence to adulthood), and the idea of tracking is mentioned again. These results are similar to those of Bell et al. but, again, many individual factors were mostly unaccounted for. The same pattern emerges: all these social factors together significantly contributed to homosexuality but each factor on its own was very small. So there are very many individual paths and stories, there are some common themes also found by Bell et al. but probably a lot of chance individual reactions to the same events. Kitzuger and Wilkinson in their survey of changes towards lesbianism remark that there were so many different psychological paths to exclusive SSA that it was impossible they were genetically controlled, a point rarely made. But their view is supported by the long list of SSA causes important to various people given in Chapter Ten, and gets support from the complexity of the paths in Figures 30 and 31.

Bem path analysis

One more, but minor, path analysis was done by Bem, father of the “Exotic makes Erotic” theory (see Chapter Three). It is very interesting because it incorporates genetic influences into a very condensed path analysis and compares them with social factors. Bell et al. did not have a means of making this comparison. Bem obtained the data from the Bailey et al. (2000) twin study on SSA, which included data on childhood gender non-conformity. Using the twin data from Bailey and others gives a measure of genetic influence.

Applying the mathematical method for path analysis he obtained Figure 32.

The diagram compares childhood gender non-conformity with genetics as contributors to later SSA. It finds genetic influence is not significant for either men or women, but that childhood gender non-conformity is modestly significant. In fact gender non-conformity is about 10 times stronger than direct genetic influences.

Bem finds more genetic influence on childhood gender non-conformity than direct genetic influence on adult SSA. The influence on adult homosexuality of childhood gender non-conformity is very
close to the findings of Bell et al., significant because the sample was completely different: Australian, not American.

So, not only does Bem confirm that Bell et al.’s strongest single factor (gender non-conformity) is important, he also finds that genetic influence is near zero in comparison.

**Summary**

The Bailey and Van Wyk and Geist path analyses have been used to argue that there is no social or familial basis to homosexuality. That conclusion is completely unjustified. Bell et al. chose to emphasise, even when their combined paths accounted for 76% of adult homosexuality, that the individual paths to adolescent homosexuality were not significant.

It would have been more accurate to add they could not find a single path to adolescent homosexuality which affected most people, but that individualistic paths were predominant. They identified paths that lend support to psychological theories of homosexual development: negative
relationships with the parent of the same sex, leading to lack of gender identification; gender non-conformity (sissiness in boys and tomboyism in girls); homosexual arousal in childhood and homosexual experience in adolescence. These explanations also have credibility among those who work with people wanting to change a homosexual orientation.

Van Wyk and Geist, although their raw material was not structured for a study of causality, nevertheless found environmental factors that overall accounted for 36% of female homosexuality and 78% of male homosexuality.

So this emphasises the importance of individuals and their experiences, which is the traditional case-study approach of clinical psychologists. Path analysis confirms that social causes as a whole are important, but the details must be filled in by narrated personal experience.

The two path analyses lend good support to the idea of a constellation of environmental factors behind homosexuality, rather than biological ones, with hints that existing paths might be strengthened if the right questions were asked, and respondents were able to volunteer reasons why they believed they became homosexual.

It is highly probable individual reactions to the same environmental factors are far more important than usually thought.

Bem’s path analysis shows that gender non-conformity is about 10 times stronger than genetic factors in the development of homosexuality.

References:


Can sexual orientation change?

One of the strongest arguments against homosexuality as an inborn, unalterable condition is change in sexual orientation. In this chapter we describe how the scientific literature shows that sexual orientation is not fixed but fluid. People move between homosexuality and heterosexuality to a surprising degree in both directions, but a far greater proportion of homosexuals become heterosexual than heterosexuals become homosexual—meaning heterosexuality is a more stable condition.

There are different types of change. A person may be attracted to both sexes, but slowly lose attraction for one sex and become exclusively attracted to the other. An increase of attraction for one sex may happen without becoming exclusive. Most interestingly, a person exclusively attracted to one sex may for the first time experience attraction to the other, which is usually a remarkable event.

Some of the change is therapeutically assisted, but in most cases it appears to be circumstantial. Life itself can bring along the factors that make the difference.

This chapter looks at change and its proponents and opponents.
The implications of change

Changes either to or from OSA (Opposite Sex Attraction) mean sexual orientation is not genetically dictated or permanent.

For some reason people find it far easier to believe a person could move from OSA to SSA than the reverse. So we will concentrate mostly on surveying SSA to OSA, though there is plenty of evidence for change in both directions. Change has been found so frequently that it has a technical name “fluidity”.

It is ironic that the group most insistent that change is not possible is the very group that has greatest fluidity, the transgender community, e.g., if a man attracted to women has a sex change operation it is a 50-50 toss-up whether he will be attracted to men or women afterwards.93,94

Spontaneous change homosexual to heterosexual

Bob is a former gay man whose father was sick most of his childhood and early teenage life. He grew up feeling homosexual attraction toward other men and had a sexual partner for two years as a teenager. Two years after the relationship ended, he suddenly realised his homosexual feelings had gone.

As I look back now I see that part of the reason was that I was working with my father [at that time] and having regular time with him for the first time in my life. I didn’t realize what was going on, but a need was being met in my life, that I didn’t know was there. I didn’t struggle with homosexuality at that point.

Bob believes that his homosexuality was a search for male affection and connection that had its origins in the lack of a childhood relationship with his father. He was much closer to his mother. When he began in his late teens to work and relate with his father for the first time, he believes he gained something from the relationship that led to a lessening of his desire for other men.

One homosexual man found that when he joined the Air Force, he began to notice women. The man was a self-identified homosexual—not seeking to change his orientation.
Being in a totally masculine environment I started to relate to men more spontaneously and feel better about my own masculinity. I felt I bridged a gap between me and the straight males...like being one of the guys and trusting each other. And as a result, all sorts of blocks broke down. I seemed to start to notice women...for the first time in my life I started having sex dreams with women in them. I was still mostly turned on by men, but suddenly, women too. It surprised the hell out of me.²

He became, in effect, bisexual. The change led the authors of the paper to remark on “the malleability and temporal unpredictability of sexuality and sexual identity.”

The sexology literature reports a huge number of examples of change of all degrees from homosexuality to or toward heterosexuality. These studies have been so numerous that West in 1977 took an entire chapter in his classic book, Homosexuality Re-examined, to review them, and commented: “Although some militant homosexuals find such claims improbable and unpalatable, authenticated accounts have been published of apparently exclusive and long-standing homosexuals unexpectedly changing their orientation.”³ West mentions one man who was exclusively homosexual for eight years, then became heterosexual.

Straight, a book written by a man with the pseudonym Aaron, in 1972, describes Aaron’s thorough immersion in the gay scene, his decision to leave it, and his arousal of feelings for women and subsequent marriage.⁴

Another well-known author in the field, Hatterer, who believes in sexual orientation change, said, “I’ve heard of hundreds of...men who went from a homosexual to a heterosexual adjustment on their own.”⁶

Among the Sambia, a Papua-New Guinean tribe in which homosexual sex was culturally prescribed for growing boys until marriageable age (when they were expected to be exclusively heterosexual), there was a significant change toward heterosexuality. Herdt,⁷ who has intensively researched the Sambia, graded individual males on the Kinsey scale for those two periods: before and after marriage. He found that the change from adolescent to married man in attitudes and behaviour equated to a move from Kinsey homosexual Classes 5 and 6 (predominantly to
exclusively homosexual) to Class 2 (predominantly heterosexual). Herdt believed the change was a real change in sexual orientation.

**Heterosexual to homosexual**

Exclusively heterosexual women can, in mid-life, develop lesbian feelings and behaviour. This is a well-known sociological feature of lesbianism.\(^3\)\(^5\) It often occurs during marriage or after marriage break-up, with no clinically observable hint of prior existence—not even lesbian fantasy, as reported by the following two therapists.

Nichols\(^5\) found among married bisexual women that “many appeared to make dramatic swings in Kinsey ratings of both behavior and fantasy over the course of the marriage” in ways that “cast doubt upon the widely held belief in the inflexibility of sexual orientation and attraction over a lifetime.”

Dixon\(^8\) surveyed fifty women who became bisexual after the age of thirty. They were exclusively heterosexual before, having had no earlier significant sexual fantasy about females, and quite heterosexually satisfied. They continued to enjoy promiscuous sexual relationships with both sexes.

Tanner\(^11\) reported that about half the lesbians she knew were heterosexual before midlife.

The work of Kinsey on male and female sexuality in the forties and fifties is probably classic in the field in its conclusions that sexual orientation is fluid and subject to spontaneous change. At an early stage in his research Kinsey (as cited by Kinsey researcher Pomeroy\(^9\)) discovered “more than eighty cases of [previously homosexual] men who had made a satisfactory heterosexual adjustment.” This was 2% of his sample. Small amounts of homosexual fantasy remained; but the typical description in those times was “adjustment”. Kinsey also found that most of the changes were as adults.

Commenting particularly on the work of Kinsey et al., Texas researcher Ross says, “Given these data...sexuality can thus be seen as a fluctuating variable rather than as a constant.”\(^10\)

A survey by the well known research team Bell, Weinberg and Hammersmith,\(^12\) published in 1981, also claimed that 2% of the heterosexual population said they had once been exclusively homosexual. Independently, Colorado researchers, Cameron et al.\(^13\) in 1985, reported
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an identical figure. Both these studies also put the percentage of homosexuality in the population at 4%. In other words nearly half the homosexual sample moved significantly towards heterosexuality. But change was occurring in both directions. About 2% of the heterosexual group became homosexual (Figure 33). More data are available from the comprehensive study by Laumann et al. (1994), 14 who reported that about half those males homosexually active as young adults were no longer active later. Granted, only one or two incidences of activity were recorded in each case, and questions were directed at activity rather than identity, but, as far as it goes, the survey supports the other studies. Rosario et al. (1996) 15,16 similarly reported in a longitudinal study that 57% of their gay/lesbian subjects remained exclusively gay/lesbian, but that the remainder had changed to varying degrees. Fox 17 reported various degrees of change among bisexual people (not undergoing therapy to change).

![Figure 33. Showing natural movement between sexual orientations](image)

The summary of these studies and an excellent rule of thumb is that about half of those with exclusive SSA were once bisexual or even heterosexual. This is stated explicitly in Sandfort (1997). 18 And about the same number have changed from being exclusively SSA to bisexual or
even exclusively heterosexual (though they obviously make up a much smaller fraction of heterosexuals).

California researcher Hart\textsuperscript{19} reported that roughly 1\% of a group of conservative Christian men spontaneously reported (in an anonymous questionnaire on sexual orientation, attitudes and behaviours, but not on change), that they had once been exclusively homosexual but now were happy and adjusted heterosexuals. Had they been specifically asked, the percentage may have been higher. Similarly in a large web survey organised among gay and lesbian youth by !OutProud!\textsuperscript{20} when asked what they thought about the possibility of sexual orientation change to heterosexual, 1\% actually volunteered they had made that change!

Studies showing varying degrees of change continue to be published in scientific journals. In a very well-known New Zealand longitudinal study\textsuperscript{21} 1000 children were followed from birth. From age 21-26, 1.9\% of men moved away from exclusive OSA, and 1\% moved to exclusive OSA. However among women, in an international record, a high 9.5\% moved away from exclusive OSA. A more usual 1.3\% moved to exclusive OSA. These and similar changes within the group led the researchers to say sexual orientation was almost certainly not caused by genetic factors.\textsuperscript{21} Similarly,\textsuperscript{22,23} various degrees of change over a few years were shown among young women in the USA. Some readers may already have heard of the LUG fad in women undergraduates at some USA universities—Lesbian Until Graduation—which shows the malleability of sexuality.

From the above we would have to conclude that homosexuality is much more fluid than heterosexuality as shown by the large proportion, 50\% (Figure 33) of homosexuals who move toward a heterosexual orientation, compared with the small proportion of heterosexuals who become homosexual.

Kinnish et al.\textsuperscript{24} surveyed in detail the type of changes that occurred, and they generally confirm the previous picture. Their results are shown in the next two diagrams, Figures 34 and 35, which assume the occurrence of SSA described in Chapter Two. (These figures are complex, see p198 for a simplified summary.) The sample was not random, and might mean that the degree of change was less than shown here, because a study on sexual orientation might attract those who had changed and were curious about why—in other words they might be over-represented in
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**Figure 34.** Movement of male adults between homosexuality and heterosexuality over a lifetime. Most movement is towards heterosexuality. Within each vertical column light grey labelled blocks indicate the previous orientation.

**Figure 35.** Movement of female adults between lesbianism and heterosexuality. Most movement is towards heterosexuality.
the group. The criteria was self-ascribed sexual orientation. The changes were over the whole lifetime, and seem to have included the unstable adolescent years. Figures do not add to 100% for the second diagram because of complications involving the “mixed” category, and insufficient detail in the paper.

**Figures 34 and 35** can be summed up like this:

- Most changes are towards exclusive heterosexuality
- Numbers of people changing towards exclusive OSA are greater than the current total numbers of bisexuals and exclusive SSA people combined. This surprising result supports the catchphrase circulating ten years ago: “Ex-gays outnumber actual gays.” About 3% of both men and women with exclusive OSA claim to have once been something else.
- Exclusive OSA is 17x as stable as exclusive SSA for men, and Exclusive OSA is 30x as stable as exclusive SSA for women. So women move about more in their sexual orientation than men.

The degree of change in bisexuals was exceptionally high—many more changed to some form of exclusivity than stayed bisexual.

No direct changes from exclusive SSA to OSA were reported in this sample. But it certainly confirms lots of change takes place spontaneously in the population.

Mock and Eibach\(^9^5\) found that over ages 40-50, 64% of exclusive lesbians change to something else, and 65% of bisexuals. Among SSA men, 9.5% changed, and 47.1% of bisexuals. No therapy was involved.

Katz-Wise and Hyde\(^9^3,^9^6\) found 63% of SSA women and 50% of SSA men, ages 18-26, had changed attraction at least once. About 20% had multiple changes, and the first change had been in late adolescence. No therapy was involved.

**Adolescent change**

Some of the most remarkable data on change is in adolescents. This is taken from a very large USA ADD-Health survey—Savin-Williams and Ream (2007).\(^2^5\)

We present the data in visual form to make them easier to follow. In the Figures below, black represents attraction to the opposite sex only, medium grey represents those who were attracted to both sexes, and
light grey those attracted to the same sex only. The diagram shows the changes in attraction in those three categories between ages 16 and 17. The survey used the term “romantic attraction” in its questions about attraction to one sex or the other, but we shall shorten it to “attraction.”

In the first diagram below (Figure 36), the bar on the left represents all males in the sample who were OSA at the age of 16. The three bars to the right show the percentage ending up in one of the three attraction classes a year later at age 17. The answers do not always add up to the height of the left-hand bar, because 15% of respondents who had romantic attraction in the first year, said they had none towards either sex in the second. Sometimes they did not answer the question at all.

**Figure 36:** Male Opposite sex attraction

**Figure 37:** Male same sex attraction
The results for OSA females in Figures 36 and 39 were almost identical to the male figures, so are not shown.

For those (many fewer) who had attractions only towards the same sex, we see something interesting in Figure 37. A very small percentage remain attracted exclusively to the same sex, but the greatest proportion by far has no longer any attraction to the same sex but experiences only attraction to the opposite sex. Same-sex attraction ceases in the course of a single year, or changes to an opposite sex attraction or perhaps these are transient attractions and there are no compatible individuals of the same sex available at age 17.

![Figure 38: Female same sex attraction](image)

Again, in Figure 38 we see considerable change from exclusive same-sex attraction to exclusive opposite-sex attraction.

There was no intervention to bring about any changes between ages 16 and 17. It seems maturation or chance was mainly responsible.

Figures 39-41 are data for ages 17-22.

This again confirms that exclusive opposite sex attraction persists, and for both sexes.

This again confirms that those who are attracted only to the same sex initially, in the usual course of events will mostly end exclusively attracted to the opposite sex. A surprisingly small percentage of those same-sex attracted in adolescence remain that way.
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**Figure 39:** Male opposite sex attraction, 17-22 years

**Figure 40:** Male same sex attraction, 17-22 years

**Figure 41:** Female same sex attraction, 17-22 years
The pattern for females is that only a very few stay exclusively same-sex attracted long term. Many become bisexual, i.e., they acquire an attraction to the opposite sex as well.

If an initial attraction was opposite sex, but a succeeding one was same-sex, depression increased.90

The conclusion of this is that there is a huge amount of change in attraction with time, certainly over five years, but even over as short a period as a year. These changes are profound, even compared with those for adults.

Are these (largely teenage) feelings real? Are they true SSA? It could easily be argued that whether OSA or SSA they not the mature form of these attractions. However, they are certainly real enough to trigger suicide when the person is rejected by their special friend, particularly if the attraction is SSA or bisexual.

From the above data for 16-17-year-olds, it is possible to estimate the degree of change from bi- or SSA, compared with the degree of change from OSA. Making the mathematical assumption that those with missing data will not affect the results, it is possible to calculate how much more likely it is that a homosexual orientation will become heterosexual than the reverse.

Men: SSA compared with OSA. 38x more likely
Bi/OSA. 57x as likely

Women: SSA compared with OSA. 28.9x more likely
Bi/OSA. 29.8x more likely

To err on the conservative side, Bisexual or Exclusive SSA is at least 25x as likely to change as OSA. (That is, 16-year-olds saying they have an SSA or Bi-attraction are 25 times more likely to change towards heterosexuality at the age of 17 than those with a heterosexual orientation are likely to change towards bisexuality or homosexuality.) This is comparable to, but even higher than, the figures derived earlier in this chapter from other papers.

Most teenagers will change from SSA. In fact, in the 16 to 17 year age group, 98% will move from homosexuality and bisexuality towards heterosexuality, perhaps experiencing some or exclusive opposite sex attraction for the first time.
Most teenagers thinking they are gay/lesbian/bi and will be for the rest of their life, will in fact probably be different the following year. It is therefore totally irresponsible, and flatly contradicted by the facts, to counsel affirmation of same-sex feelings in an adolescent on the grounds that the feelings are intrinsic, unchangeable, and the individual is therefore homosexual.

This is not a new finding. Tiffany Barnhouse, Professor of Psychiatry at Southern Methodist University emphatically remarked 20 years ago,

*It is impossible for me to state strongly enough that to present this [homosexual] model to young people, or to allow them—as often happens in the contemporary climate of open discussion—to imagine that their transitory adolescent experiments are truly indicative of a settled homosexual disposition, is not only evidence of psychiatric ignorance, but is specifically wicked as well.*

On the other hand 16-year-olds who claim they are OSA will overwhelmingly remain that way and this is a realistic assumption.

So whether adult or adolescent, a large degree of spontaneous change takes place. Rather than SSA being an unalterable condition, it is actually a good example of a changeable condition. So much change takes place that Savin-Williams and Ream questioned whether the idea of sexual orientation of teenagers had any meaning at all.

**Where are all the ex-gays?**

At this point the natural question arises—if there are so many “ex-gays” in the population, where are they? Very few readers will ever have met any that they know of. It is no wonder the GLB community is very sceptical about whether real change occurs, though the best estimate of the researchers involved is that it does, and spontaneously, without clinical intervention, as life goes on.

There are good reasons why this group has remained hidden.

- Most who have changed to OSA have some embarrassment about their previous life, and don’t like to talk about it
- Many believe the change to OSA has been real and permanent, and OSA is now their core identity. They don’t want to talk about their previous sexual orientation. Life has moved on.
• If they are now heterosexually involved, admission of previous SSA may jeopardise a present relationship

• If they publicly admit their previous SSA they will be subject to often hostile, public and relentless attacks by members of the gay community. Since many of these “ex-gays” are on the more timid end of the confidence scale, they keep their heads down. The late therapist, Dean Byrd, from his experiences with clients said

  “…do you know what happens when someone says he or she is ex-gay? Their lives and the lives of their families become a living hell. They are taunted by the activists, their families humiliated”

• Few of the changes are to 100% OSA and many people who have changed are perhaps uneasy about the few percent SSA that remains, since activists tend to argue in an absolutist fashion that even a remnant few percent SSA shows that real change does not happen.

• In contrast, a currently exclusive gay who was once OSA is likely to say his previous OSA was a superficial layer covering a core SSA identity, and will be more willing to discuss his previous identity—often for political reasons.

The degree of hostility towards those who have changed is extreme, and close to a total denial of free speech. Posters that appeared nationwide in the USA in the nineties showing a large group of people and a message saying: “Can gays change? We did”, infuriated members of the gay community. Some posters were torn down. A national advertising offensive was mounted in disparagement and denial. Most heterosexual people would find such a claim intriguing, but not insulting to the GLB community. But one gay spokesman at Penn State where this occurred called this “the most dangerous expression of heterosexism I have yet seen.”

Faculties in universities have sometimes intervened to order removal of such posters and have shut down organisations on campus backing their message. Why? This threatens SSA people to a degree which heterosexuals find hard to appreciate. Maverick gay activist Camille Paglia talked of
...fascist policing of public discourse in this country by nominal liberals who have become as unthinkingly wedded to dogma as any junior member of the Spanish Inquisition. Why should the fluidity of sexual orientation threaten any gay secure in his or her identity?

But, as we saw above, gay/lesbian orientation is much less secure than heterosexual orientation, so suggestions that change is possible naturally stir up much anxiety.

The best summary of this section would be that there is a large degree of spontaneous change, admitted by all researchers except the extremely ideologically motivated.

Assisted change

If considerable swings in sexual orientation toward OSA can happen without therapeutic intervention, perhaps a motivated person with therapeutic assistance might change further or faster. The first recorded instance of assisted change may be in the New Testament. In I Co 6:9ff, Paul, writing to the Corinthians, said about homosexuals (the word translated homosexuals is arsenokoitai in the Greek, meaning “male/coitus”)

...that is what some of you were. But you were washed, you were sanctified, you were justified in the name of the Lord Jesus Christ and by the Spirit of our God.

They changed, and it is reasonable to believe—given the emphasis in Christianity on inward attitude rather than merely outward behaviours—that the change was not merely behavioural.

Assisted change has been attempted since last century, using many techniques, including hypnosis, aversion therapy, behavioural therapy, psychoanalysis; some methods rather brutal, some a lot more successful than others. At an early stage in his research Kinsey “recommended a pattern of treatment to those who wished to change”9 In prescribing this course to those who wanted to take it, Kinsey always warned that “he had known it to be successful in many cases, but he had also seen it fail.” But it seems whatever the therapy used there were always some who changed toward heterosexuality as reported by the following therapists.
Reuben Fine, Director of the New York Centre for Psychoanalytic Training, remarked,

If patients are motivated to change, a considerable percentage of overt homosexuals (become) heterosexuals.\textsuperscript{27}

Bernard Berkowitz and Mildred Newman:

We’ve found that a homosexual who really wants to change has a very good chance of doing so.\textsuperscript{28}

Edmund Bergler concluded after analysis and consultations with 600 homosexuals over thirty years:

Homosexuality has an excellent prognosis in psychiatric/psychoanalytic treatment of one to two years duration…provided the patient really wishes to change. Cure denotes not bi-sexuality, but real and unfaked heterosexuality.\textsuperscript{29}

After twenty years of comparative study of people with SSA and OSA, Irving Bieber wrote:

Reversal [homosexual to heterosexual] estimates now range from 30% to an optimistic 50%.\textsuperscript{30}

Bieber followed some of his psychoanalytical clients for as long as ten years and found they had remained exclusively heterosexual.\textsuperscript{31}

Charles Socarides said:

There is…sufficient evidence that in a majority of cases homosexuality can be successfully treated by psychoanalysis.\textsuperscript{32}

Scientists Masters and Johnson, after work with 67 homosexuals and 14 lesbians who requested reversion therapy, reported a success rate of 71.6% after a follow-up of six years. Although they have been criticised for serious flaws in their post-therapy follow-up and assessment, it seems certain they produced many real and lasting reversions.\textsuperscript{33}

Psychologist, Gerard van den Aardweg, after twenty years research into treatment of homosexuality, stated,

Two thirds reached a stage where homosexual feelings were occasional impulses at most, or completely absent.\textsuperscript{34}
Psychiatrist William Wilson claimed a 55% success rate in treating homosexuals who were professing Christians.\textsuperscript{35}

According to Robert Kronemeyer, a clinical psychologist,

About 80% of homosexual men and women in syntonic therapy have been able to free themselves, and achieve a healthy and satisfying heterosexual adjustment.\textsuperscript{36}

Ex-gay support groups say hundreds of homosexuals have moved significantly toward a heterosexual orientation as a result of Christian commitment and the specialised support and services they offer.

UK sexuality researcher, West, summarising the mainstream material up to the seventies\textsuperscript{3} says that behavioural techniques appeared to have the best rate of success (never less than 30%). Although psychoanalysis claimed a great deal of success, the average rate seemed to be about 25% (but 50% of bissexuals achieved exclusive heterosexuality.)

One developmental research psychologist, Moberly, argued that the success rate of psychotherapy in homosexual reparative therapy has not been higher because of inadequate understanding of the causes of homosexuality, rates of success obviously reflecting the relevance of the treatment model. Moberly maintains that, until the eighties, psychotherapy was still viewing homosexuality as an opposite-sex problem rather than a difficulty in relating with the same sex. In her opinion, this explains the disillusionment of many homosexuals who unsuccessfully sought therapy in the past. It may be that the increasingly widespread adoption of Moberly’s treatment model in the last twenty years is reflected in the higher than average levels of change claimed by various more recent groups.

However, even where it is inadequately informed, psychotherapy produces change wherever it impinges on issues relevant to the causes of homosexuality. This means that even dealing with the depression, substance abuse or suicidality commonly accompanying SSA may make some difference to the SSA. As West comments in his review of the literature, “Every study ever performed on conversion from homosexual to heterosexual orientation has produced some successes.”\textsuperscript{3}
Reuben Fine similarly remarks,

all studies from Schrenk-Notzing [Victorian era] on have found positive effects virtually regardless of the kind of treatment used.”27

According to West, those most likely to respond to treatment are clients with a good level of motivation, a history of some heterosexual feelings, and who have entered the gay lifestyle later.

Brutal methods such as aversion therapy, e.g., electric shock) do not seem to have been used for many decades. Therapists these days strive to achieve professional standards of therapy as understood currently. Their rule of thumb is still that about one third of clients achieve rather dramatic change, one third achieve significant change and one third do not change. These rates are much higher than non-therapeutic spontaneous adult change. However we must reflect that in the current climate therapists are more likely to see the extreme cases. Given that, the reported clinical rates of change are quite good.

One well-documented change37 happened by accident, and involved medication. Two Florida medical professionals reported in 1993 that they treated a homosexual man for social phobia—he had extreme anxiety in any social setting. He had been exclusively homosexual in fantasy and practice since adolescence, but this was unconnected with his request for treatment; he was quite happy as a homo sexual. The drug Phenelzine helps many cases of social phobia and certainly did in his case. By the fourth week, he had become more outgoing, talkative, and comfortable in social situations. He spoke spontaneously in groups without blushing. But, curiously, he reported a positive, pleasurable experience of meeting and dating a woman.

During the next two months, he began dating females exclusively, reportedly enjoying heterosexual intercourse and having no sexual interest in males. He expressed a desire for a wife and family, and his sexual fantasies became entirely heterosexual…In retrospect [he] decided that the combination of his anxiety when approaching and meeting people, the teasing rejection by heterosexual males, and the comfortable acceptance by homosexual
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males who pursued and courted him, had helped convince him of his homosexuality.

So this report is of someone clearly exclusively homosexual whose behaviour, in three months, became exclusively heterosexual. This is an exceptionally fast change.

Homosexuality and the mental health professions

In 1973

In 1973, the American Psychiatric Association (APA) removed homosexuality as a disorder from its Diagnostic and Statistical Manual of Psychiatric Disorders (DSM-II), and redefined it as a condition only to be treated if the client was distressed—in which case he or she could be counselled to come to terms with the orientation. More recently, the APA Board recommended a resolution banning homosexual repara-
tive therapy. The move failed only because of aggressive lobbying by the resolution’s opponents.  

In view of the evidence that change is possible, what was going on?

The APA’s decision to declassify homosexuality as a disorder has been acknowledged by gay activists as one of their victories. The details are well documented, and the role of gay activists in the process is not really disputed. The APA, after months of harassment and intimidation by activists (who disrupted scientific research and conferences, forged credentials, and physically intimidated psychiatrists) made a “medical judgment” to remove homosexuality from the diagnostic manual by a vote of only 34% of its members.  

It was acknowledged at the time that the motive was mostly to prevent discrimination against people with SSA, and that research needed to be done to demonstrate that there was no abnormality associated with SSA. However the research was never done, in fact was then strongly discouraged as “discriminatory”. Although a survey conducted by the journal Medical Aspects of Human Sexuality four years later showed 69% of the 2500 psychiatrists who responded opposed the 1973 action the effect of the decision was to stop scientific research. In an age of minority rights and gay activism, reparative therapy became politically incorrect.
According to Nicolosi, one of the founders of NARTH (see below), the decision effectively silenced professional discussion of homosexuality as a disorder.\(^4\) Many mental health professionals are now simply rejecting of change, don’t know how to bring it about, lack the personal courage to stand against the tide, or are ideologically committed to the gay agenda.

\textit{In 2000}

In 2000, the APA went further. Its Commission on Psychotherapy by Psychiatrists issued a statement, approved by the entire APA leadership, that made the following recommendations:

1. APA affirms its 1973 position that homosexuality \textit{per se} is not a diagnosable mental disorder. Recent publicized efforts to repathologize homosexuality by claiming that it can be cured are often guided not by rigorous scientific or psychiatric research, but sometimes by religious and political forces opposed to full civil rights for gay men and lesbians. APA recommends that the APA respond quickly and appropriately as a scientific organization when claims that homosexuality is a curable illness are made by political or religious groups.

2. As a general principle, a therapist should not determine the goal of treatment either coercively or through subtle influence. Psychotherapeutic modalities to convert or “repair” homosexuality are based on developmental theories whose scientific validity is questionable. Furthermore, anecdotal reports of “cures” are counterbalanced by anecdotal claims of psychological harm. In the last four decades, “reparative” therapists have not produced any rigorous scientific research to substantiate their claims of cure. Until there is such research available, APA recommends that ethical practitioners refrain from attempts to change individuals’ sexual orientation, keeping in mind the medical dictum to first, do no harm...
This rigorous research was not demanded of other therapies. And such rigorous research would have been unethical. It would have demanded a treatment and non-treatment group, and the suicidality, substance abuse, depression and sexual abuse issues of those coming for help meant non-treatment was simply not an option.

In 2009

The American Psychological Association (also APA), came out with a long study in 2009 (APA Task Force, 2009). This included the following comments

…The APA concludes that there is insufficient evidence to support the use of psychological intervention to change sexual orientation

…The APA encourages mental health professionals to avoid misrepresenting the efficacy of sexual orientation change efforts by promoting or promising change in sexual orientation when providing assistance to individuals distressed by their own or other’s sexual orientation.

…advises parents, guardians, young people and their families to avoid sexual orientation change efforts that portray homosexuality as a developmental disorder.

The APA in its study was simply not convinced that change was possible, but readers of this book will be able to judge this for themselves. Change to varying degrees unquestionably happens. But this APA was demanding a level of proof that reparative therapy worked that it was not requiring for other therapies. Politics resolutely overrode the evidence.

The criticisms take little account of the fact that most who come for treatment are strongly motivated to change, and disillusioned by their experiences in the gay lifestyle. Reparative therapists would strongly agree that care must be taken not to harm clients, and they will rarely use the word “cure”, but they may insist that it is potentially lethal to remain in the gay lifestyle and worth trying to change. Nor do they make exaggerated claims about the outcomes of therapy.
Whether the trait is a mental illness or not, seems a very minor issue among them compared with the importance of helping by any valid means clients with a considerable burden of difficulties.

Many other professional associations have adopted similar stances to the APA’s, relying on their supposedly authoritative statements.

**Intimidation by professional bodies**

The National Association for Research and Therapy of Homosexuality (NARTH) was founded in 1992 by those psychiatrists who believed homosexuality was treatable. It sought to provide services to such clients and publish scientific evidence of change. After one year, about 50 professionals had joined, and by 2007 membership had swelled to over 1500, showing considerable dissatisfaction with the APA stance. It operated on a rather shoe-string budget and certainly did not receive monies from right-wing organisations in spite of rumours to that effect.

It continues to operate in the face of denigration and strong opposition from the professional organisations and gay activism, e.g., the publisher of a book by Joseph Nicolosi—a founder of NARTH—received dozens of angry phone calls and about 100 letters protesting at the publication of his book discussing reparative therapy for male homosexuals.

By 2005 the attitude of the professional organisations had become so politically driven, that a number of dissident senior members of the American Psychological Association in 2005 published a book in which they said,

> The APA has chosen ideology over science…censorship exists…even under the McCarthy era there was not the insidious sense of intellectual intimidation that currently exists under political correctness.

The authors attempting to recruit writers for chapters in their book, found “many…declined to be included, fearing loss of tenure or stature and citing previous ridicule and even vicious attacks”.

They said the attacks on reparative therapy “deny the reality of data demonstrating that psychotherapy can be effective in changing sexual preferences in patients who have a desire to do so.”

This is all an alarming indictment of a professional organisation. The APA is now complicit in attempts to silence and intimidate
researchers and practitioners of reparative therapy. These are tactics as bad as those used in the former Soviet bloc.

Reluctantly therefore we must conclude that no statement about homosexuality from the APA, and other professional organisations following suit, can be trusted without scrutiny in spite of their aura of authority.

By 2010 in a few instances, papers actually accepted for publication by various journals and approved on scientific grounds were subsequently rejected at higher editorial levels on political grounds only.

Burden of proof now on the APA

Because of the politically hostile atmosphere in these official bodies, there is now an enormous burden of proof upon them to establish they are putting forward an unbiased scientific case on this subject rather than making politically correct statements backed by misrepresented science. This level of hostility towards those who claim that change is possible, has almost no historical precedent in a professional organisation. However, modern research supporting the assertion that change is possible continues to be published in spite of the great difficulties.

Robert Spitzer, a prime mover in the 1973 decision to remove SSA from the Diagnostic Manuals as a mental illness, nearly thirty years later interviewed 200 people who claimed they had changed. He concluded that real and extensive change had occurred in many cases. This was an extreme and self-selected sample, but showed unequivocally that change, sometimes large, is possible for some motivated individuals.\(^45\) The study, published in 2003, attracted a large amount of criticism and abuse from the gay lobby, though any impartial observer would have agreed Spitzer had established beyond reasonable doubt that change does take place for some people. In view of the previously published literature, Spitzer's conclusion was no real surprise.

But after his study Spitzer received death threats so disturbing that he withdrew from making public comment about the subject because he said he had to protect his family.

A contrary study seeking those who had experienced some harm was then undertaken, and indeed showed harm to some people who had passed through therapy,\(^46\) the harm showing up in poorer self-image and suicidal thoughts. It also included accounts of people who
said they had been helped. This was followed by a doctoral project by Karten $^{47}$ who interviewed other people who claimed they had been helped and had changed. His results were very similar to Spitzer’s, and support the idea that change is possible. He described “considerable change in sexual identity.”

Jones and Yarhouse $^{48,97}$ found very substantial changes in 15% of their survey group, with many others changing significantly. This study was non-random like all the others, but a unique longitudinal study, and subsequent to the APA review. Many testified, “It felt like a complete change of orientation.” Although the authors could find traces of homosexuality in these people they described them as “heterosexual in a real sense”. About half had had professional therapy. This is clear evidence that change can take place, at least in those religiously motivated.

An important book by Janelle Hallman (2008)$^{49}$ describes various degrees of change reported among lesbians, and details of the process.

In 2012 Spitzer said he no longer wanted to assert that change was possible for a few motivated individuals because they might have been lying to him. The problem with accepting his statement is that if this sceptical and veteran researcher took the line that his respondents may have been lying to him then all survey results on sensitive matters cannot be trusted.$^{98}$

One study$^{103}$ found few changes and some harm amongst LDS (Mormon) people who had tried change. However the local specialist LDS ex-gay group had declined to participate in the survey because they had experienced past research abuses.

Santero, Whitehead and Ballesteros studied 125 men recruited to supply both positive and negative results of attempted sexual orientation change. Almost all underwent therapy, and half had a support group. Their overwhelming reason for the (secular) therapy was religious, but this included Roman Catholic, Protestant, Jewish, Mormon and other “non-denominational Christians”. The Mormon sample showed typical amounts of change. About one third of the 125 men changed a vast amount, one third a significant amount, and one third did not change. Many experienced OSA for the first time. Effects had lasted for three years, and 70% reported only beneficial results. There were also strong positive effects for depression, suicidality, self-esteem. The negative experiences were almost all slight. The effect sizes, positive and negative
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experiences were all comparable to those for other unrelated therapies, in other words quite unexceptional. All these results were supported with an orthodox statistical treatment, endorsed by experts.

Surveys like those of Santero et al, and Jones and Yarhouse show degrees of harm indistinguishable from standard psychotherapy of other conditions, i.e., 5-10%, and the harm is mostly slight.

Of course even one published case of documented change would be sufficient to disprove the assertion that change is impossible, but there are hundreds. Those changes are of varying degree, but the majority are satisfying to those involved—and that is one of the main ideals of psychotherapy.

The formation of “ex-gay” groups

An interesting development followed the American Psychiatric Association’s decision in 1973 and the companion move by the American Psychological Association. Looking for therapeutic help that was no longer easily available, men seeking to change their orientation began to set up support groups to help each other. Late in the seventies, they began to network and proliferate. Such groups are now active in the USA, Europe, South East Asia, and Australia. They came to be known as “ex-gay” groups—the largest being a confederation of groups called Exodus which disbanded itself in 2013 following strong internal dissen-sion. Most of its member groups are now regrouping under a new organisation, the Restored Hope Network, which continues to affirm that change is possible. Few of them like the word “ex-gay” however, and have actively sought alternatives, e.g., gender-affirming groups, none of which has generally caught on. We continue to use the term “ex-gay” here, but agree to some extent with the term’s detractors.

In 2016 a similar Jewish group was forcibly disbanded by a court relying only on the dubious statements of the APA.

Parallels with AA

There is an interesting parallel between the rise of ex-gay groups and that of Alcoholics Anonymous (AA). AA came on the scene at a time when the medical profession believed alcoholism was incurable, or at least didn’t know how to help. Bill Wilson, a recovered alcoholic and founder of AA, was invited to speak on May 24, 1949 at an alcoholism symposium
presented by the APA in Montreal. According to the record, a past president of the APA said to him later: “Outside of the few AAs in the room, and myself, I do not think a single one of my colleagues believed a word of your explanation.” When Bill Wilson expressed surprise because of the applause he had received, the man replied, Well, Mr Wilson, you AAs have a hundred thousand recoveries, and we in the psychiatric profession have only a few. They were applauding the results much more than the message.50

Alcoholics Anonymous came on the scene when the medical profession had no answers for the alcoholic; ex-gay groups surfaced at a time when the APAs distanced themselves from any attempts to change SSA.

AA had its detractors: people said the stories sounded spurious or they didn’t like the “God rackets” (AA’s Twelve Steps require a relationship with God—as He is understood). Bill Wilson’s right hand man relapsed, some members got drunk again, one at least committed suicide. The ex-gay movement has plenty of detractors too, and for similar reasons. Gay activists in particular like to quote the relapse of an ex-gay leader, Michael Bussee, in the ex-gay movement’s early history, and relish any failures.

AA today has wide credibility and an unofficial success rate of something like 25%. At some point in the future the general public may be as aware that gays can change their orientation as they are now aware that alcoholics can achieve permanent sobriety—the difference being that the reformed alcoholic believes he can never take another drink, but the former homosexual can form non-erotic relationships with other males and long-term sexual relationships with women.

Those who insist on 100% success rates in any field of therapy as proof of its effectiveness will never find them. AA believes that those who “work the program” will find their way out, and that many, for their own reasons, do not work the program. Success rates of about 25% are not uncommon in many programs offering recovery from problem behaviours with a strongly addictive component. Therapists talk of clients who find it easier to continue with the default solution than deal with underlying drives. Homosexuality appears to be little different. According to psychiatrist Cappon, psychologists can be confident that change occurs “at least as frequently in homosexual persons as in people with any other personality disorder.”51
Voluntary therapeutic groups have now been in existence for more than 30 years in spite of bitter opposition. They continue to exist because they have observed sufficient change in people to make it worthwhile to continue. Surveys have shown general client satisfaction even amongst those with minimal change and the number of disaffected clients has been small enough that ex-ex-gay groups are quite rare.

But numerous surveys now show that many people change their sexual orientation without targeted interventions. Those who come for therapy are the hardest cases, and not typical. So change is much easier on average than generally thought.

**Why does gay activism resist change?**

Gay activism usually comes up with any or all of the following arguments.

- The individuals concerned were never homosexual in the first place.
- The alleged change in orientation that has taken place is brief and illusory. (Given time the person will revert; the change is only the result of suppression of homosexual feelings which will resurface.)
- A person can change his or her identity but not the orientation. (You can stop acting homosexually, but you can’t stop being inwardly homosexual.)
- Those who say change is possible are “homophobic” (hating or fearful of homosexuality and homosexuals). That is, they are forcing homosexuals to become heterosexual because they don’t like homosexuality or homosexuals.
- Homosexuals who undergo this change are emotionally damaged in the process, become depressed, lose self-esteem, and become suicidal because they are doing violence to their true selves and “internalising” the “homophobia” that is forcing change on them.

Gay activism attempts to discredit any research that shows change is possible or anyone who claims to have changed. Why?
We believe this is why.

People with SSA who came to adulthood in the last several decades of the 20th century lived for a long time with the growing awareness of their homosexual orientation, well-aware of prevailing attitudes towards homosexuality, fearful of disclosure, and not knowing what to do about
it all. Many tried alone for years to change but failed. Some genuinely sought help from counsellors, ministers of religion, psychologists, or psychiatrists—often at considerable expense—but got nowhere. It’s not too surprising that many believe it’s impossible to change. “If it were possible, I would be heterosexual today,” some of them say. If they turned to religion, as many of them did, and found only censure, rejection, and no help to change, they will be cynical about the church unless it accepts them unconditionally. (Nearly 40% of gays say that because of their homosexuality they have become less religious than they were.)

Gays who find no way to change their orientation have few options, but one of them is to summon the considerable personal courage required to accept the label “homosexual” and “come out” to themselves, families, and others. Some gays organise themselves into lobbies and campaign for policy changes in all institutions. Naturally, when governments begin granting political protections, and homosexuality begins getting backing from the church, the judiciary, education, the medical and mental health professions and the media, and apparent “scientific” backing, change is not something a self-identified gay person needs to give much thought to—especially if there are rewarding patterns of sexual and emotional gratification to give up.

As one ex-gay, Frank Worthen, put it, after about 35 years out of homosexuality, “Sex (for males) has met their needs for closeness for so long that the prospect of giving it up is very threatening.” He goes on to say, “There is no-one in the lifestyle who cannot make the change—but many will be too fearful to seek it.”

Now, of course, the “right” to be gay and/or sexually active is enshrined in large parts of the West, so that any suggestion change might be a better option can almost be a criminal act, e.g., an Anglican bishop in north-west England wondering publicly whether being homosexual was an advisable lifestyle was visited by police and grilled. There is widespread and increasing official resistance to anything but acceptance and endorsement of the homosexual orientation.

In Australia a few years ago, a counsellor who enrolled and started a post-graduate university course on sexology was soon expelled, solely on the grounds that she was in favour of change therapy, and told “Don’t try to fight this. We have friends in high places.”
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It is much easier to argue that heterosexual intolerance and discrimination are the only reasons homosexuals want to change their orientation, than to believe change is possible or beneficial. Ross, for example, argues no homosexual’s request for help to change is voluntary in spite of surveys showing that a main reason for seeking therapy is genuine dissatisfaction with the gay life-style, and that pressure from others is a very minor factor.

In the seventies about half of lesbians and about 62% of gay men wanted to change their orientation at some time in their lives. According to Bell and Weinberg in 1978, about one in four lesbians and one in five males actually tried to do something about it, and almost half of them made two or more attempts.

There are no figures available for the period since, and almost certainly changed attitudes towards homosexuality have greatly lowered those figures. But people still seek help to change. They come for the following reasons.

Why do gays seek to change their sexual orientation?

Short-lived and unstable relationships

Some homosexuals find after a time that, homosexuality does not yield the promised satisfaction. Mr. Right doesn’t appear, or does, but sooner or later becomes Mr. Wrong. One gay man described the lifestyle as “the search for monogamy, from bed to bed.” Researcher Hooker found that almost all homosexuals have “an intense longing for relationships with stability, continuity, intimacy, love and affection but are unable to find it.” West comments that male relationships frequently break up “from internal dissension rather than outside pressure.” Sixty percent of male relationships last less than a year, and most lesbian relationships less than three years. Affairs of five years or more are exceptional. The real life of the overt male gay is “replete with jealousy, competitiveness, insecurity, malice, tantrums and hysterical mood shifts” says West. Pollak says homosexual relationships are “often bedevilled from the start by dramas, anguish and infidelities,” intense dependency, jealousy, and rage.

Sexual difficulties within homosexual relationships are about twice those within heterosexual relationships.
The median relationship length for the 50+ studies we have been able to find is 4.7 ± 2 years for both gays and lesbians. In contrast, heterosexual couples in the United States have almost a 50:50 chance of reaching their silver wedding anniversary (25 years). The contrast with heterosexual couples is so great that it is obvious there is much less stability. The chances of achieving a 25 year relationship are only a few per cent and this quest cannot ethically be recommended by counsellors.

The reason for this could lie in the work of Karten who found that 86% of those in his subject group who had sought change reported that being gay was not emotionally satisfying. This was the second most common reason for therapy.

Unfaithfulness
Even in spite of “intense longings for stability and continuity,” gay monogamous relationships are rarely faithful. “Monogamous” seems to imply some primary emotional commitment, while casual sex continues on the side. McWhirter and Mattison, a gay couple (psychiatrist and psychologist), attempted to disprove the notion that gay relationships did not last. In their book, The Male Couple, they reported the results. They finally located 156 male couples who had been together between one and 37 years, two thirds of whom had entered their relationships with expectations of faithfulness. Only seven had been able to maintain sexual fidelity, and, of those, none had been together more than five years. They could not find one couple who had been faithful beyond five years. Unfaithfulness is less tolerated in lesbian relationships than in male gay relationships. Although faithfulness is not promoted as a gay norm, unfaithfulness is the norm, and another reason why some seek change. Frank Worthen again, “Gay relationships may start out with idealistic dreams of life-long loving companionship but this usually degenerates into impersonal sex; a snare of using and being used.”

Compulsive behaviour
Terms like “compulsive,” “hyper-sexual,” and “addictive” are turning up more and more in studies of gay sexuality, sexual addiction being three times as common than among heterosexuals. Researchers Quadland and Shattls, remark:
For some a lack of choice is involved… They reported not feeling in control of their sexual behavior, reported having more sex than they wanted, and reported feeling victimized by their frequent sexual activity… the primary motivation and satisfaction appeared often not to be purely sexual… A pattern of sexual control emerged which seemed most closely related to that of overeating.63

Another researcher Pincu, comments that the main features of addictions are present in much gay sexual behaviour, and the behaviour is mood-altering.

The excitement is not unlike that of a child discovering something new or forbidden, is a strong motivating force in the continued search for gratification and temporary self-esteem… All the traditional defences of repression, rationalizing, minimizing, and intellectualizing are used by the compulsive individual to avoid admitting there is a problem and that his life is out of control.64

Homosexual promiscuity is well documented. Before AIDS almost half of white homosexual males had had at least 500 different partners, and 28% had had 1000 or more, mostly strangers.52 Homosexuals still have 3-4 times as many partners as heterosexuals,14,65 (when medians rather than means are compared) and between 13% and 50% of gays continue to practice high risk sex post-AIDS, evidence of an addictive drive. This is in spite of high levels of knowledge of HIV transmission routes, AIDS prevention counselling, positive HIV status, special safe-sex campaigns, and deaths of friends through AIDS.66,67 A significant amount of homosexual behaviour is out of control. NARTH therapists mention a figure of 30% sexual addiction among their clients.68

Sexual behaviour that is out of control does not increase self-respect; ultimately it leads to a sense of helplessness and depression.69 Ex-gay groups say men seeking help often say they feel used. This is not to say that all homosexuals are promiscuous. Some are celibate, but they appear to constitute only a small minority of self-identified homosexuals. According to a long-term study of homosexual men in England and Wales published in 1992, only 6% had had no sex in the last year.70 West noted an “obsessive preoccupation with sexual topics whenever
gay circles foregather” and “often a dislike of being tied down, leading to many partners”.

**Loneliness with increasing age**

Male homosexuals become isolated with age. Kinsey Institute sociologists Gagnon and Simon comment, “serious feelings of depression or loneliness are often attendant on…the middle to late thirties.”

A future with no family life, children, or grandchildren can mean a bleak future for the non-married homosexual who becomes less attractive as he ages and feels less accepted by either the homosexual or the heterosexual community. Modern gays seek to deal with that by pressing for gay marriages and families.

**Other problems**

Those who come for help are often burdened with depression, suicidal-ity, substance abuse (with a mean of three such problems, according to the Santero study) and are seeking answers for those as well.

**Early death**

A less common motive for therapy (41%) is fear of death. After AIDS emerged there was an initial concentration on safe-sex precautions, but safe-sex campaigns since 2005 are increasing being ignored. Even anti-HIV drugs are not preventing deaths as they might. The risk of anal cancer in AIDS patients is 20 times higher than in the general population and epidemiologists consider 20 times an astonishingly high factor. Even in HIV+ (but non-AIDS) patients the paper reported percentages about three times higher. The inevitable medical truth is also that unprotected promiscuity whether associated with OSA or SSA is the ideal milieu for infectious disease, some of which will be life-shortening.

Rotello points out the hard mathematical fact that a community becoming HIV+ at current typical rates of 1-2% per year will lead to 50% infection in the long-term, which would mostly occur in cities in suburbs in which gay people predominate.

**Conscience**

The gay lifestyle is not unrelieved misery. Some gays and lesbians don’t leave it for any of the above reasons. They have plenty of good times and
would be happy to stay where they are if it weren’t for what they would probably call their conscience—a persistent sense that what they’re doing is not what they’re meant to be doing. The root of this is often religious conviction and they would be reluctant to describe this as “internalised homophobia,” an increasingly common phrase. Conscience is the most commonly cited reason for seeking therapy.

**Ignorance of the possibility of change**

Ex-gays who have spent years in the gay scene say many gays would get out of the scene if only they knew how. Given the abundant statistical evidence of change, the attempt by gay activists to discredit the change process is a culpable form of discrimination against a significant group of homosexuals who want to change. Fine remarks,

> The misinformation…that homosexuality is untreatable by psychotherapy does incalculable harm to thousands of men and women.\(^{27}\)

Bergler insists,

> The homosexual’s real enemy is his ignorance of the possibility that he can be helped.”\(^{29}\)

Masters and Johnson comment,

> No longer should the qualified psychotherapist avoid the responsibility of either accepting the homosexual client in treatment or…referring him or her to an acceptable treatment source.\(^{33}\)

Tiffany Barnhouse, Professor of Psychiatry at Southern Methodist University stated,

> The frequent claim by ‘gay’ activists that it is impossible for homosexuals to change their orientation is categorically untrue. Such a claim accuses scores of conscientious, responsible psychiatrists and psychologists of falsifying their data.\(^{75}\)
The change process

Ex-gay groups, and those therapists working with homosexuals seeking to change, identify several major issues often needing attention. There is frequent co-occurring suicidality, sexual abuse, depression and substance abuse. Specifically associated with homosexuality there are often severe breaches in the relationship with the parent of the same-sex and refusal to role-model, rejection by same-sex peer groups, usually eroticisation of unmet needs for affection, confusion of sex with love, a mind-habit of same-sex erotic fantasy, and frequently an addictive cycle of sexual gratification. In females the addictive cycle is less sexual than emotional.

The groups say the problem is deep-seated (at least in those who come for help) and to beat it takes commitment, patience, honest self-examination, and a lot of support. Ex-gays tend to say two things are essential: a complete break with the gay lifestyle (leaving the current relationship, and the gay milieu, moving out of the area if necessary), and a strong heterosexual support network to replace the gay support structure.

Ex-gay groups belong to a family of support groups dealing with problem behaviours. Most of these make an appeal to a higher power. In ex-gay groups, the appeal is specifically to God, who is represented as loving and understanding—unlike many gay perceptions of God. They work to raise levels of self-esteem. Groups say that accountability, constant support, help in dealing with the addictive cycle (identifying and avoiding triggers), and forming non-defensive, non-erotic (or mentoring) friendships with people of the same-sex, and inclusion in functional families, lead to gradual but steady shifts in sexual orientation toward heterosexuality and the development of heterosexual attraction. Members are encouraged to forgive parents and reconcile. Lesbians in particular receive help for high levels (85 to 90)% of male sexual abuse.

Surveys with varying degrees of formality—particularly now one very careful 6-7 year study—have shown (for males) that the factors most helpful in the process are affirmation by other heterosexual males, male group activity, e.g., for weekends, and mentoring (if a mentor can be found). These factors were more important than therapy itself, or support groups, though these received some plaudits.

Ex-gay groups are often unwilling to specify a time frame for the transition process, but change appears to be slow and steady, with
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relapses. Some therapists and ex-gay groups say compulsive drives can fall to controllable levels in eighteen months to two years and steadily diminish thereafter. It appears that after he or she is no longer acting out compulsively, the “ex-gay” is not too different from people seeking help for heterosexual problem behaviours.

Courses run by ex-gay groups often examine and help group members resolve “underlying” attitudes that they say prop up the homosexual condition, like resentment, unforgiveness, fear, anger, insecurity, rejection, envy, isolation, pride, anti-authority attitudes, defensive ways of relating, low self-esteem, manipulation, and the need to be in control. Ex-gay groups claim that those who have worked through the issues are genuinely no longer homosexual on the inside—not merely suppressed homosexuals who appear heterosexual on the outside. (A fuller discussion of the change process may be found elsewhere.33) Many ex-gays go on to marry, but early marriage with an opposite sex partner is usually a disastrous form of therapy and is discouraged until much later.

Gay activists have attacked the change process, saying it is injurious to self-esteem and can make gays suicidal and depressed46. However, a survey by Mesmer found the opposite. He surveyed 100 people who had sought help toward a change of sexual orientation. He found that 88% felt “more able to have friendly relationships” and felt “more self-respect.” Ninety-seven percent of men felt more masculine, and 77% of women more feminine. Seventeen of the respondents had married, 55% reported “exclusively heterosexual interest,” and 47% some homosexual interest that they “rarely felt compelled to act out.” Thirteen per cent still had some homosexual behaviour. Ninety four percent felt closer to God.76 A NARTH survey also found an improvement in psychological well-being and inter-personal relationships as a result of reparative therapy as did the careful study of Jones and Yarhouse,48 and the study of Santero et al.

Ex-gay groups argue that homosexuality itself is a symptom of poor self-esteem, saying that a boy or girl who has not bonded with a same-sex parent, has felt different from or excluded by peers, and has often been sexually abused, will not have high levels of self-esteem. Sexual behaviour which is out of control also leads to depression. Bisexual women cut themselves 20x more often than heterosexuals100 and GLB people attempt suicide roughly three times more often
than heterosexuals\textsuperscript{52} a statistic that has often been blamed on societal attitudes. But, two of the most important reasons for gay suicide attempts, when they are directly related to homosexuality, are over the break-up of a relationship—romantic, friendly or familial—and inability to accept one's sexual orientation, not discrimination by others.\textsuperscript{52,83,84,89} The literature also shows the rate of attempted suicides amongst SSA in various countries is not directly related to discrimination and other attitudes in society,\textsuperscript{87} though they are probably an indirect factor.\textsuperscript{85} Self-rated health and well-being are similarly not directly related to perceived level of GLB acceptance in European countries.\textsuperscript{86} Studies which have tried to demonstrate the direct influence of societal oppression have so far not succeeded, rather they have identified psychological coping mechanisms (emotion-based, rather than problem-solving) as being the major factor.\textsuperscript{78-82,88}

It is unreasonable, therefore, to claim, as gay activism does, that those who try to help motivated homosexuals change are homophobic. To be consistent, they would have to argue that Alcoholics Anonymous hates alcoholics.

Although gay activists say that those who claim to have changed were obviously never homosexual in the first place, hundreds of homosexuals making the transition can talk of years of homosexual attraction and sexual activity, or of lovers, live-in relationships, promiscuity and political activism. One former gay man, David Kyle Foster, often answers those who doubt he was ever really homosexual in the first place, “Would making love to over 1000 men count?”

Although gays want proof that no homosexual thought will ever occur again, ex-gay groups say such a demand is unrealistic—like saying a former alcoholic will never again have a momentary urge to reach for the bottle. Such an urge can be seen for what it is: some old trigger which has now lost its power. Groups report that homosexual urges gradually become controllable and continue to diminish steadily, while heterosexual interest begins to develop. Many ex-gays marry happily. One former homosexual man, a veteran in the ex-gay movement, Alan Medinger, said, “some little thing might zing ‘em periodically. But it’s really nothing more than a nuisance.” Ex-gays in treatment are encouraged to identify what they are really seeking when a homosexual impulse occurs,
and to set about getting it non-erotically. In males, it is often a need to be affirmed as a male by another male, he said.

**How much can people change?**

We have noted van den Aardweg’s statement that in two thirds of cases in his therapeutic experience, homosexual impulses became only occasional or completely absent. Ex-gay groups also speak of such people, even though their help is less professional. Large change is possible for some individuals.

What does the fact that there are a variety of outcomes mean? It certainly means that change is worth trying if someone is deeply dissatisfied with their current state. The fact that some people change to a remarkable extent is valuable because it shows what may be possible for many more people in future as research continues.

Does the fact that some people do not change, negate the change in those who do? Of course not. No-one would not look at failures of cancer therapy and say no cancer therapy should be allowed. Long-term remission from cancer occurs and inspires greater efforts to overcome it. In Spitzer’s, Karten’s and Santero’s groups of subjects there was a lot of religiosity (mainly Judeo-Christian). As in AA, those who had changed, believed they had been helped by a Higher Power. However different degrees of religiosity had little effect; within his group, Karten did not find a clear correlation between change of feelings and degree of religiosity. The conclusion from other studies is that change occurs more often with some religiosity rather than none. A general conclusion from the Spitzer and Karten, Jones/Yarhouse, and Santero et al. surveys is that change from exclusive homosexuality to exclusive heterosexuality is rarer, but that there is general satisfaction with whatever change occurred.

There are no sound statistics on the extent to which such people ultimately form satisfying opposite sex relationships; anecdotal evidence suggests that quite a proportion of those who change become reasonably satisfied singles. Many in our modern society, view sexual gratification as a human right and object that heterosexual celibacy is insufficient evidence of change. But the person who opts for easy sexual gratification can have little to say to someone who has achieved a personally satisfactory outcome though some years of deep and difficult self-examination.
Summary
There is abundant documentation that people with SSA do move toward a heterosexual orientation, often with therapeutic assistance, but mostly without it. Some achieve great change, some less, but it is clear that sexual orientation is fluid, not fixed, so that it is impossible to argue it is genetically pre-determined. There is a good possibility that various degrees of change will happen with the right support, including therapy of various kinds. The problem in the present social climate may be finding such support.

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Summary

Those researchers who know most about genes and SSA⁴⁵ say, “Your genes did not make you do it”. Let’s review the evidence bearing in mind that many of the following arguments apply to all human behaviours. These summary statements are deeply explored in each chapter.

Change
The huge amount of change in sexual orientation is one of the clearest evidences that homosexuality is not hard-wired by genes or anything in the biological environment.

large studies now show that:

For adults:
About half of those with exclusive SSA move towards heterosexuality over a lifetime. Put another way, 3% of the practising heterosexual population (both men and women) claim to have once been either bisexual or homosexual.

- These changes are not therapeutically induced, but happen “naturally” in life, some very quickly.
- Most changes in sexual orientation are towards exclusive heterosexuality.
- Numbers of people who have changed towards exclusive OSA are greater than current numbers of bisexuals and exclusive SSA people combined. In other words, “Ex-gays outnumber actual gays.”

⁴⁵ SSA is same-sex attraction; OSA opposite-sex attraction.
Exclusive OSA is 17 times as stable as exclusive SSA for men, and 30 times as stable as exclusive SSA for women. (Women move about more in their sexual orientation than men.)

For adolescents:

- Most teenagers will change from SSA. In fact, in the 16 to 17 year age group, 98% will move from homosexuality and bisexuality towards heterosexuality.
- 16-year-olds saying they are SSA or Bi-attracted are 25 times more likely to say they are opposite sex attracted at the age of 17 than those with a heterosexual orientation are likely to identify themselves as bi-sexual or homosexual.
- 16-year-olds who claim they are opposite sex attracted will overwhelmingly remain that way.

Genes

Twin Studies:

- These very complex comparisons of identical twins and non-identical twins definitively rule out genetic determinism. Identical twins with identical genes are about 11-14% concordant for SSA. If homosexuality were “genetic,” identical co-twins of homosexual men and women would also be homosexual 100% of the time. In classic twin studies the genetic fraction is less than 22% for men and 37% for women, and may be as low as 10%. Twin studies continue to find steadily lower genetic fractions for homosexuality as methodology improves and samples become larger. Everyone has at least a 10% genetic influence in his or her thinking and behaviour—simply because without genes there can be no human activity or behaviour of any kind. Twin studies show that individualistic reactions to chance events (in which one identical twin reacts differently from the other) are by far the strongest contributors to homosexuality. In other words randomness is a strong factor.
A scan of the whole genome has not found any genes unequivocally and directly linked to homosexuality, unlike the case for schizophrenia (which has still identified only 4 genes directly linked to schizophrenia, and that to only 3% of schizophrenia.)

From an understanding of gene structure and function there are no plausible means by which genes could dictate SSA (or other behaviours) in a person.

So far, genetically dictated behaviours of the “one-gene-one-trait” variety have been found only in very simple organisms. Generally, geneticists agree that many genes (from at least five or six to many hundreds) contribute to any particular human behaviour.

Any genetic influence is believed to be weak and indirect.

No genetically determined human behaviour has yet been found. The most closely genetically-related behaviour yet discovered (mono-amine oxidase deficiency leading to aggression) has shown itself remarkably responsive to counselling.

A genetically dominated SSA caused by a cluster of genes could not suddenly appear and disappear in families, as it does. It would persist through every generation for many generations. It is genetically implausible that many “heterosexual” genes could switch off at the same time.

The human race shares most of its genes—something between 99.7% and 99.9%. That means all ethnic groups will have most of them. This has three implications.

- If homosexuality is genetically dictated, homosexual practices will be identical or very similar in all cultures. But the enormous range and diversity of homosexual practice and customs in different cultures (and within cultures), argues against this.
- There would be a similar percentage of homosexuality in all cultures. But homosexuality has been unknown in some cultures and mandatory in others.
Changes in homosexual practice and behaviour in different cultures would take place very slowly, over many centuries. But this is not what history shows. (The decline of whole models of homosexuality (the Greek, over a couple of centuries, and the Melanesian, within a century); the relatively sudden (in genetic terms) emergence of the present Western model over a couple of centuries; and abrupt changes of practice within an ethnic group, even over a single generation, are not consistent with anything genetic. Even less so the swiftly changing sexual practices within the current Western model.)

The occurrence of SSA in the population is too frequent to be caused by a faulty pre-natal developmental process, so it is not innate in that sense either. This includes epigenetic processes.

If SSA were genetically determined, and led to same-sex contact only it would have bred itself out of the population in only several generations. It would not exist today.

The age of first same-sex attraction could be about 10% genetically influenced and opposite sex attraction about 15%. (Remember, everyone has a 10% genetic input into anything they think or do.)

First attractions (both SSA and OSA) occur on average at age 10 and are rarely “earliest memories,” meaning attraction is mostly socially induced.

Hormones

There have been many studies, none of which has shown any convincing relationship between homosexuality and exposure to pre-natal hormones, although several have shown very weak links between pre-natal hormone exposure and infant play.

The idea that homosexuality results from immune attack on male brain characteristics by the mother is poorly supported. In that case male testes and genitalia (having the largest cluster of male-specific targets) should be attacked but are not.
Brain structure

- Numerous older studies of brain micro-structures, e.g., in the nineties, have failed to come up with replicable differences between adult heterosexual and homosexual brains.
- Modern studies show male and female brains at birth are not structurally different, making the likelihood of a specifically “homosexual” brain remote indeed. The main consistently replicable difference, from about age two or three, is their size.
- The environment has effects on the brain from birth to puberty and beyond.
- Sexual dimorphism of the brain has been linked with prenatal testosterone, but this is a weak effect, and only 16-27% of the occurrence is explained.
- Neuroscientists are finding that the brain is extraordinarily plastic. The scientific consensus now is that even as an adult, we are what we are making our brains even though we may not be aware of the constant ongoing process. These changes in microstructure are visible in brain scans, within months.
- If differences are found between homosexual and heterosexual brains they could easily be the result of years of conditioning (repeated thinking patterns and behaviour).

Social, sociological

Intersexes

- About 90% of Western “intersex” children (those born with ambiguous genitalia) choose to remain in their gender of upbringing when puberty reveals their true genetic gender and surgical interventions are offered. Often, this choice is made in the face of very contrary physical and hormonal characteristics. It argues for predominant environmental influences on the formation of gender orientation and behaviour.
Other

- Divorce of parents doubles the risk of later homosexuality in children
- The stages of psycho-social development toward adult heterosexuality are well defined and accepted by developmental psychologists, and are so obviously learned that heterosexuality is clearly not genetically mandated. In surveys of adult homosexuals many show differences in several of these developmental stages—suggesting that homosexuality is cultural and environmental rather than genetic.
- There is a much higher occurrence of homosexuality among those who have been raised in large cities, rather than in rural areas, arguing that the environment is much more powerful than genes in the development of homosexuality.
- A scientific/sociological tool, Path Analysis, has been argued to show that there is no social or familial basis to homosexuality, but rather a biological one. However, social and family paths leading to homosexuality were collectively significant, though individual paths were not. In contrast genetic paths were collectively insignificant.

Instincts

- Our instincts, such as self-preservation, hunger, and reproduction, are among the most deeply embedded and strongest impulses we have, but these are able to be controlled and even adapted. If we want to argue homosexuality is also a deeply ingrained instinct, we can also argue it should be malleable and responsive to training.

Genetic content of homosexuality is minimal
Geneticists, anthropologists, developmental psychologists, sociologists, endocrinologists, neuroscientists, medical researchers into gender, and twin study researchers are in broad agreement about the role of genetics in homosexuality. Genes don’t make you do it. There is no genetic
determinism, and genetic influence at most is minor. Individualistic reactions to random factors are very important.

Those who say homosexuality is genetically influenced are correct, but only to about this degree:

If a girl becomes pregnant at age fifteen, we could argue that she is genetically predisposed to. We could say that in her culture, her genes gave her the kind of face and figure that send male hormones into orbit and bring her under a level of pressure that she is unable to resist. But that’s about the strength of the genetic influence. There are a huge number of environmental factors that could also have brought the pregnancy about, from cancellation of the basketball game she was going to watch with a girlfriend, permission to use Dad’s car, her boyfriend’s company, the movie they had just viewed together, and failure to use a contraceptive, to big environmental factors like personal values systems, peer group pressure, and an emotionally distant father.

Is this consensus likely to change? Might some major biological link be discovered which could change everything?

For most of these scientific disciplines, the findings have been clearly established from facts that will not change. But what of future studies of brain micro-structure, or detailed analysis of genetic composition and function? Will they reveal links between brain structure and human behaviours, or behaviours and genetic sequences?

Of course they will. Papers will continue to be published. But we can safely conclude that even authors wanting to find such links will almost always include the standard scientific caveats that the influence is minor, and that the environment is important. What we can reasonably say about future research is that it will enter new fields and come up with new links, but none of them will be definitive.

This is proved once and for all by studies of identical twins. They have identical genes, but if one is homosexual the identical brother or sister usually isn’t. There is only an 11-14% chance he/ she is homosexual. This includes all the influences we know about now and those
we know nothing about and have yet to discover. All of them, added
together, only have a rather weak effect.

The first edition of this book in 1999 floated the trial balloon that
the genetic content of SSA will ultimately turn out to be 10%. That is
quite imprecise, and could be in the range 0-20%. But even if the final
result is 20% this is a weak influence. At the time of writing, 2020, our
assertion still holds, and is stronger than in 1999.

The following factors (see relevant chapters) support a 10% influ-
ence on SSA: age of first attraction, urbanisation, intersex choices, OSA
(genetic influence including prenatal testosterone measurements),
degree of brain formation after birth, summed gene effects and twin
studies.

Homosexuality, as a genetic inevitability, has probably been gay
activism’s most effective PR initiative in the campaign for equal rights
and special protections. Although it is no longer politically correct or
fashionable in many circles to say that homosexuals can change, it is
scientifically accurate to say so. We are not speaking only of behavioural
changes but changes in attraction.

The fact is that nothing makes us do anything—neither our genes
nor our environment. Liszt may have had long fingers but it didn’t make
him a composer for the pianoforte.

What is the cause of SSA?

There is no one cause. No single genetic, hormonal, social, or envi-
ronmental factor is predominant. There are similar themes: childhood
gender non-conformity, sexual abuse, peer and family dynamics, sexual
history, but the mix varies greatly with individuals, making distinctive
individual responses the single overriding factor. Two children from
the same family and social environment can interpret incidents very
differently. So random reaction, if it structures itself into self-image,
can become a significant contributor to homosexuality—as twin stud-
ies show. The overriding outcome is a homo-emotional focus on certain
people of the same sex—emanating from a necessary but (often) barely
conscious drive to make up love and gender identity deficits. At puberty,
this can get confused with genital sex, leading to a pattern of sexual
encounters resulting in eventual self-identification as “homosexual.”
It can be changed. Where responses are deeply entrenches it takes courage, commitment, perseverance, effort, self-understanding and support from others. The shallower the involvement, the easier the passage out; but many have achieved it.

Is it all worth it? Is it worthwhile to gain the freedom where nothing rules over you and to find others who have shared the same struggle? Is it satisfying to join a group of people who are the real heroes? Is it worthwhile to come out of what did not involve conscious choice, by an adult conscious choice? Is it worthwhile to do what others say is impossible? If a change like this is possible, what else may be possible?

DNA is a ladder of nitrogenous bases and sugars that is a recipe for proteins, not sexual preferences. But it is also a ladder of destiny, a Jacob’s ladder, and we can decide whether angels or demons will walk up and down it. We can decide to capitulate to the “genetic argument.” Do your genes make you do it? You choose.

I saw, struggling in a stagnant pool, a bee which had somehow fallen in. It flapped its wings futilely and tried to dog-paddle, but made no progress. It seemed to be drowning. All around the bee were little creatures called water-fleas who hopped round, trouble-free on the surface of the water. They didn’t seem interested in the bee at all.

I took the bee out of the pool using a dead leaf from a tree, and set it down nearby on the slate surround. The bee staggered off the leaf, drunkenly wandering in its new freedom, headed straight back to the pool and fell in again.

I lifted it out once more, and the bee staggered round rather aimlessly and seemed quite lost. I transferred it further away onto some grass. It tried to use its wings, but it looked to me as though they might be torn, and it might never fly again. It staggered from blade to blade, under some and over some in the three dimensional maze of the herbage. It even hopped from one blade to another, perhaps pathetically imagining it was flying.

Then—suddenly—after I had practically given up, it flew! It wove a surprisingly straight course through the airy
dimensions and was out of sight in seconds. I never saw it again.

This I know: that bee reached heights the water-fleas couldn’t even dream of and so can you.
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