

## Can genes create sexual preferences?

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 Rembrandt's 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.<sup>21</sup>

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). (Not On-line Sexual Activity!) 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

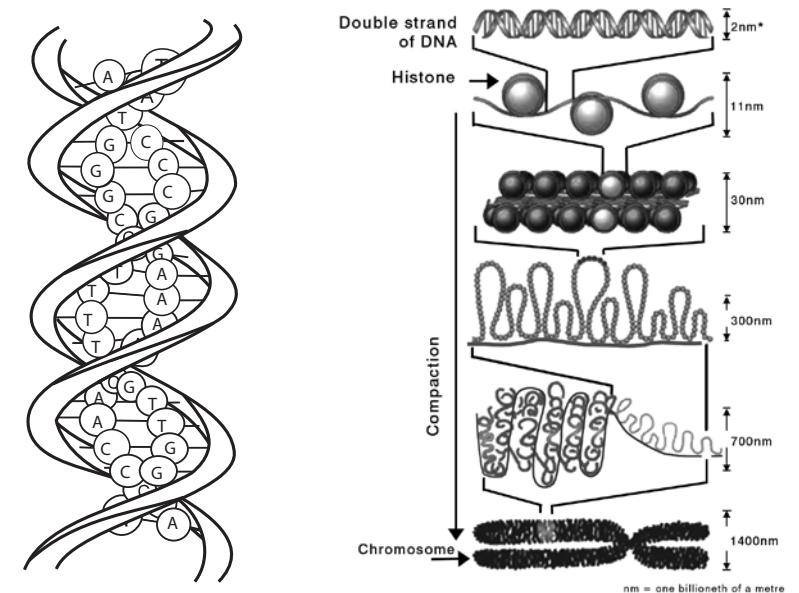
But first, let's visit the nucleus of a single human cell for a moment and look at some of the fundamentals of genetics.

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 highly folded strand of deoxyribonucleic acid (DNA) made up of an extraordinary twisted ladder 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.<sup>1</sup> 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 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 floodwaters.

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.<sup>15</sup> 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 the entire genes in one small bacterium in only four



**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 on p27.) The highly folded DNA on the right occurs only during cell division.

hours, and the minimum number of genes for a viable scientifically-designed cell was estimated to be 430. By 2010 it was even possible to make a simple synthetic DNA capable of making a bacterial cell function and reproduce. One paper published 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.

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 has been some puzzlement about the function of these “waste” stretches of DNA but by 2006 geneticists strongly suspected 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 makes us desperate to sleep. We hope this account won’t!) The arrangement 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 transfer of hereditary information from one generation of cells to the next and from one generation of humans to the next. The entire chromosome is made up 64 different 3-letter sequences of code that 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 biochemistry 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 biochemical. The DNA contains genetic coding that spells out the instructions mostly for making 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 those from the immediately adjacent 3 letters 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 fertilized 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 co-operation 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.

### 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

\* This was calculated from the energy used by a typical cell compared with the energy of a typical chemical bond.

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.”<sup>2</sup> 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,<sup>3</sup> 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. A recent paper found 567 interactions between 268 of the genes in yeast,<sup>18</sup> 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 cell biology contains 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). Plomin, a well known English expert in behavioural genetics, says more than 4000 gene effects due to mutation are known 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 very fast.<sup>5</sup> 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 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,<sup>7</sup> 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 and also creates borderline mental retardation. 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.<sup>8</sup> 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:

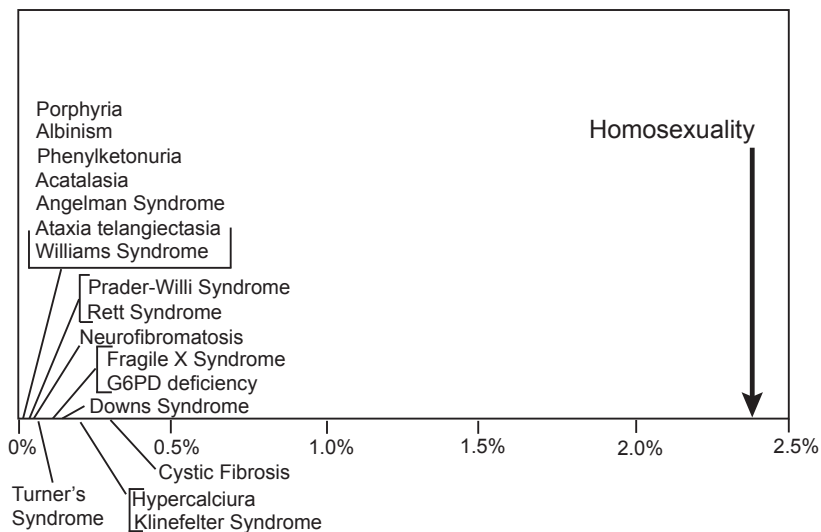
One of the most closely genetically-linked human behaviours known to science is only weakly influenced.

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.<sup>6</sup>

### *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**, typical data taken from PEDINFO on the internet at <http://w3.ihl.uab.edu> in 1999, and verified from another source in 2010).



**Figure 2.** Percentage of population with genetic disorders, compared with homosexuality

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.<sup>9</sup> Homosexuality, at 2.4% of the population does not fit into the category of genetic disorders because its incidence is 90 times too high.

Angelman and Prader-Willi syndromes are examples (discussed in the next section) of epigenetic alterations. Homosexuality is also too common to be in the epigenetic class.

So SSA does not seem to be a mutation.

### **Any behaviour links are with many genes**

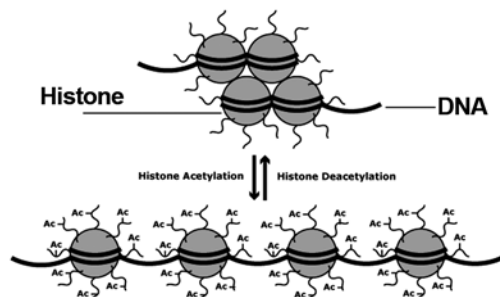
If you're going to argue that human sexual behaviour is dictated, or influenced, by genes in any way, then many genes are involved. But the "many genes" hypothesis doesn't explain homosexuality either because as we'll see, it changes too fast from generation to generation.

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.<sup>4</sup>

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.<sup>5</sup> Thirty mice generations is equivalent to about 1000 years of human breeding. The mice were deliberately bred to create two strains of behaviour: activity and passivity, tested by aversion to space and intense light. Those which reacted positively (exploring the space) were active, those which 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 their activity could not be distinguished from zero. 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 hundreds of genes—with each contributing a tiny part of the whole effect.



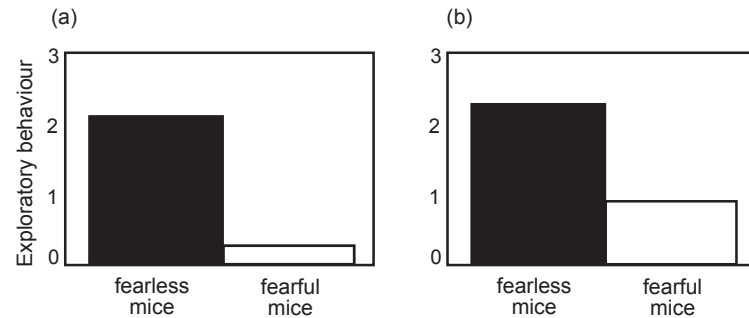
**Figure 3.** The effect of histone changes on the DNA. Acetylation (Ac) of the histones allows genes to be expressed, deacetylation represses genes.

### *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 alteration pattern alters behaviour, and quite often dramatically.

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<sup>22</sup> 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



**Figure 4.** (a) shows the two categories of mice produced at the end of 30 generations of selective breeding. (b) shows the effect in one generation on offspring of the fearful mice handled in a nurturing way by humans. Nurtured mice became much less fearless in a far shorter time than those produced by selective breeding.

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.

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 beside the point because it is becoming increasingly clear that thousands of genes are involved in behaviours rather than hundreds. \*\* A search for a responsible individual gene is almost 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 nature/nurture will both always be involved, right now the pendulum is swinging back to the environment side.

\*\* The authors<sup>22</sup> 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.

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

Moving from mice and dogs 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.<sup>6</sup>

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 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<sup>14</sup> in a study published in *Nature Genetics*, scientists used two strains of fruitfly 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 fruitflies) 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 one into another strain of fruitflies 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.

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.



### **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? The implications of “many genes” for homosexuality would reflect what happened with the mice, or fruitflies: 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 and goes against the known evidence.

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 unusually weak. On average, surveys show homosexuals tend not to have close relationships with their biological families.<sup>11</sup>

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.

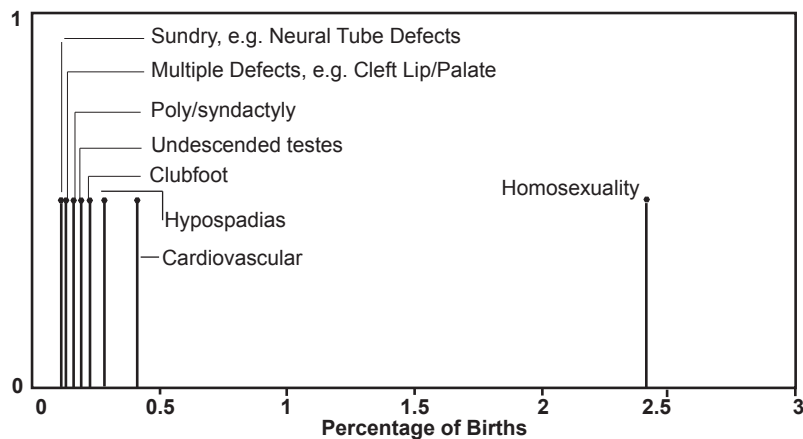
### **Is SSA a fetal development error?**

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 epigenesis, 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). Epigenesis 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



**Figure 5.** Occurrence of pre-natal developmental disorders compared with homosexuality

SSA is (five times or more) higher than any single occurrence of epigenetic abnormality, and hence is very unlikely to arise from some random 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 large post-birth random factor in its development. One crucial difference is that the region of DNA involved in left-handedness is now known, but no genes have been identified clearly for homosexuality (Chapter Nine).

### 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 known events 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  $9.4 \pm 1.1$  years for men and  $11.1 \pm 1.8$  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.<sup>16</sup> for 114 male subjects with SSA and these results, rather typical of others published, are in **Figure 6** below.

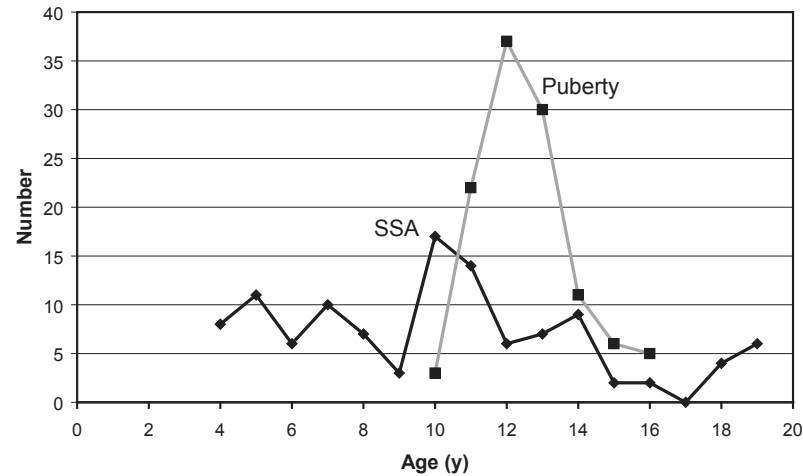


Figure 6. Comparison of puberty and first SSA in males.

The important point about the graph is that puberty is tightly clustered around age 12, and is thought to be 90% genetically influenced<sup>20</sup> 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 others<sup>17</sup> 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).<sup>10</sup> 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.<sup>12</sup>

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?

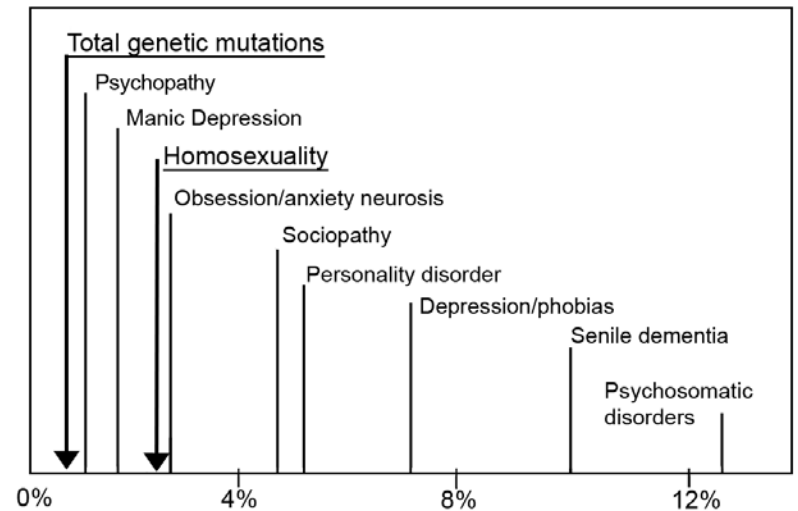


Figure 7. Psychological symptoms in the population compared with occurrence of homosexuality

## 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 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 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 starts 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 recognizes 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.”<sup>13</sup>

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. 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? But we are the ones who must take the first steps beyond them.

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