The Causes of Homosexuality: What Science Tells Us

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First printed in 2006

Published by the Jubilee Centre
Jubilee House, 3 Hooper Street, Cambridge CB1 2NZ

Contents

Introduction

In recent years scientific accounts of homosexuality have shot to prominence in the world media. As a result many now believe that people are born gay and not responsible for their own sexual orientation. Few will challenge this idea but if anyone does they are likely to be met with the rejoinder that science has *proved* that this is so. Yet if pressed for details about this proof most will only recycle the tired old soundbites of the 'gay brain' and the 'gay gene'. In one way science has moved a long way from such simplistic notions. There is an increasing recognition that genetic, hormonal and neurological mechanisms are likely to be complex and interacting and not reducible to the action of a single gene or brain nucleus. However in another way the science of homosexuality has never moved on from either of these. Many still tend to think of biological mechanisms simplistically and deterministically and in isolation from socio-environmental factors. In particular there has been considerable reluctance to address developmental and other psychosocial factors and integrate these into theories of homosexuality. The question remains then, what exactly does science tell us about the causes of homosexuality?

The purpose of this study is to explore in some depth the scientific evidence relating to the causes of homosexuality. It is intended to be a critical evaluation of the various theories focussing on both historical and contemporary evidence. This study will not examine the morality of homosexuality or any of the theological, social and political issues that it raises. However it is important to point out that a number of scientists studying homosexuality are themselves gay and most, but by no means all, of the advocates of re-orientation therapy are religious. With regards to the science this study will examine three areas in particular: neurohormonal, genetic and psychological theories. It must be recognised that there is often considerable overlap between these fields. For reasons of historical precedence neurohormonal theories are considered first, particularly as some genetic theories can be considered a reaction against them. Psychological theories are considered last as these often make the attempt to synthesise biological and social factors.

Finally a word must be said about the definition of homosexuality used in this study. The vast majority of studies use the Kinsey classification, as devised in 1948 by Kinsey and Pomeroy. Subjects are assessed by means of a specially designed questionnaire (which includes both behavioural and psychological components to sexuality) and then placed on a seven-point scale ranging from 0 which is exclusively heterosexual and 6 which is exclusively homosexual.[1] This classification assumes a continuous spectrum of sexual orientation, which is in itself a point of debate. Certainly if such a spectrum exists it is asymmetrical and heavily weighted towards heterosexuality. It is also unclear exactly how a continuum scale phenotype of homosexuality can be correlated to a discrete genotype.[2] For this reason alone an extreme degree of care is needed in interpreting some of the genetic evidence. However its sheer ubiquity means the scale cannot be ignored.

[1] Kinsey and Pomeroy (1948) p 656
[2] Roughgarden (2004) pp 248-54

Neurohormonal Theories

Overview

One of the key areas in the science of homosexuality has been the investigation of the link between brain development and differentiation and the presence or absence of different pre- and post-natal hormones. This area has its roots in the attempts of the nineteenth and early twentieth centuries to correlate different aspects of human behaviour and character to biological and neurological determinants. Beginning with crude estimates of skull size and brain volume and continuing in the twentieth century with the ever more detailed examination and categorisation of different brain structures. In our own time the advent of advanced imaging technology has led to a massive expansion in this enterprise. Now for the first time, displayed upon our computer screens in 3-D and technicolour, we can see the inner workings of the human brain and the myriad different patterns and pathways of neuronal activation.

At the same time we have also come to a much greater understanding of the chemical nature of the brain. The study of neuroendocrinology has revealed the many complex links between the body's nervous and endocrine (hormonal) systems. Hormones are chemical messengers from one cell or group of cells to another which are secreted into the bloodstream. They work by combining with the appropriate receptor which is most often embedded on the surface of the target cell. The interaction between hormone and receptor triggers a cascade effect within the target cell which often has major physiological effects. Within the brain hormones are secreted from the anterior and posterior pituitary glands. These glands link neurons and hormones via complex feedback mechanisms and serve to regulate important aspects of human behaviour such as metabolism and the reproductive cycle. It is for this reason that neuroendocrinology is of particular importance in the debate over human sexuality.

Early Studies

In the second half of the twentieth century experiments carried out on animals suggested that pre-natal exposure to androgen, a male sex hormone, led to the masculinisation of genitalia and brain. In particular genetically female offspring of mothers who had been injected with male sex hormones were observed to have masculinised external genitalia and display more male-typical sexual behaviour.[3] Similar hormonal experiments involving male homosexuals led Dörner to formulate his famous hypothesis of the gay brain. This was that male homosexuals have a female differentiation of the hypothalamic region of the brain caused by underexposure to pre-natal androgen.[4] However Dörner's evidence for this was both indirect and controversial and so the hunt turned to identifying the specific brain structures which might be responsible for human sexuality.

Even before Dörner first posed his hypothesis important steps had been taken in this direction. A region of the anterior hypothalmus known as the preoptic area (POA) had long been implicated in male sexual behaviour. It was known for example that lesions in the POA often caused male vertebrates, across a wide range of species, to lose interest in mating with females. In 1978 Gorski and his colleagues had identified a region of the rat POA which was sexually dimorphic. Seven years later in 1985 Swaab and Fliers at the Netherlands Institute for Brain Research managed to identify a nucleus in the POA larger in men than women which they named the sexually dimorphic nucleus (SDN). However when Gorski examined the human POA he was unable to decide which of the several nuclei there might be homologous to the sexually dimorphic nucleus in the rat. For this reason he named the four neuron groups of the human POA the interstitial nuclei of the anterior hypothalamus (INAH) and numbered them in sequence with the SDN in humans numbered as INAH-1. Further studies by Gorski found INAH-3 to be larger in men than women but failed to find a sexual dimorphism in INAH-1. The reasons for this are unknown but as Breedlove suggests may be because Gorski used a much smaller sample than Swaab and the Dutch group.[5]

These results all paved the way for LeVay's influential study of 1991 comparing the hypothalamic structure of the brains of heterosexual and homosexual men. It is this study in particular which was to be a major milestone in the study of homosexuality even though the significance of LeVay's results are still unclear to this day. Using brain tissue from homosexual and heterosexual men who had died of AIDS (at that time the only reliable way of obtaining homosexual brain material) LeVay examined the four INAH neuron groups. In doing so not only was he able to replicate Gorski's finding that INAH-3 was larger in men than women but he also found that this nucleus was on average twice as large in heterosexual men compared to homosexual men. That is to say that INAH-3 in

[3] Bocklandt and Hamer, *Journal of Endocrinological Investigation* (2003)

[4] Dörner, *Archives of Sexual Behavior* (1988)

[5] Breedlove, *Endocrinology* (2004)

homosexual men was nearly identical in size to the same nucleus in heterosexual women – potentially a highly significant result.[6] Thus LeVay's results seemingly offered confirmation of Dörner's pre-natal androgen hypothesis and identified a promising biological correlate of sexual orientation.

At around the same time two other studies identified differences in brain structure between homosexual and heterosexual men. The first carried out by Swaab and Hofman had in fact been published before LeVay's. This found the suprachiasmatic nucleus (SCN), an area of the brain governing daily rhythms, to be twice as large in homosexual men compared to heterosexual men.[7] Another study published by Allen and Gorski in 1992 found the anterior commisure of homosexual men to be 18% larger than heterosexual men and 34% larger than heterosexual women. Like LeVay the authors of this study considered their results to lend considerable support to the theory that developmental factors, whether pre- or neo-natal, were responsible for the sexual differentiation of brain structure and function and the subsequent formation of sexual orientation.[8]

Although LeVay's study was in general greeted optimistically a note of caution was also sounded in the scientific literature. It was pointed out that his findings contain no direct evidence that the difference he observed in INAH-3 was a causation factor in homosexuality.[9] This was something LeVay himself had noted pointing out that 'the results do not allow one to decide if the size of INAH-3 in an individual is the cause or consequence of that individual's sexual orientation, or if the size of INAH-3 and sexual orientation co-vary under the influence of some third, unidentified variable'.[10] A worry that dogged this study and others at the time was that AIDS and not sexual orientation might be the cause of variation in these nuclei. In his defence LeVay pointed out that heterosexual men who had died of AIDS were still observed to have a larger INAH-3 than homosexual men who had died of the disease.[11] However LeVay's sample size was really too small to make such definitive statements. As Allen, an expert in this field, admitted at the time 'AIDS pathologies could influence the size of the nuclei'.[12]

Another important set of studies was published by Swaab, Hofman and Gooren in 1993 and 1995. Not only did these replicate the authors' original results of sexual differentation in the SDN (INAH-1), they also provided a plausible reason why Allen and Gorski had failed to observe this – their sample was biased towards ages where size difference is minimal. However their most important finding was that although there was no difference in the SDN between homosexual and heterosexual men there was a considerable difference in the size of the SCN (again this confirmed their earlier findings). The SCN in homosexual men was observed to be 1.7 times larger in heterosexual men and to contain about twice as many cells. While the authors did not examine INAH-3 they did point out that LeVay had not carried out cell counts for this nucleus but only volume measurements. The implications of this were to render his results even more provisional. The real significance of these studies however was in its refutation of Dörner's hypothesis. Earlier hormonal experiments by Gooren had replicated Dörner's result in heterosexuals as well as homosexuals and had managed to produce both a positive and negative feedback on hormonal injections which cast doubt on Dörner's methodology. The final nail in the coffin for his theory (at least as far as these authors were concerned) was the finding that the human SDN did not differ between homosexuals and heterosexuals. However their discrediting of Dörner's hypothesis did not mean that they completely abandoned the hormonal hypothesis; instead they merely restated it in a form consistent with their results.

In recent years no consistent correlations between brain structures and homosexuality have emerged.

Swaab and his colleagues suggested the need to re-evaluate the relationship between sex dimorphism of the SDN and pre-natal or peri-natal sex hormones. They pointed out that although during the second half of gestation there is a big peak in sex hormone levels (during which the genitalia are formed) there is no significant sex difference in the SDN. Also no oestrogen, androgen nor progestin receptors were found in the foetal brain at this point. After birth the SDN cell number is then observed to increase rapidly in *both* boys and girls up to the age of 2-4 years. It is only after this point that sexual differentation in the SDN occurs due to cell death in girls but not in boys. The fact that significant developments in the SDN occur post-natally and not pre-natally suggested that social factors and not just hormones might be significant. Although the authors did not state it such an account rendered oversimplistic accounts linking homosexuality to pre-natal sex hormones somewhat untenable.

The human SCN is considered to be the principle component of the human biological clock, both generating and co-ordinating hormonal, physiological and behavioural rhythms and likely to be involved in

[6] LeVay, *Science* (1991)
[7] Swaab and Hofman, *Brain Research* (1990)
[8] Allen and Gorski, *PNAS* (1992)
[9] Baringa, *Science* (1991)
[10] LeVay, *Science* (1991)
[11] LeVay, *Science* (1991)
[12] Baringa, *Science* (1991)

some way with reproduction. Swaab and his colleagues suggested that a large SCN in homosexuals could be ascribed to early processes of brain development. Like the processes affecting the SDN they envisaged these processes as taking place mainly after birth rather than before. This is because it is only after birth that rapid growth of the SCN occurs reaching a peak at 13-16 months. After this point cell death usually occurs meaning that adult men normally have a much smaller SCN than this peak value. An enlarged SCN in homosexual men suggested to them that for some reason programmed post-natal cell death seems to have been reduced in homosexual men perhaps by global developmental factors influencing both sexual orientation and SCN size. However although the authors did not think it likely that homosexual behaviour could alter the size of the SCN they did concede the possibility.[13]

In recent years no consistent correlations between brain structures and homosexuality have emerged. In fact such research has stalled significantly. The whole field of sexuality and the brain has been plagued by uncertainty and contradictions. To give a well-known example there have so far been twenty-three studies into sex difference in the corpus callosum. The first such study found both size and shape differences in the splenium of the corpus callosum but none of the other twenty-two were able to replicate this result. Results for orientation differences have been just as confused. Byne and Parsons attempted a careful replication of LeVay's original 1991 experiment. Unlike LeVay they found no variation in gender for the size of INAH-3 which also undermines any putative sexual orientation variation.[14] More recently Byne and his team also found large overlap between heterosexual and homosexual men for the size of the anterior commisure casting doubt on Allen and Gorski's original results.[15] Furthermore Swaab's results for the SCN are also yet to be replicated.

Clicks and Sniffs

Given the confusion of the early research into brain structures and homosexuality the best that could be said was that hormonal mechanisms still remained a promising avenue of research. The arrival of imaging technology has meant that much more sophisticated observations and analyses of brain mechanisms are now possible. Also in recent years attention has turned to body structures or functions where hormonal influence during development could cause noticeable differences. These include indicators such as hand preference, finger-length ratios, auditory systems and body odour preference. Both avenues of research have allowed the study of homosexuality in women as well as men, most previous studies had the disadvantage of neglecting this important area.

The research published by McFadden and Pasanen in 1998 examined the auditory systems of male and female homosexuals compared to male and female heterosexuals. In particular they measured click-evoked otoacoustic emissions (CEOAE) which are echo-like waveforms emitted by the inner ear in response to a transient stimulus. They were known to be stronger in females than in males as well as in the right ear compared to the left ear. McFadden's interest in these waveforms was due to the fact that females from opposite sex dizygotic twin pairs had been found to have OAEs more like those of males. It was hypothesised that this was due to higher levels of pre-natal androgens circulating in the womb. By evaluating OAEs from homosexuals compared to heterosexuals McFadden hoped to shed new light on hormonal theories of male and female homosexuality. What McFadden found was that in females CEOAEs were stronger in homosexuals than heterosexuals whereas in males there was no significant difference observed. The most interesting implication of this result was not the evidence against Dörner's hypothesis of a globally female brain in male homosexuals but the suggestion that the auditory systems of female homosexuals might have been masculinised by overexposure to pre-natal androgens. However McFadden noted that this interpretation of the results was not unproblematic, particularly as the only study which had been done of opposite sex dizygotic twins did not show any increased prevalence in female homosexuality. McFadden also pointed out that the possibility that homosexual lifestyle could diminish OAEs could not be entirely ruled out.[16]

Within the last two years Savic and her Swedish research team have been able to carry out much more advanced studies of brains of homosexuals using positron emission tomography to monitor cerebral blood flow. The two sets of experiments, one carried out on homosexual men the other on lesbians, were designed to study the brain activation patterns on smelling (putative) male and female pheromones. In the animal kingdom choice of mating partner is highly influenced by sex-specific chemical messengers called pheromones which are processed in the anterior hypothalamus. Although it was (and still is) uncertain whether human pheromones exist two compounds called AND (a testosterone derivative found in male sweat) and EST (an oestrogen derivative found in female urine) had previously been proposed as possibilities. In previous laboratory experiments it had also been found that women smelling AND and men smelling EST

[13] Swaab et al., *Hormone Research* (1992)
[14] Byne and Parsons, *Archives of General Psychiatry* (1993)
[15] Byne et al., *Brain Research* (2002)
[16] McFadden and Pasanen, *PNAS* (1998)

caused a sex-differentiated reaction in the anterior hypothalamus. However when men smelled AND and women smelled EST the reaction was only as would be expected for a common odour. The interpretation was that AND and EST act bimodally as odours and pheromones allowing them to activate different regions of the brain. This made them ideal candidates to test whether differing reactions existed between homosexuals and heterosexuals as well as between males and females.

Savic found that when homosexual men smelled EST the primary area activated was the olfactory region of the brain with the anterior hypothalamus only showing minor activation. Hypothalamic activation in homosexual men was only observed when smelling AND, in common with heterosexual women, and was not shared with heterosexual men. Her conclusion was that homosexual men process AND congruently with heterosexual women rather than heterosexual men.[17] Interestingly Savic found rather different results in her experiments with homosexual women. She observed that lesbians did not show a differentiated pattern of activation with AND and EST but engaged the classic odour processing circuits when smelling both of these. By contrast, as before, in heterosexual women AND activated the anterior hypothalamus whereas EST activated the olfactory regions. The only congruence between lesbians and heterosexual men was in a shared hypothalamic cluster activated upon smelling EST. Unfortunately it was unclear whether these differences were a factor of the compound (EST is much less studied compared to AND and seems to have less prominent pheromone-like features) or of the nature of female homosexuality. Savic was also clear that her data did not allow discrimination between cause and effect. This meant that differing patterns of activation might have been fixed from development (for example by pre-natal hormones) in the homosexual brain or they could have been sensitised by behaviour, for example activation with AND for heterosexual women might reflect sexual exposure to men[18]

A similar study to Savic's was published by Martins in 2005. However instead of using candidate pheromones such as AND and EST this focussed on body odour preference. Humans produce individually unique body odours called odour prints. These are perceptible to others and may also act as pheromones. Differences between these odours have also proved distinguishable with humans able to discriminate between kin and non-kin odours and newborn babies able to pick out scent of their mother from other women. In order to test the link between body odour preference and sexuality Martins obtained odours from heterosexual and homosexual men and women and mixed these to produce four different odour samples. Participants in the experiment were then forced to choose between four different odour pairs: heterosexual men and homosexual men, heterosexual men and heterosexual women, heterosexual women and homosexual women, homosexual men and homosexual women. The most interesting result was that gay males exhibited strikingly different odour preferences from heterosexual men and women and lesbians: gay men preferred odours of other gay men and heterosexual women, whereas for all other groups odours from gay men were the most non-preferred. This preference was found not to depend on perceived intensity but on perceived pleasantness.

Martins interprets her results as favouring a sophisticated neurohormonal hypothesis. She points out that individual's odour prints are primarily established by the major histocompatibility complex (MHC) which is a linked set of genes regulating the immune system. Individuals tend to prefer odours from individuals with some MHC alleles in common but to avoid odours from individuals with either one or no allele matches as well as those which are entirely identical. Martins hypothesises, based on similar work in mice, that sensitivity to sex hormones may be linked to genes in the MHC. Thus differences in MHC genes influencing sexual orientation may relate to differences in MHC regulated odour production (and subsequent preference). Ingenious as such a theory is it remains speculative. In particular the genetic evidence that candidate genes for homosexuality might regulate hormone production or sensitivity remains highly provisional.

Although Martins refers to the alternative theory that differences in production or perception of body odour may result from a variety of environmental factors such as odour familiarisation or differences in lifestyle she does nothing in the way of assessing this.[19] This seems signally unwise, particularly as she so frequently draws attention to the fact that humans can discriminate between kin and non-kin odours as well as recognise the odours of those they know. In fact as Throckmorton suggests issues of odour familiarisation may seriously call into question Martins' interpretation of her own results. The fact that participants could recruit their friends to the study (apparently as donor or smeller) may have meant that many of the gay men knew each other. This coupled with the fact that friends recognise the odour of friends could be the reason why gay men so strongly preferred the odour of other gay men. This also may have helped screen the significant result that gay men found the odours of heterosexual women by far the most pleasant even compared to the odour of other gay men. Throckmorton also points out that if odour

[17] Savic et al., *PNAS* (2005)
[18] Savic et al., *PNAS* (2006)
[19] Martins, *Psychological Science* (2005)

preference is meant to reflect sexual preference in some way then her results are rather strange and even contradictory.[20]

Taken together it seems far from clear that recent results on odour processing and preference support either neurohormonal theories or ideas of innate homosexuality. As Savic pointed out in a 2005 article in the Chicago Tribune 'I want to be extremely cautious – this study does not tell us anything about whether sexual orientation is hardwired in the brain, it doesn't say anything about that'.[21] In her article Savic also quotes a 2002 study in *Nature* suggesting that smell is highly subject to learning and habituation.[22] This makes plausible the theory that men and women learn to respond to chemical signals as a result of sensitisation through sexual experience (the one possibility that Martins offered little consideration of). Whether or not McFadden's findings are amenable to a similar environmental analysis is an entirely different question which remains to be answered in either the positive or negative.

Fingers, Hands and the FBO

For a long time a link between homosexuality and left handedness has been bandied about at the popular level. In more recent years a number of scientific studies have examined this question in detail. These have often been combined with studies of the fraternal birth order effect (FBO). The FBO was first observed in a survey carried out by Blanchard and Bogaert in 1996. Very simply this is that the more older brothers a boy has the more likely he is to grow up to be gay. No effect is seen with older sisters or younger siblings of either sex. The FBO could be relevant for up to 15% of homosexuals and has been replicated a number of times since it was first observed.[23] The FBO has been interpreted by its discoverers and others as evidence in support of a (modified) pre-natal hormone theory called the maternal immunisation hypothesis. For a first son the mother has very little exposure to his male-specific proteins since the placenta acts as a barrier. However upon delivery there will be an inevitable mixing of maternal and foetal blood meaning that the mother's immune system will for the first time experience these alien proteins. If her body mounts an immune response it is hypothesised that antibodies remaining in her bloodstream directed against male-specific proteins could effect sexual differentiation in the brains of subsequent children making them more likely to become homosexual in later life.[24]

One possible testing ground for the FBO and pre-natal hormones is finger length ratio in men and women. Like auditory and olfactory functions these are considered to be useful hormonal indicators. In women the index finger (2D) is almost the same length as the fourth digit (4D), whereas in men the index finger is more often shorter than the fourth digit. This sex difference is thought to reflect levels of pre-natal androgens. In addition the sex difference is greater in the right hand than the left which seems to indicate that the right hand ratio is more sensitive to foetal androgens. In 2000 Breedlove published a paper analysing data from a survey of sexual orientation, handedness, finger length ratio and sibling number carried out in San Francisco. This survey found that the right hand 2D:4D ratio in homosexual women was not appreciably different from heterosexual men. In contrast the 2D:4D ratio in homosexual men was not different from heterosexual men for either hand. Separating out the male participants based on birth order confirmed the FBO, finding a correlation between number of male siblings and incidence of homosexuality. It was also found that the male 2D:4D ratio varies with the number of older brothers with the ratio usually more masculine for men with a number of older brothers.

Breedlove interpreted the masculinised 2D:4D right hand ratio in lesbians as indicating exposure to a higher level of foetal androgens in the womb. For homosexual men however there was evidently no simple process involved. Instead, going against the trend of previous theories of underexposure to pre-natal hormones, Breedlove proposed a hyper-androgenisation theory. This he used to explain the more masculine 2D:4D ratios for boys with more older brothers by suggesting that maternal immunisation might increase foetal androgen levels for subsequent births. Although Breedlove's results are interesting the theory he uses to explain them remains highly speculative. The details of the mechanism behind 2D:4D finger length ratio determination are completely unknown. Any link between this unknown mechanism and the maternal immunisation hypothesis, which is itself only speculative, must be considered very tenuous. In addition although Breedlove admits that maternal influence on finger growth of subsequent sons may occur after birth he gives no consideration of environmental or social factors which could be relevant.[25]

Breedlove based a number of his ideas on Lalumière's study of the link between homosexuality and

[20] Throckmorton: http://www.drthrockmorton.com/article.asp?id=147
[21] Chicago Tribune (May 9, 2005)
[22] Throckmorton: http://www.drthrockmorton.com/article.asp?id=146
[23] Blanchard and Bogaert, *Am. J. Psychiatry* (1996)
[24] Breedlove et al., *PNAS* (2006)
[25] Breedlove, *Nature* 404 (2000)

handedness which was also published in 2000. Examining previous research which had been carried out Lalumière found that the odds of non-right-handedness (nrh) were 34% higher for homosexual than for heterosexual men. This established handedness as a reliable correlate of sexual orientation. Lalumière also gave some consideration to possible mechanisms which might explain the connection between homosexuality and nrh. One of these was yet another modification of the pre-natal androgen hypothesis. Lalumière suggested that homosexuals may have been exposed to varying levels of testosterone in the womb. Excess testosterone at one stage might influence sexual orientation while lack of testosterone at another stage influence the development of handedness or alternatively excess testosterone might influence both orientation and handedness (the hyper-androgenisation hypothesis as used by Breedlove). Lalumière's other proposal was a theory of developmental instability. This suggested that homosexuality and nrh might coincide because susceptibility to one perturbation in the womb also implies susceptibility to other perturbations (this seems similar to Swaab and Hofman's suggestion of global development factors influencing orientation as well as brain structures).[26]

One of the most recent studies on the FBO and handedness is by Blanchard one of the co-discoverers of the FBO. In this article he pointed out the mass of evidence which had now accumulated for the FBO, incorporating data from a survey of over 10,000 people.[27] Pooling together a number of previous studies with new research he hoped to consider the interaction of FBO and handedness in the development of male homosexuality. His results suggested that the positive correlation between homosexuality and older brothers held only for right-handed males. More specifically for men with no older brothers homosexuals were more likely to be nrh, whereas for men with one or more older brother they were less likely to be nrh. The odds of homosexuality were found to be higher for nrh men or those with older brothers relative to men who satisfied neither of these requirements. Taken together the odds for men who were both nrh and had older brothers were similar to those for men who satisfied neither. Quantatively odds of homosexuality were 41% higher for nrh men and 50% higher for men with older brothers.

In essence Blanchard's results suggest an 'unknown factor' associated with nrh which increases the odds of homosexuality in first male births but which also prevents older brothers from increasing the odds in subsequent male births. His findings are complex and admit of multiple explanations but the two that he gives are variants of those given by Lalumière. The first more complex theory is that of foetal testosterone levels. This assumes that males who are more sensitive to foetal testosterone are more likely to be nrh and homosexual. It also requires the supplementary proposition that the effects of too much testosterone (hyper-androgenisation) on development are the same as the effects of too little testosterone (hypo-androgenisation). Blanchard points out the paradoxical nature of this idea and stresses that it has never been observed in humans (although may have been in animals). He also remarks on the contested nature of the theory that high levels of testosterone produce nrh.[28] The clever part of this theory comes when it is combined with the FBO. This suggests that the reason why nrh with older brothers are insulated from higher incidences of homosexuality is that the effect of high testosterone levels in over-masculinising the brain are exactly counteracted by the effect of maternal immunisation in which anti-male antibodies feminise the brain. A much simpler hypothesis and unprecedented in the literature is a variant of that of developmental instability. Blanchard proposes a novel selection effect suggesting that the number of nrh homosexuals with older brothers is lower than expected because the combination of being nrh and a late birth is toxic enough to render subjects unavailable for research (either by death or severe-impairment). This theory tallies with previous observations that both older brothers and nrh correlate with low birth weight. It suggests that boys who are late birth and nrh while being healthy will have a higher probability of becoming homosexual.[29]

In his article Blanchard did not give any consideration to social explanations of the FBO. However a recent study by his co-discoverer of the FBO was specifically designed to compare pre-natal and post-natal theories of this effect. Bogaert examined the parental and sibling characteristics of homosexual and heterosexual men from across a wide variety of family backgrounds. His hypothesis was that if rearing or social factors caused the FBO then all older brothers as well as amount of time raised with them should predict sexual orientation. Whereas if a pre-natal mechanism caused the FBO then only biological older brothers should matter and time reared should have no effect. Using a variety of analyses Bogaert found, as he expected, that only the number of biological older brothers was a

There certainly seems a good deal of evidence to suggest that brain mechanisms (if not architecture) can alter with behaviour.

[26] Lalumière, *Psychological Bulletin*, (2000)
[27] Blanchard, *J. Theor. Biol* (2004)
[28] Previc, *Brain Cogn.* (1994)
[29] Blanchard et al., *Journal of Hormones and Behavior* (2006)

significant predictor of homosexuality. Unsurprisingly he therefore suggested that these results favoured the pre-natal hormone and maternal immunisation hypothesis. However while the overall trend does support Bogaert's hypothesis some of the individual results suggest social factors could be important. Thus for example for adopted children the time reared with non-biological older sisters turns out to be almost as good a predictor of homosexuality as the number of biological older brothers (significance parameter of 0.15 compared to 0.22). This raises the possibility that other hidden social factors or biases may have a role to play in explaining the FBO.

New Perspectives on Neurohormonal Theories

As we have seen research into some of the pre-natal theories of homosexuality has progressed from investigation of brain structures to investigation of brain function and surveys of hormonal indicators. While many of the early results such as those of LeVay and Gorski are still debated, some more recent studies do seem to point to functional differences in the brains of homosexuals and heterosexuals. However as is often the case in these situations we are here confronted with the perennial question of the chicken and the egg. Which came first the brain patterns or the homosexuality? There certainly seems a good deal of evidence to suggest that brain mechanisms (if not architecture) can alter with behaviour. For example is it not possible that repeated sexual behaviour and exposure could cause a particular pattern or response to become so ingrained that it seems innate? That question shall be considered in more depth later.

There certainly are many scientists who favour the pre-natal hormone hypothesis. It has been repeated so often now that for some it has become a dogma – the *only* possible explanation of homosexuality.[30] However as Blanchard points out all of the mechanisms proposed are highly tenuous if not contradictory. Traditionally hormonal theories have tended to be bolstered by examples from the animal kingdom. A good recent example is a paper by Breedlove published in 2004. This attaches great significance to the discovery of a sexually dimorphic nucleus in sheep which is smaller in those rams who prefer mounting other rams – the equivalent of LeVay's purported result with INAH-3. However the problem with this is that none of the SDN nuclei in rat, sheep or human can be shown to be homologous to each other which makes any comparisons very difficult.[31] As one rat researcher remarked at the time of LeVay's seminal discovery 'extending that kind of data to humans involves a huge step'.[32] It may be also that an equivalent mechanism which has a major effect in rats or sheep is comparatively unimportant in humans compared to other factors.

One scientist who has expressed serious dissatisfaction with the prevailing hormonal theory is Bocklandt (although admittedly he has his own agenda to advance). He points out that experiments on animals have a number of key differences to humans. In particular hormonal treatments on animals go far beyond any naturally occuring variation in androgen levels even going so far as to render alterations in their external genitalia. He also points out that two candidate genes involved in the testosterone pathway – androgen receptor and aromatase – have both been studied and found to be unimportant in homosexuality.[33] This is particularly interesting as aromatase was one of the compounds that Breedlove suggested might be important based on the experiments on sheep.

Finally it seems unlikely that Bogaert has had the last word on social factors being involved in homosexuality. An important survey carried out by Bearman and Bruckner on a large sample of adolescents, which shall be returned to in more detail below, points to an older brother effect contrary to that observed originally by Blanchard and Bogaert. It also offers a very interesting critique of their methodology and that of most other similar surveys, suggesting that recruiting from homophile organisations could introduce serious bias. Even those surveys which have confirmed the FBO suggest that it is a relatively minor effect. One recent study (again referred to below) suggests that the FBO can account for less than 7% of the variance of sexual orientation in their sample population.[34] At most therefore neurohormonal theories can only account for a small fraction of incidence of homosexuality (and even then it is perilous to ignore social factors) and so can neither be invoked as an explanation of the whole nor a proof of innateness.

[30] Quinsey

[31] Breedlove, *Endocrinology* (2004)

[32] Baringa, *Science* (1991)

[33] Bocklandt and Hamer, *Journal of Endocrinological Investigation* (2003)

[34] Camperio-Ciani, *Proceedings of the Royal Society: Biological* (2004)

Genetics of Homosexuality

Animal Genetics

The modern science of genetics began with Gregor Mendel's famous experiments with pea plants in the Abbey gardens at Brno in the nineteenth century. The same century saw the publishing of Darwin's ground-breaking book *On the Origin of Species* and the attempt of him and others to understand the underlying patterns of inheritance and evolution. The consensus which emerged, after the dust had finally settled, was of man as a higher animal - the end (and not necessarily the goal) of an evolutionary continuum stretching right back to the simplest bacterium. For our society today it is hard to understand the true significance of this paradigm shift. For the first time it became possible, even desirable, to see animal features and behaviour as analogues of more complex human features and behaviour. In recent years there have been a number of important studies into the genetic substrates of complex animal behaviours. Many of these have focussed on reproductive behaviour and some have even touched on homosexuality. An overly-simplistic reading of these studies could be used to bolster the claim for innate homosexuality, however, as we shall see, the application of animal genetics to human behaviour is by no means simple.

Monogamy is an example of one complex social trait that it has proved possible to analyse genetically. Although common in birds monogamy is comparatively rare among mammals where it has an incidence of only around 3%. Within the last ten years our understanding of this social behaviour and how it may have evolved has been significantly advanced by experiments on two different species of American vole: monogamous Prairie voles and closely-related promiscuous Meadow voles. Studies of the brains of these two species have brought to light a significant difference between them in a region known as the ventral pallidium. In this region prairie voles highly express vasopressin 1a receptors (V1aR), by contrast in meadow voles these receptors are largely absent in this region. For this reason it was hypothesised that vasopressin acting in consort with dopamine was a crucial regulator of monogamous behaviour. To test this hypothesis Young and his group used genetic transfer by means of injecting a virus to overexpress V1aR in the ventral pallidium of the meadow vole. These genetically modified voles were then socialised with a female vole for 24 hours and then given the choice of the established partner or a novel female. The results showed that the modified voles displayed a much higher degree of partner bonding than two other control groups (one with another gene expressed in ventral pallidium and one outside ventral pallidium). However when the modified voles were then injected in the ventral pallidium with a dopamine receptor (D2) antagonist they reverted back to promiscuous behaviour. In a similar way the modified voles were also tested for time spent socialising with pups. Again it was found that they displayed considerably more paternal behaviour than the control groups. However by injecting the same receptor antagonist into the medial amygdala it was found possible to suppress paternal behaviour without effecting the unusual degree of partner bonding.

Vasopressin receptors are involved in the formation of social memory. Based on his results with voles Young suggested that during partner bonding there is a concurrent activation of neural pathways associated with social recognition and reward or pleasure. This causes convergent V1aR and D2 activation in the ventral forebrain of the voles which leads to an association between the rewarding nature of sex and the partner's olfactory signature (main means of social recognition among voles). Reinforced by repeated encounters this finally results in a biologically conditioned partner preference. The fact that unmodified meadow voles have very few V1aR in their ventral forebrain therefore explains why they are promiscuous and do not form partnerships. Young was also able to point to a plausible genetic reason for this difference in social behaviour. Analysis of the gene underlying V1aR reveals that monogamous voles have an expansion of repetitive microsatellite DNA in the 5' regulatory region of the gene whereas promiscuous voles do not. Microsatellite DNA is highly unstable and is able to rapidly expand and contract which could give rise to different brain V1aR expression patterns.[35] A follow-up study has found some (slightly weak) evidence for a correlation between V1aR levels and length of microsatellite DNA in individuals.[36] However regardless of exact mechanism it is clear that changes in regional expression of a single gene can have profound effects on animal behaviour.

Along with mice the ubiquitous fruit fly (*Drosophila Melanogaster*) has long been used as a standard exemplar of genetics. It is therefore no surprise that the most important studies of genetic mechanisms underlying sexual behaviour have involved this humble fly. *Drosophila* have a complex courtship ritual which involves the exchange of visual, olfactory, gustatory, tactile, acoustic and mechanosensory stimuli between

[35] Young et al., *Nature* (2004)
[36] Young et al., *Genes, Brain and Behavior* (2005)

sexes. Normally males court other females but in the 1960s scientists discovered *fruitless* mutants which court males (in exclusive male company). The *fruitless* gene found in these mutants, termed *fru*, was first cloned in 1996. It was then discovered that through a complex series of mRNA mediated mechanisms *fru* is able to splice male- and female-specific transcripts which act on the nervous system of the flies (Fru^M and Fru^F).[37] In a series of elegant experiments Dickson and his group in Vienna have demonstrated that male Fru proteins are necessary and sufficient for male courting behaviour. By forcing male flies to splice the female-specific transcript Dickson produced males with the same characteristics of the most extreme *fru* mutants – that is they were sterile and more interested in courting males than females. In the same way females forced to splice male-specific transcripts courted other females even forming identical courtship chains to those seen in males. In a final coup-de-grace Dickson was able to feminise the male abdominal glands to produce female pheromones. When these altered males were placed with females expressing the male-specific transcripts, role-reversal was observed with females courting males.[38]

It seems highly unlikely that a simple neural network could specify such a complex behavioural trait as human sexuality.

Both Dickson's group and another team of scientists also generated flies in which they were able to stain the parts of the nervous system (around 2%) which show sex-specific expression of Fru. The central nervous systems of both males and females were found to be very similar in Fru expression and *fru* products were found in almost all sensory organs implicated in courtship. However olfactory sensory neurons showed some sexual dimorphism with pheromone-response receptors larger in some areas of the brain for males than females. By inhibiting Fru^M function in the olfactory neurons of males and 'masculinised' females courtship behaviour was able to be considerably reduced and even switched off (by transient inactivation of the particular neurons). This proved that these receptors were implicated in male courtship rituals and suggested that *fru* could act as a single gene swich between male and female behaviour regardless of morphological sex.[39] A subsequent study by a Japanese group has identified a subset of Fru^M expressing neurons showing marked sexual dimorphism in number and projection pattern. This suggested to the authors that Fru^M expression produces a male-specific neural circuit implicated in heterosexual courtship which is otherwise programmed to die during development. Importantly this group also suggested that such neurons could act as input sites for pheromonal information conveyed by gustatory contact. Such contact has been shown to be involved in *Drosophila* partner choice. This suggests that the enhanced homosexual preference in *fru* mutant males might be due to a failure to integrate pheromonal information crucial for partner choice and mating decisions. That is, it could all be a case of mistaken identity.[40]

The discovery that a single gene is responsible for regulating a behaviour as complex as the courtship sequence of *Drosophila* has opened up a whole new field in behavioural genetics. Specifically it suggests that neural circuits underlying other complex behaviours (in animals and perhaps even humans) may be the result of similar specified genetic hierarchies. Baker and others have suggested detailed criteria for showing that a genetic mechanism specifies a behaviour (regardless of the importance of environmental factors). In essence it must be shown that a gene is necessary and sufficient to direct the outcome of a process. Necessity can be established if a gene's absence leads to a failure in the process and sufficiency by showing that the gene's expression in atypical cells will still direct the process. Thus for example expression of *eyeless* genes in the wings of *Drosophila* leads to the subsequent development of extra eyes. In terms of behavioural traits (as opposed to morphological features such as eyes) this means that a gene must be proved both necessary and sufficient for the construction of dedicated neural networks. Genetic specification however does not necessarily exclude adaptibility. As Baker suggests it is possible that the potential for experience to modify behaviour may be genetically (or otherwise) built into the neural circuits themselves.[41] Studies of the African fish *Haploch burtoni* provide evidence for this thesis. This fish comes in two forms of males: the dominant which is agressively territorial, brightly coloured and has high levels of circulating testosterone and reproductive success, and the subordinate which differs in all respects. It has been found that the brains of dominant males contain larger gonadotropin releasing neurons than subordinates, with the difference particularly marked in the POA region. This neurological difference has been found to have a specific genetic substrate. The most interesting feature of this genetic mechanism regulating gonadotropin release is that its results are found to be dependent on the fish's social status. Therefore a subordinate fish that becomes dominant will show a corresponding increase

[37] Kyriacou, *Nature* (2005)
[38] Demir and Dickson, *Cell* (2005)
[39] Dickson et al., *Cell* (2005); Manoli et al., *Nature* (2005)
[40] Kimura et al., *Nature* (2005)
[41] Baker et al., *Cell* (2001)

in neuron size and a dominant fish that becomes subordinate, a corresponding decrease.[42]

Genetic studies on animals have raised the question of whether neural circuits specified by transcription factors or other gene regulatory mechanisms could be responsible for human homosexuality. However it needs to be always borne in mind that although animal behaviours can be complex and intricate they often are found to have a simple instinctual root. Thus in *Drosophila* a complex series of courtship manoeuvres is seen to be dependent entirely on 'chemical' instinct mediated by pheromones. Whilst it is true that humans do show some instinctual behaviour it seems highly unlikely that a simple neural network could specify such a complex behavioural trait as human sexuality. This is particularly true given, as we have already seen, the many questions that remain unanswered about the role of neurological determinants in human sexuality and relevance or even existence of pheromonal mechanisms in human sexual interaction. The fact that humans have lost over 50% of their olfactory genes while in general conserving over 90% of other genetic material suggests caution in applying animal genetics straightforwardly to humans.

An important study into the neuroanatomy of speech and language suggests a possible ideal for other human behavioural genetic studies. The discovery of a mutation in a transcription factor called *FOXP2* in a family with inherited speech and language disorders enabled scientists to trace the neural expresssion of this gene and thus track the effects of its mutation on brain structure and function. For example it was found that affected subjects had reduced volume caudate nuclei and different brain activation patterns compared to their unaffected family members. This suggested a mechanism whereby *FOXP2* mutation might lead to the development of alternative networks controlling orofacial musculature and therefore manifesting as a speech impairment. In fact researchers were even able to specify a putative neural network although were unable at the time of writing to prove this by establishing the one remaining link in the chain.[43] The *FOXP2* study serves as a benchmark to assess other genetic studies. Applying Baker's criteria, to prove an sole underlying genetic cause of homosexuality (or more weakly some types of homosexuality) it must be shown that a gene or genetic mechanism exists which is both necessary and sufficient for the development of adult homosexuality. Anything less than this cannot constitute a reasonable degree of proof for such a strong statement.

Twin Studies

Twin studies have been an important component of genetics from its inception. Their main use has been in the study of heritability of various traits. All twin research is based on the simple premise that since identical twins have identical genotypes, any difference between them can solely be attributed to environmental factors. Therefore for any one trait, even as complex as homosexuality, it is hypothetically possible to separate out the various components of genetic and environmental influence. Twin studies can also be rendered more sophisticated by exploiting the differences between different types of twins. Monozygotic (MZ) or identical twins occur when a single egg is fertilised to form one zygote which then subsequently splits to form two embryos. By contrast dizygotic (DZ) twins occur when two fertilised eggs are implanted into the uterus at the same tim, these twins are extremely unlikely to share the same chromosomal profile. Twin concordance studies often compare MZ twins, DZ twins and normal siblings to try and come up with a detailed picture of familial inheritance.

The first 'proper' scientific twin study of homosexuality was Bailey and Pillard's seminal study of 1991. Before this there had been various other studies but these had all reported extraordinary high concordances and were tainted by poor methodology and small sample sizes. An example is Kallman's study of 1952 which found 100% concordance for monozygotic (MZ) twins compared to only 15% for dizygotic (DZ) twins. It was soon realised that Kallman's results were distorted by his recruitment of homosexuals from hospitals and psychiatric institutions as well as his lack of zygosity analyses. In order to obtain more reliable and widely applicable results Bailey and Pillard recruited through homophile publications rather than institutions. They also attempted to obtain the best possible zygosity and sexual orientation classifications possible through means of detailed questionnaires. Analysing their results Bailey and Pillard found concordance rates (for homosexuality and bisexuality) of 52% for MZ twins, 22% for DZ twins and 11% for adoptive brothers. Assuming a base rate of 4-10% homosexuality in the general population this gave heritability between 0.31 and 0.74. This caused the authors to conclude that homosexuality was likely to have a strong genetic component, although in fact their method was not able to discriminate between genetic and environmental factors. The study also had sought to examine a purported link between homosexuality and a disorder called childhood gender non-conformity but concluded that although a strong link existed it did not constitute genetic 'loading' for homosexuality.[44]

This study was followed up with another on female homosexuality in 1993. At the time (and to a lesser

[42] Robinson and Shahar, *Genes, Brain and Behavior* (2002)

[43] Vargha-Khadem et al., *Nature Neuroscience* (2005)

[44] Bailey and Pillard, *Archives of General Psychiatry* (1991)

extent even today) this was a highly neglected area of research and very little of any importance was known about it. The results were found to be very similar with concordance rates of 48% for MZ twins, 22% for DZ twins and 6% for adoptive sisters. Estimated heritabilities were again high ranging from 0.27 to 0.76. Again the study suggested that neither childhood gender non-conformity nor extreme homosexual behaviour constituted genetic loading. Although, again, the study did not allow quantitative discrimination between genetic and environmental factors it was used to illuminate qualitative considerations. For example, MZ cotwins who differ in sexual orientation can only do so due to environmental factors (whether pre-natal or post-natal), suggesting the general conclusion that effective environment for female homosexuality comprises factors not even shared by the twin. This loosely implied that post-natal environmental factors were likely to be the most relevant for sexual orientation, particularly the 'idiosyncracies of the parent-child relationship'. We shall return to this point later when we give a detailed examination of childhood gender non-conformity. Finally the observation that probands had significantly more homosexual sisters than brothers suggested to the authors that male and female homosexuality were probably independent etiologically.[45]

In the years since Bailey's two seminal studies there have been a number of attempts made to refine his methodology. Both the 1991 and 1993 studies had three serious flaws: firstly the ascertainment bias applicable to all twin studies, secondly the problem of recruiting through homophile organisations and thirdly the fact that concordance data was derived from only one twin in a pair. The last of these was potentially the most serious problem. One study (by Bailey himself among others) gave a less than 50% chance that heterosexuals will know their twin is non-heterosexual as well as the fact that homosexuals were more likely to misidentify their heterosexual twin as homosexual.[46] All or almost all of these biases can be avoided using twin registry data. The first study to do this was Kessler's American one of 2000. He found concordance rates for MZ twins of about 32% which suggested that previous results had certainly been distorted by serious bias. Applying biometrical twin modelling this still suggested genetic factors as dominant with heritabilities up to 0.65, although it also conceded a considerable role to environmental variation.[47]

In the same year Bailey published a study based on sampling of nearly 5.000 carefully ascertained twins from an Australian twin registry. This gave an MZ concordance rate of 20% for men and 24% for women. It also confirmed the importance of childhood gender non-conformity in understanding the causes of homosexuality, something which will be returned to in the discussion of social factors. However despite being the largest and most sophisticated study of its type it still did not have enough statistical power to resolve the genetic and environmental components of homosexuality. This is a major flaw of all such concordance studies and is one of their most serious limitations. Twin studies are also compromised by an inherent problem in the concept of heritability. The measure was originally designed to make sense of carefully controlled agricultural experiments. Its extrapolation to human behavioural traits is therefore a highly problematic task. As an illustration of the weakness of this method Jaccard and Dodge have used standard techniques in behavioural genetics to calculate absurd heritabilities, such as 28% for tropical fish lovers and 38% for riding a taxi![48] Therefore to obtain anything more than a broad-brush picture, direct genetics was deemed to be essential.

Genetic Linkage

By the beginning of the nineties the powerful method of DNA linkage analysis had become available for genetic studies of homosexuality. Alleles that are on the same chromosome are more likely to be inherited together and are thus said to be linked. However due to cross over of genetic material in chromosome segregation even alleles on the same chromosome can be separated during meiosis and end up in different cells. The further apart the alleles are on the chromosome the greater is the probability of this occuring. A measure of the probability can be used to calculate physical distance between two genes, since the higher the percentage of offspring that do not share two genetically linked traits the further apart the appropriate genes must be. These distance measurements can then be used to build up a linkage map showing the position of genes or markers relative to each other. These linkage maps are crucial in identifying the genes which cause various diseases or behavioural traits. By calculating the distance of a paticular genetic trait from known markers on a linkage map it is possible to locate and then isolate the gene responsible for this trait.

The first genetic linkage study of homosexuality was that of Hamer published in 1993. Participants for the study were recruited through homophile organisations and their family histories were collected and collated. Pedigree analysis then showed the highest rates of homosexuality to be found in brothers (13.5% compared to a background of 2% in the general

[45] Bailey et al., *Archives of General Psychiatry* (1993)
[46] Bailey et al., *Archives of Sexual Behavior* (1999)
[47] Kessler et al., *American Journal of Psychiatry* (2000)
[48] Bearman and Bruckner, *ISERP* (2001/2)

population), maternal uncles and the sons of maternal aunts. The higher incidence rate of homosexuality in the maternal line suggested to the researchers that homosexuality might be associated with a gene on the X chromosome through a linkage mechanism. Since males receive their X chromosome exclusively from their mother any trait associated with an X-linked gene would be expected to be passed preferentially through the maternal line. For any X-linked genetic marker the chance that brothers share the same allele is 0.5. If brother pairs share the same X-linked alleles more frequently than expected by chance this is an indication that they may share a trait due to a gene on the shared portion of their X chromosome. Out of the forty pairs of homosexual brothers analysed by Hamer thirty three showed a match to the Xq28 chromosomal region (an excess of thirteen over the expected). For this group of people the correlation between genetic markers in the Xq28 region and sexual orientation was found to have a statistical confidence level of more than 99% . In the parlance of behavioural genetics the linkage had a multipoint lod (logarithm of odds) score of 4.0 meaning that likelihood of observing a positive result if no linkage was present would be only 1 in 10,000.

While Hamer's study was trumpeted by the worldwide media as conclusive evidence for the existence of a 'gay gene', the scientific community was in general far more cautious. Hamer himself had conceded that proof for the involvement of genes in a behavioural trait must consist of chromosomal mapping of the loci and isolation of the relevant DNA sequences – something his study had not even attempted.[49] John Maddox writing for *Nature* pointed to the 'formidable list of caveats' which must be added to Hamer's results. In particular he pointed out that until further research had been carried out it was still impossible to out separate genetic and environmental factors. For example he proposed that Hamer's X-linked gene could hypothetically be involved in discerning the 'over-loving' behaviour of the mother. If so the 'determinants would remain those of nurture rather than nature'. More importantly Maddox remarked on the unknown frequency of the X-linked gene within the general population, suggesting that a higher incidence than Hamer's estimate of 2% would considerably weaken the linkage result.[50] Also reviewing for *Nature* Mary-Claire King drew attention to possible information gaps in the pedigrees that Hamer used. The sexual orientation of subjects' relatives was in general only determined indirectly. Even then this was known to be considerably unreliable and thus left Hamer's results in rather a precarious position.[51] The most sceptical analysis of Hamer's study was that of Baron writing in the *British Medical Journal*. He held that a 'single gene or a particular genetic mechanism is unlikely to explain most of the variance in a phenomenon as complex as sexual orientation'. He also pointed out that in order to prove X-linkage alternative mechanisms of paternal transmission must be ruled out, especially in the sons of affected men. Since homosexual men are far less likely to marry and have children such certainty is, in practice, very elusive.

Hamer's research group followed up his study with another publication in 1995 which confirmed Xq28 linkage to homosexuality in men but not in women. The new study found that 22 out of the 32 pairs of homosexual men analysed shared a marker at Xq28. As before the percentage sharing expected by chance was 50%, so the new result represented an excess of 17% - significant if somewhat weaker than before.[52] Despite this confirmation and that of another group [or two?] a number of subsequent studies were in direct oppostion to these results. One pedigree study of homosexuality carried out by Bailey and others found no evidence for maternal transmission of homosexuality. From analysis of three independent samples the researchers concluded that 'none of the samples showed a significantly greater proportion of maternal than paternal homosexual uncles or homosexual male maternal first cousins'.[53] Another study by Rice and a Canadian group sought to exactly replicate Hamer's original method. Out of 46 pairs of homosexual brothers recruited only 20 were found to share markers at the Xq28 position. Even including effects from two other families with homosexual sibling trios the total sharing was found to be only 55%, hardly different from that expected by chance. This null result suggested to Rice that Xq28 had little role to play in the determination of male homosexuality, although of course it did not rule out the possibility of significant effects from elsewhere in the genome (either X-linked or otherwise). Hamer of course was not willing to give in quite so easily. His immediate response was to point out that Rice's selection criteria for research, not discriminating between maternal and other transmission, had actually hidden the Xq28 contribution. He claimed that if non-maternal transmission was excluded from Rice's results then excess sharing could in fact be seen. However as Rice pointed out in a subsequent rejoinder, to do this is to construct an artificial phenotype for homosexuality which is not borne out empirically. Rice's conclusion, based on all the available evidence, was that an X-linked gene for homosexuality could not exist in the general population with any sizeable frequency and so could not

[49] Hamer, *Science* (1993)
[50] Maddox, *Nature* (1993)
[51] King, *Nature* (1993)
[52] Hu et al., *Nature Genetics* (1995)
[53] Bailey et al., *Behavior Genetics* (1999)

be considered as a universal substrate of homosexuality.[54]

A recent study by Mustanski and others (including Hamer himself) has largely confirmed Rice's view. Published in *Human Genetics* in 2005 this study is also particularly important for being the first genomewide scan of male homosexuality. A sample of 456 individuals from 146 unrelated families (of which 73 families represented entirely new data) was selected on the criterion of exclusive maternal transmission with each individual also having to have at least two gay brothers. While DNA analysis of blood samples from each sample did show three definite peaks (with mlod higher than 1.8) Xq28 was not among them. In fact for the full sample Xq28 only showed an mlod of 0.35. Re-analysis of Hamer's original 73 families using updated marker positions gave an mlod of 1.99 – considerably higher but still way short of Hamer's original result. The authors suggested that the main reason for this was the lower marker resolution that they had to use in order to reduce costs. Although this is plausible the fact that doubling the sample size dramatically reduced the linkage rather seems to confirm Rice's suggestion that Hamer's result was highly population specific and therefore not of general application. It also renders their other linkage data at best provisional and needing to be confirmed by a much larger sample.

The authors proposed a number of genetic mechanisms which could be linked to the three peak areas of 7q36, 8p12 and 10q26:

7q36 – Of the three areas this had the highest mlod of 3.45 and displayed roughly equivalent maternal and paternal transmission. One interesting candidate gene in this area is called VIPR2, this is a responder to VIP which is an important neurotransmitter and neuroendocrine hormone. In mice VIPR2 is essential for the development of the hypothalamic SCN, which in humans has been implicated (inconclusively) in male homosexuality. Another possible candidate gene called Sonic Hedgehog is important in determining brain symmetry of the early embryo which could again be hypothetically linked to homosexuality.

8p12 – This had an mlod of only 1.96 but like 7q36 had roughly equal maternal and paternal transmissions. This region has a number of interesting candidate genes particularly given the purported relationship between pre-natal hormones and homosexuality. One of these called GNRH1 stimulates luteinising and other hormones which are important regulators of gonadal processes. Two other proposed candidates are directly involved in brain processes. STAR is a regulator of the hypothalamic-pituitary region while NRG1 controls the growth and differentiation of neuronal cells.

10q26 – This had the weakest mlod of 1.81 and unlike the other two regions only had a maternal contribution. This region may be linked with an important genetic process called genomic imprinting. This link is mediated by chemical structures called methylated CpG islands which regulate the expression of an imprinted gene over several hundred kilobases. This is potentially significant due to a recent study of Hamer and Bocklandt (see directly below) which suggests a link between sexual orientation and genomic imprinting.

Despite the interest generated by these results and the linked genetic mechanisms they do need to be put in some perspective. Currently none of the three peak areas match the standard Lander and Kruglyak criteria for genomewide significance (an mlod above 4.0). Although 7q36 only just misses this mark the other two regions actually fall below the level required even for suggestive linkage.[55] This means of course that the genetic mechanisms proposed are completely hypothetical. The only convincing evidence for the existence of such mechanisms would be in specific candidate gene linkage studies. As mentioned above [page 7] similar studies have already been carried out for the androgen receptor gene and for the aromatase gene both yielding null results. This suggests we should be highly cautious in handling the currently available genetic evidence: results remain very tentative.

New Horizons in Genetic Studies

A number of recent studies have sought to move away from linkage statistics to try and elucidate plausible genetic inheritance mechanisms for homosexuality. One particularly novel approach has been the 'beyond hormones' theory of Bocklandt and Hamer. The authors' desire to move beyond hormones is not a denial of the role that androgens play in sexual differentiation and development of the brain but an attempt to elucidate genetic factors upstream of this process. For this reason they suggest that brain specific transcription factors are likely to be good candidate genes for sexual orientation. In the same way as *fruitless* regulates the male courtship circuit in *Drosophila*, human transcription factors might regulate an analogous circuit for sexual attraction or orientation in our brain. One possible way to narrow down the search for such transcription factors is to consider genomic imprinting. Alluded to above this is a phenomenon where a small subset of genes in the genome become expressed according to their parent of origin. Errors in

[54] Rice et al., *Science* (1999)

[55] Mustanski et al., *Human Genetics* (2005)

the imprinting of genes are known to lead to genetic diseases such as Prader-Willi and Angelman syndrome. Although no examples have yet been discovered it is hypothesised that imprinting of X chromosome genes could lead to sex-specific expression. Since males only receive the X chromosome from their mothers they can only inherit a maternally imprinted Xm, whereas females, who receive X chromosomes from both parents, can inherit a maternally imprinted Xm or a paternally imprinted Xp. Due to X-inactivation (this is a process where female cells randomly inactivate one of their X chromosomes in dosage compensation for male cells which only have one X) a gene expressed only on Xm will be expressed in males twice as much as females. An Xm gene could be a 'masculinising' gene making one attracted to females, whereas an Xp gene (exclusive to females) could be a 'feminising' gene making one attracted to males (these 'masculinising' and 'feminising' genes would be brain transcription factors) . Incorrect imprinting of these genes could influence a son's sexual orientation by giving unusual expression of the 'feminising' gene or lack of the 'masculinising' gene or both to compound the effect. Such imprinting errors could be either inherited, leading to familial patterns of homosexuality, or occur as a one-off. The authors predicted that errors in genomic printing could lead to increased skewing of X-chromosome inactivation in the mothers of gay men.[56]

A recent study by Bocklandt is the first to empirically test the link between X-chromosome inactivation and homosexuality. The X-inactivation ratio at two loci (including the androgen receptor locus) was measured in 40 previously reported and 57 newly recruited mothers with at least one gay son and compared to a suitable control population. Bocklandt found that only 4% of controls displayed extreme skewing of inactivation ratios compared to 13% of the mothers of gay men. In addition among those mothers with at least two gay sons skewing was at 23%. Both the total number of sons and the number of heterosexual sons was observed to have no effect on the skewing. On the basis of these results Bocklandt hypothesises that one central neuronal pathway is responsible for establishing sexual attraction to males or females and usually to the opposite sex. In some men differences in maternal genomic imprinting (perhaps caused by Mustanski's 10q26) combined with the effect of other genes leads to alteration in this pathway and establishes same-sex attraction. However while ingenious this hypothesis is founded entirely on circumstantial evidence. There is no proof even that the skewing effect observed was caused by genomic imprinting. As Bocklandt admits, any of a number of different known factors could have the same effect.[57] Even if imprinting errors were the cause of the skewing observed that does not exclude (indeed has nothing to say about) the possibility that homosexual orientation is mediated through other intervening factors. The fact that over three-quarters of the mothers of even two gay sons did not show this skewing lends support to this idea.

Another recent study by Camperio-Ciani and his Italian group also focuses on maternal genetics. However unlike Hamer and Bocklandt they are much more willing to consider global factors which might interact with the population genetics. In particular their study attempts to answer the Darwinian paradox of why a disadvantageous reproductive strategy like homosexuality should still be present in today's population and not instead have been eliminated long ago by natural selection. Using targeted sampling methods the authors obtained family data from 98 homosexual and 100 heterosexual men. Applying weighting methods to control for any biases they found that in addition to the effects of the FBO homosexuals also differed from heterosexuals in having more fecund maternal (but not paternal) relatives. This difference was particularly seen in fecundities of the mothers and maternal aunts of homosexuals and heterosexuals. These results suggest that genetic factors transmitted in the maternal line have the effect of increasing fecundity in female offspring as well as probability of homosexuality in male offspring. Not only does this solve the Darwinian paradox, by suggesting that there may be reproductive advantages involved in sustaining homosexuality in a population, but it also constrains the possible genetic mechanisms involved. On the basis of obtained statistics an X-linked allele beneficial to female fecundity would only be slightly detrimental to male fecundity. This predicts that such an allele would be very common whereas homosexuality is of course uncommon. This implies the necessity of a polygenic mechanism with one or more X-linked alleles. The fact that over 79% of the variance in sexual orientation is not accounted for by excess of maternal homosexual kin or number of older brothers suggests the importance of social factors and individual experience as a determinant of orientation. The authors even suggest the possibility that higher incidence of homosexuality in the maternal line could result from culturally rather than genetically inherited traits. Thus acknowledging that in a 'maternal' society such as Northern Italy the mother and her family background is one of the most important sources of many of a child's behavioural and attitudinal traits.[58]

[56] Hamer and Bocklandt, *Journal of Endocrinological Investigation* (2003)

[57] Bocklandt, *Human Genetics* (2006)

[58] Camperio-Ciani, *Proceedings of the Royal Society: Biological* (2004)

Psychology of Orientation and Re-Orientation

Psychosocial Factors

From the time of Freud up to about the 70s attributing homosexuality to pyschosocial factors such as distortions in the father-son relationship or lack of affirmation in masculine identity was widely accepted and acceptable. However with the onset of political lobbying as well as the rise of genetic and hormonal theories of homosexuality such theories became widely discredited. It is only in recent years, with the comparative failure of genetic and hormonal paradigms, that there has been something of a renaissance in these studies. In general people are now more aware that biological factors, while certainly important, do not generally operate in isolation from social and psychological factors.[59] However the converse is also true and it is noteworthy that some of the psychological theories of homosexuality have given considerable room to biological factors.

One important recent study is that of Bearman and Bruckner published in 2001/2 by Columbia University. This represents an ambitious attempt to discriminate between various social, genetic, evolutionary and hormonal theories of homosexuality by using opposite sex twins as a testing ground. In particular the authors realised that opposite sex (OS) twins could provide a powerful test of both hormonal influence (due to potential transfer of excess hormones in utero) and childhood socialisation theories, as well as of different patterns of familial inheritance. The study itself employed a large sample of 5,552 adolescent (7th to 12th grade) OS twin pairs drawn from a nationally representative US data source. The results indicated that male but not female OS twins disproportionately reported same-sex attraction. In fact male OS twins were twice as likely as their peers to report such attractions (16.8% compared to a background of 8.6%). Even more interestingly male OS twins without older brothers reported a high same-sex attraction of 18.7% compared to only 8.8% for those with older brothers (indistinguishable from the general background). For female OS twins no comparable effect of older sisters was observed. Their large sample size also allowed Bearman and Bruckner to obtain reliable twin concordance data for same-sex attractions. Interestingly they found very little difference between MZ, DZ or full-siblings with all concordances at around 6 or 7%. Finally the authors also carried out statistical analyses of their whole sample and found no confirmation of any significant genetic, evolutionary or hormonal patterns. When all of this evidence is combined this provides strong evidence for a link between a specific childhood socialisation pattern and same-sex attraction. While not entirely excluding genetic influence the study does relativise the importance of such influence compared to the social factors which condition it.[60]

Bearman and Bruckner's study also provides further confirmation of the hypothesised link between homosexuality and childhood gender nonconformity (CGN), a type of psychological disorder characterised by children showing behaviours, attitudes and personality traits of the opposite sex from their own. The most comprehensive examination of this link remains a review article of Bailey and Zucker's published in 1995. In this they examined both prospective and retrospective studies and considered their associated advantages and disadvantages:

Prospective – These avoid the main pitfalls of retrospective studies. However they obviously more difficult to arrange and more time consuming. They also often only focus on 'at risk' children. These may have psychological disorders and thus be at the extreme end of the homosexual spectrum. One study published by Green in 1987 sampled 66 feminine boys and 56 controls at childhood and adulthood. This found that around 75% of the feminine boys became homosexual or bisexual compared to only about 4% of the controls. A series of follow-up studies summarised by Zucker in 1990 found that 63% of feminine boys became homosexual in adulthood.

Retrospective – While these are easier to undertake than prospective studies and many more have been done their main pitfall is that of biased recall. Some have even gone as far as to suggest that societal stereotypes may cause homosexuals to 'misremember' their childhood, but there is little actual evidence for this. Combining 41 of these studies Bailey found large effect sizes for both men and women. His distributions showed that 81% of lesbians exceeded the heterosexual median (of sex-typed behaviour) whereas only 12% of heterosexual women exceeded the lesbian median. Similarly 89% of gay men exceeded the heterosexual median while only 2% of heterosexual men crossed the homosexual median. The larger effect size for men meant that on average around 51% of cross-gender boys will become homosexual compared to only 6% of cross-gender girls.

Bailey and Zucker suggested two possible psychosocial explanations for their results (apart from pre-natal hormones). The first was a suggestion that sexual

[59] Baldwin, *Human Fertility* (2004)

[60] Bearman and Bruckner, *ISERP* (2001)

orientation could be rooted in the dynamics of the parent-child relationship. The second was in child socialisation. Although the authors suggested that this might only account for within-sex differences in orientation, the evidence from Bearman and Bruckner is that this could be more significant than we have hitherto realised.[61]

Another suggestion of Bailey's is that his most recent Australian twin study, which as we have seen points to familial factors influencing both CGN and sexul orientation, might lend some confirmation to the 'exotic becomes erotic' psychosocial theory of homosexuality.[62] This theory was originally advanced by Bem in 1996 to account for both opposite-sex and same-sex desire and also to account for sex differences in orientation. In particular it sought to suggest a plausible developmental pathway from biological markers to sexual orientation, something which Bem notes is crucially lacking in all current biological or genetic theories. The central proposition of this theory is that individuals become erotically attracted to a class of individuals from whom they felt different during childhood. It is therefore an attempt to integrate the available biological, psychological and social evidence in a meaningful way. Thus Bem suggests that genes and pre-natal hormones code for certain childhood temperaments rather than sexual orientation. These temperaments predispose a child to enjoy some activities more than others and lead to gender conforming or nonconforming behaviour. Reinforcement of these temperaments and tendencies leads CGN children to an entrenched feeling of being different or 'exotic' from their own sex, whereas normal children will naturally feel different from the opposite sex. Bem then suggests that feeling exotic leads to enhanced physiological arousal (usually boys have contempt for girls and girls are timid with boys) which in puberty becomes eroticised. For CGN children physiological arousal is around their own sex and thus sexual arousal will be manifested as homosexual desire.

Bem's theory rests on three central planks: childhood temperaments mediating between biological factors and CGN, strong feelings of exoticism or difference and the mechanism by which exotic becomes erotic. With regard to the first plank Bem points out that such mediating temperaments should be plausibly linked to childhood activities that define gender conformity, should be normally sex differentiated and on the basis of the evidence should be signficantly heritable. This suggests two possible candidates: aggression and activity level. It is interesting that available studies do show significant differences between homosexuals and heterosexuals in these areas. We have already seen how Bailey and Zucker's study (which Bem cites) confirms very strong feelings of homosexual exoticism (from same-sex peers). Bem particularly draws our attention to a study of Bell in 1981 which found that around 70% of gay men and women reported feeling different from same-sex peers during childhood. Another similar study including adolescents as well as adults found that a staggering 88% of homosexual males reported feeling different in childhood. Finally for the third and crucial stage there is good statistical evidence that CGN mediates directly between biology and sexual orientation. Using genetic path analyses (of Bailey and Martin's twin data) Bem found a significant pathway between both genotype and CGN and CGN and sexual orientation but *no* remaining direct link between genotype and sexual orientation. However while this confirms a general picture it has little to say about the detailed mechanism of such a process.[63]

This provides good evidence for the importance of childhood experience in the development of homosexuality.

Another important psychosocial theory of homosexuality is that of 'defensive detachment'. Proposed by Nicolosi, a Catholic and key advocate of re-orientation therapy, this is both a variant and refinement of the father-son theory of classical psychology. Nicolosi suggests that homosexuality, at least in the case of his patients, is essentially a reparative drive in which a person attempts to satisfy unmet same-sex needs (affection and approval) and to correct gender identity deficits. In particular he sees homosexual behaviour as an attempt to show assertion and relieve shame. Importantly Nicolosi bases his ideas on his experiences with the fathers of homosexual men as well as the men themselves. He points out that, while in other respects generally kind and loving, fathers of homosexuals he had counselled were all emotionally avoidant. This meant that they showed an inability to relate to other men at a deep emotional level. This insensitivity meant that they were able to easily wound their sons sense of masculinity (perhaps from very early childhood) but not able to easily repair such a wound or counter their son's 'defensive detachment' from them. In the case of the son such detachment from masculine identity leads to a constant yearning for affection and affirmation. In puberty such a yearning then becomes eroticised and transposed into homosexual desire. While seeing a rupture in the father-son relationship as extremely common in the causation of male homosexuality, Nicolosi recognises that it is not necessarily the only significant social factor. Apart from

[61] Bailey and Zucker, *Developmental Psychology* (1995)

[62] Bailey, *Journal of Personality and Social Psychology* (2000)

[63] Bem, *Archives of Sexual Behavior* (2000)

the combination of insensitive father and sensitive son he suggests that hostile older brothers, extremely affectionate or 'feminising' mothers, child abuse, school bullying and the allure of a gay counter-culture can all also play a pivotal role.[64]

In confirmation of Nicolosi's observations it is particularly interesting that a 10-year literature survey carried out by Bradley and Zucker indicates that CGN sons often perceive relationships with their fathers as distant, negative and conflicted.[65] Sadly there is also a considerable body of evidence linking sexual abuse in childhood to later onset of homosexuality. One survey by Bramblett and Darling found that among adult male survivors of such abuse 14% perceived themselves as gay and 32% as bisexual compared to 88% heterosexual and 12% in a non-abused control group.[66] Studies have also shown that homosexuals report a disproportionately high percentage of incestuous sexual relationships during childhood. In one study 35% of homosexual men reported sexual abuse compared to only 5% in a heterosexual control group.[67] Taken together this provides good evidence for the importance of childhood experience in the development of homosexuality as proposed by both Bem and Nicolosi and a number of others.

Other recent evidence comes from a study in Canada of Roman Catholic seminarians reports that 24 respondents who self-identified as homosexual reported a significantly lower mean level of intimacy with their fathers than 130 respondents who self-identified as heterosexual. Interestingly they also did not find any significant differences between the two groups for intimacy with mother or intimidation from father and mother. The authors therefore proposed the 'father-son unit as the basis for analysis of homosexuality'.[68] Although their sample size is too small to justify a firm conclusion, a 2006 Danish study provides very strong evidence for such a conclusion. Using a population sample of over 2 million Danes aged between 18 and 49 the researchers assessed detailed marriage records for men and women marrying a same-sex partner between 1989 and 2001. Denmark is a society noted for its tolerance and even endorsement of homosexual lifestyle, being the first country to legalise gay marriage. The sample size used, which was almost the entire population, also avoids the selection biases which normally plague sexual orientation studies. The results of the study are striking. The researchers found that both men and women from unstable or broken families or with 'unknown fathers' were more likely to marry homosexually. To put some figures on this men whose parents divorced before their 6th birthday were 39% more likely to marry homosexually than their peers while men who ceased cohabiting with both parents before the age of 18 years old were also far more likely (55-76%) to contract such a partnership. Children with older mothers, only-childs and urban-born children were also found to be significantly more likely to marry homosexually suggesting the importance of other factors. From this the authors concluded that their study provided 'population-based, prospective evidence that childhood family experiences are important determinants of heterosexual and homosexual marriage decisions in adulthood'.[69] It hardly needs to be added that this represents research into sexual orientation on an unprecedentedly large scale.

Re-orientation Therapy

Evidence either for or against sexual re-orientation is very important in determining the etiology of homosexuality. If orientation can be shown to be malleable then this suggests that homosexuality is neither ultimately innate nor finally immutable, although it may still be very deep-seated. The question of the efficacy of reparative therapy for homosexuality is a highly emotive one. In particular many in society find the notion of re-orientation insulting or even repugnant. However, to reiterate on a point made in the introduction, it is not the intention of this study to examine the morality or otherwise of any aspect of sexual orientation or re-orientation but only to assess the available scientific evidence on these matters.

The most extensive survey on this topic has been carried out by Throckmorton, an important proponent of re-orientation. Although this may colour his interpretation of some of the evidence, his general claim that far more research was carried out into re-orientation prior to the mid 70s can be proved by no more than a cursory glance at the corpus of available literature. The reason for this dramatic shift is entirely simple, 1973 was the year when the APA declassified homosexuality as a mental disorder thus automatically invalidating the notion of treatment for homosexuality and strongly disincentivising research into this area. It is only recently that this trend has begun to be reversed, although useful studies are still very thin on the ground. Reviewing all studies prior to 1998 Throckmorton found that they reported change rates to exclusive hetereosexuality (although not necessarily from exclusive homosexuality beforehand) between 18 and 44%. In addition rates for any shift in orientation were reported to be much higher than this. Throckmorton also found as a general rule that clients with both prior

[64] Nicolosi: www.narth.com/docs/fathers.html
[65] Bradley and Zucker, *Journal of the American Academy of Child and Adolescent Psychiatry* (1997)
[66] Bramblett and Darling, *Journal of Sex and Marital Therapy* (1997)
[67] Rosser et al. [cf Rosik]
[68] Seutter and Rovers, *Journal of Psychology and Theology* (2004)
[69] Frisch and Hviid, *Archives of Sexual Behavior* (2006)

heterosexual experience and motivation for change were the most amenable to therapy.[70]

Rosik, another proponent of re-orientation therapy, has reported on some of the more recent studies in this area. One of the largest and most significant of these was by Nicolosi, Byrd and Potts. Surveying 882 clients involved in re-orientation therapy they focussed on 318 individuals who had reported exclusive homosexuality before beginning treatment. On finishing such therapy 18% were found to have become exclusively heterosexual and 17% almost entirely so, only 13% remained exclusively or almost exclusively homosexual.[71] Two other studies have specifically examined religiously motivated attempts to change orientation. These showed a behavioural success rate – defined as voluntary abstinence from homosexual contact – of around 60% for men and 70% for women. These studies also pointed out that while success rates were considerable, in general change only occurred a long way into therapy. This may be an explanation of why some studies have seen only uncertain impact of therapy on changing sexual orientation.[72]

Undoubtedly the most important recent study, as well as the most controversial, is Spitzer's published in 2003. For our purposes this is also the most interesting as Spitzer is not only a distinguished scientist but was also the main architect of the 1973 declassification of homosexuality. Before embarking on his research he was therefore highly sceptical of re-orientation therapy. His subsequent volte-face on the issue is thus particularly significant. Spitzer analysed 200 respondents (143 men and 57 women) who reported a change from a homosexual to heterosexual orientation lasting five years or more through therapy. To qualify for research each respondent had to have a sexual orientation score of at least 60 on a 100-point scale (0 heterosexual, 100 homosexual) before therapy began and had to have experienced a change of at least 10 points towards heterosexuality. While only 11% of the men reported 'complete' change, among women the percentage was a significant 37%. In addition there were many examples of change from a predominantly homosexual to a predominantly heterosexual orientation. These enduring results finally convinced Spitzer of the viability of re-orientation therapy.[73]

Upon publication the Spitzer study met with a whole range of different reactions. Unsurprisingly the most bitter criticisms of his results came from his former friends. One of them even cruelly caricatured him as an ageing pantomime horse pathetically seeking the limelight or 'a court jester hoodwinked by a scheming religious right'. On a purely scientific level Spitzer was accused of using an unrepresentative sample of homosexuals. In particular it was contested that he had chosen only religious and married homosexuals who had rejected their own orientation.[74] On a simplistic level this accusation is entirely true: an overwhelming 93% of respondents classed religion as either 'extremely important' or 'very important'in their lives, 19% were mental health professionals or directors of ex-gay ministries and 67% said that homosexuality conflicted with their marriage or potential marriage.[75] However while the sample is unrepresentative of *all* homosexuals, it is in fact highly representative of that group of homosexuals who wish to undergo re-orientation therapy. It makes sense that it is the married, emotionally dissatisfied and religious homosexuals who will want to change and indeed be motivated enough to succeed. Spitzer's study therefore proves that for such as these change *is* possible.

Re-orientation Theory

Re-orientation therapy is founded on the assumption that homosexuality (at least in its particularity) is rooted in developmental experience. For this reason it focusses on treating individual cases of homosexuality rather than subscribing to an overarching theory of causation. The reasoning behind this is that while particular psychosocial factors play a crucial role in the onset of homosexual orientation, the exact interrelation between all of these for any individual is often highly complex, particularly when biological factors such as genes, brains and hormones are also brought into the equation. However for obvious reasons re-orientation theorists do not regard homosexuality as immutable (unless the human will not to change could be considered immutable). Nor do they regard homosexuality as *ultimately* innate, although it would not be impossible for them to conceive of it as in some way *initially* innate in the sense of genetic programming or neural hardwiring – if so they would see re-orientation as some form of non-invasive mental reprogramming or rewiring.

Re-orientation theory therefore says that therapy is a corrective measure for developmental experiences which have shunted sexual orientation in a homosexual direction. Through counselling and other psychological methods the patient comes to term with these experiences and other aspects of their life (including the pain and guilt of homosexual experience) and thus becomes able to change both their behaviour and motivation. For this reason there is here often a considerable overlap between psychology and spirituality. In Christian language re-orientation is seen

[70] Throckmorton, *Journal of Mental Health Counselling*, (1998)
[71] Nicolosi et al., *Psychological Reports* (2000)
[72] Rosik, *Journal of Pastoral Care* (2001)
[73] Spitzer, *Archives of Sexual Behavior* (2003)
[74] Besen, *Journal of Gay and Lesbian Psychotherapy* (2003)
[75] Sptizer, *Archives of Sexual Behavior* (2003)

as a redemptive process of healing encompassing body, soul and spirit. The only evidence available to test such remarkable claims of transformation is in personal testimony and changed lives. Whether one accepts this as true will entirely depend on both belief in God and belief in the sincerity and honesty of the available testimonials. This therefore becomes a question entirely outside the realm of science. If we choose to ask it we may be forced to engage with sexual orientation and homosexuality not just on a scientific or psychological basis but also on moral and spiritual grounds. So far few have chosen to take this challenge seriously.

Conclusion

In this study we have sought to focus on the scientific evidence relating to the causes of homosexuality. The evidence is complex and often admits of various interpretations. This in itself should warn us away from an overly simplistic or reductionist view. Our quest for a scientific understanding of human sexuality may in fact have only just begun. Gender, for example, is a fundamental and foundational part of human experience whether sexual or otherwise. In this light the lack of scientific consensus in this matter is remarkable. The last fifty years have been more dedicated to tearing apart traditional notions of gender than building up our understanding. This is seen in particular with the weakening, even severing, of the link between biological sex and gender. In society as well as in the scientific community both intersexuality and transsexuality are seen as challenging the view of gender as a bipolar feature. Instead people are now more inclined to think of gender as a spectrum or continuum ranging from masculine to feminine. Thus Johnson points to the eroding of the idea that the sex-gender-sexuality triplet can only come as a 'neat package of two readily defined phenotypes' – male and female heterosexual.[76] In addition many have bought the sociological view that gender is merely a human construct into which children are conditioned. However there is at least some evidence that scientists, with regards to both intersexuality and transsexuality, have normalised rare chromosomal, genetic or psychosexual disorders (including things like CGN) and then fed back these *normalised* experiences into their overall gender construct.[77] If this is true then the question of normality may turn out to be the pivotal one in our understanding of human sexuality. How useful science will be in answering this question remains both unclear and disputed.

If our understanding of human gender is so limited then it is likely that our understanding of sexuality will be similarly constrained, particularly because the latter builds so much upon the former. However throughout we have hopefully been able to identify a number of key points:

i) Genetic effects are certainly important in any consideration of human sexuality. Although animal evidence suggests that transcriptional mechanisms can shape complex sexual behaviour it must be borne in mind that such behaviour is often instinctual and almost entirely mediated through pheromones. By contrast available human evidence suggests that genetic influence on homosexuality is likely to involve a number of gene interactions and to be relatively weak. While it is entirely possible that a genetic predisposition to homosexuality exists it flies in the face of the evidence to treat this as an inevitability. It is also now widely recognised that the effect of genes cannot be isolated from the environment they are expressed in.

ii) Hormonal effects on human sexuality also should neither be neglected or negated. However it must be recognised that many current hormonal theories are provisional and often based on animal models. While some hormonal markers do seem to differ between homosexuals and heterosexuals the precise developmental implications of this are often unclear. It is also possible that hormones may only indirectly effect sexual orientation. In particular their effect on the brain is still a matter of considerable debate.

iii) Psychosocial factors have long been neglected but a number of recent studies point to their manifest importance. In particular childhood and adolescent experience seem to be determinative of future orientation. Particularly significant are the high proportion of homosexuals who report a distant father-son relation and a feeling of being 'exotic' and separate from their same-sex peers. In addition, as some have suggested, psychosocial factors may turn out to be at the root of the difference between gay and lesbian orientation. In our society gender nonconforming boys are far more often singled out from the crowd than girls. This singling-out may have the effect of reinforcing their feelings of difference and thus entrenching their orientation. For girls there is not the same degree of singling-out so orientation is likely to be much more fluid and even superficial.

iv) Neural plasticity is also an important factor to take into account. There is at least some scientific evidence that significant orientation shifts are possible for both men and women. However homosexual orientation can also be very deep rooted to the extent that for some people it becomes a seemingly indispensable and defining feature of their identity. Any etiology must recognise both these features.

Weaving these four threads together we can formulate a provisional model for the formation and strengthening of homosexual identity. To start with it seems likely that a biological disposition to homosexuality does exist.

[76] Johnson (2004)

[77] Swaab, *Gynecological Endocrinology* (2004)

Perhaps, as Bem suggests, this is mediated through childhood temperaments such as lack of aggression and heightened sensitivity. However the triggering or activation of such a disposition seems to be determined by developmental and familial experiences, particularly rooted in the parent-child relationship. Reinforcement of such experiences may then lead to gender detachment coupled with a longing for affirmation. In puberty it is easy to see how a yearning for same-sex affirmation could then become eroticised and give rise to same-sex desire. Subsequent sexual experience and initiation into the gay subculture will only lead to an entrenching of homosexual identity. From here the path divides further. Some will remain homosexual to the day of their death never dreaming or even desiring change. Others however will choose to retrace all of their steps, however painful and gradual a process this may be, and hope to eventually emerge on a different path. The fact that it is largely motivation that separates these two groups is a salutary reminder that choice should never be left out of the equation of human sexuality.

Simon Burton studied Natural Sciences at Magdalene College, Cambridge, followed by a Diploma and MPhil in Theology and Religious Studies focusing on 17th century Church History and Theology, also at Magdalene College.

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