

ON THE BIOLOGICAL BASIS OF HUMAN SEXUAL ORIENTATION

INTRODUCTION

Public opinion remains deeply divided as to how far homosexual behaviours - resulting from same-sex attraction (SSA) - can be regarded as normal traits within a wide spectrum of human and animal sexual orientations. Both within the various religious traditions and beyond, there are many people who regard SSA as an aberrant and sinful tendency that is chosen (even unconsciously) by homosexuals. In the Anglican tradition, this issue has most recently been called into question by the deliberations of Lambeth 1998, and the subsequent actions of the Episcopal Church USA (ECUSA) in consecrating an openly gay bishop and the Diocese of New Westminster (British Columbia) in adopting a liturgy that sanctions same-sex unions by the church. These actions by two of the constituents of the consortium have resulted in a major report (the Windsor Report) on the impact these decisions have had on the rest of the Anglican Communion. These actions were deemed to be outside the pale of Anglican ethos, and the Report suggests ways in which the Communion can deal with disparate actions by the autonomous collage of churches that comprise the Anglican Communion. *Inter alia*, the Windsor Report suggests a major consolidation of power within the central axis of the communion that would effectively deal with actions deemed inappropriate by participating churches that comprise the Communion. This is a major change in direction for the Anglican Communion that had previously been seen as an autonomous confederation without centralized power. This proposal may result in the fragmentation of the tradition over issues of biblical authority and how the Bible is to be interpreted. Already, the church in Nigeria has begun to distance itself from the Anglican Communion over issues of human sexuality and biblical authority. If science could provide clear evidence to support one or more biological mechanisms underlying homosexuality - whether grounded in terms of genetics, hormonal influences, or neuroanatomy (none of which can be freely chosen) - then any faith basis for homophobic attitudes and prejudice should require much closer scrutiny. This is perhaps a naïve hope, since it cannot address issues of scriptural interpretation, and in any case science is unlikely ever to meet the standards of unequivocal proof that some require. Among Anglican provinces and dioceses that have grappled with the homosexuality question, New Westminster deliberately eschewed commissioning a report on the associated biological issues because the science was seen as inconclusive. In contrast, the diocese of Massachusetts (USA) has recently published a helpful ecumenical report on the subject (1). Before reviewing the current scientific consensus and controversies surrounding human homosexuality, two related issues should be dealt with very briefly:

- The first concerns the occurrence of homosexual behaviour among animals. Contrary to a popular but mistaken impression that such behaviour is purely human, it is in fact widespread among many different species (2), and sometimes occurs in mixed-sex groups where members of the opposite sex are available (2, 3). Whether this tendency depends on particular local experiences or arises independently, the main point is that homosexuality cannot be described as unnatural, from the perspective of behaviour patterns seen in the natural world. It therefore cannot be regarded as a peculiarly human form of sinful behaviour.
- A wider but pertinent question relates to the purpose or function of sexual behaviours, which (for obvious reasons) are intuitively regarded as linked solely to reproduction. A relevant example here is the pygmy chimpanzee or bonobo (3), where both homosexual and heterosexual acts seem to serve a variety of social functions, including conflict resolution and obtaining food. Therefore, homosexuality and SSA in humans should not be regarded as abnormal merely because they have no reproductive potential. For example, not all heterosexual couples choose to have children, and sexual intercourse is commonly practiced without any intent to procreate; indeed, this may be manifestly impossible (e.g. in cases of

complete infertility). These relational aspects are integral to an Anglican understanding of sexuality (cf. the marriage liturgy).

While the studies detailed in the following report deal principally with male homosexuality, female SSA is also of equal import. However, much less is known about the scientific basis of female SSA and lesbianism - except that it clearly exists as one expression in the spectrum of human sexuality. There is no *a priori* reason to expect either identical or complementary underlying mechanisms, although the latter are more often proposed in the literature. **Twin studies - the paradigm of genetic identity.** It is common to want to compare the relative influence of nature (genetics) and nurture (environment) in human growth and development. People commonly refer to this polarity as nature **versus** nurture, which is a misnomer because both contribute to the ultimate development of the individual (4). Monozygotic (MZ) or identical twins share exactly the same (100%) genetic make-up, because they arise through the splitting of a single fertilised egg (zygote) during early embryonic development. It is worth noting that MZ twins share a common environment within the womb, and are often (but not always) raised together. However, there may also be subtle differences in the uterine micro-environment, which can have lasting effects on the postnatal development even of MZ twins. Dizygotic (DZ) twins, on the other hand, arise from two separate fertilisation events and therefore share only 50% of their genes - just like any other two siblings with the same biological parents. However, DZ twins also share environmental influences within the womb (and postnatally) to a much greater extent than do ordinary siblings. Finally, adopted siblings are genetically unrelated to other members of the family, but still share the same family environment along with natural children of the adoptive parents. At least in theory, twin and sibling studies could begin to differentiate between genetic and environmental influences on homosexuality. An important issue in this regard is the prevalence of homosexuality within the population as a whole, generally put at between 1 and 5% in Western societies, but ranging higher depending on the exact criteria used to define homosexuality or to select the study population (some sampling methods can undoubtedly bias the results). If SSA were wholly genetic, then MZ twins should always show identical sexual orientations (i.e. there would be 100% concordance for this trait), while concordance for DZ twins and ordinary siblings should be no more than 50% (depending on how many genes are needed for the SSA trait to develop). If the womb environment is important (e.g. via hormonal influences during pregnancy), then DZ twins should show greater concordance than ordinary siblings. Conversely, if genes and the womb environment play **no** role whatsoever in SSA, then neither MZ nor DZ twins should show higher rates of concordance than ordinary or adopted siblings. Several studies of this type have been reported in the literature, and these present a fairly consistent trend, even though the prevalence of SSA in various sub-groups varies according to the criteria used to select the study population. In a study requesting homosexual males to volunteer information about twins and siblings (5), concordance rates for SSA were found to be 52% for MZ twins, 22% for DZ twins, 11% for adoptive siblings, yet only 9% for normal siblings (lower than expected, though other studies do suggest a higher figure). Because this study group was self-selected through homophile publications, these figures may err on the high side. In a large group of non-selected twins, where SSA was assessed through questionnaires (6), concordance for SSA was 31% for MZ twins, 13% for DZ twins of the same sex, but only 8% for all DZ twins. However, the statistical power of this later study was poor due to the low prevalence of SSA within this group (3.1% for males; 2.5% for females). Taken at face value, these figures suggest that genetic factors **do** influence homosexuality, since concordance for SSA among MZ twins is about 10-fold more common (30%) than among unrelated people in the population (3%). Concordance for SSA among DZ twins is much less common than among MZ twins, implying that shared genes affect SSA over and above any effects of a shared womb environment or family upbringing. But this still falls far short of the 100% concordance expected among MZ twins for a wholly genetic trait, so other factors (epigenetic or environmental influences; see later) may also play an important role. At least some of these influences must be experienced within the womb, since concordance for SSA seems to be substantially more common among DZ twins than among

ordinary siblings, even though both pair types have exactly the same probability (50%) of sharing any given gene. However, neither genetic nor womb-environmental factors can wholly account for the patterns of SSA prevalence observed in these studies (which are largely confined to male SSA) - allowing scope for additional later-acting environmental influences including choice.

ARE THERE "GENES" FOR HOMOSEXUALITY?

Scientifically, this title is totally misleading - but unfortunately it has become a convenient label for media use. To date, there is no known single gene that is responsible for any complicated behavioural trait, including SSA. Even without definitive evidence from twin studies (above), one might expect SSA to involve multiple genes and complex interactions between them. Thus far, the task has been to identify genetic variants that seem to be significantly more prevalent among male homosexuals than in the general population. There are accepted criteria for establishing the significance of such associations, which have been widely used to track down human gene-variants associated with particular diseases (some purely genetic in origin, but many involving both genetic and environmental factors). This approach depends on the presence of different polymorphic microsatellite markers (PMM), which are highly repetitive DNA sequences comprising relatively short repeats. The **number** of repeats present varies greatly between individuals, and (importantly) these features are inherited from parent to offspring. PMM repeats are distributed at irregular intervals along the human chromosomes under study. A gene search is more likely to succeed if several such markers are positioned nearby the gene(s) of interest, but less so if these markers are sparse or absent across the region where a putative gene actually resides. Note that this kind of search identifies only a specific pattern of markers that correlates with the trait under study - i.e. most affected individuals are associated with this pattern, whereas most unaffected individuals are not. This highlights a particular region of DNA that may possibly contain a variant gene associated with the trait. It is then necessary to sequence that DNA region to identify any unusual gene variants present within it that might help to explain the biological basis of the trait. Even then, it needs to be established that any such variant gene is largely confined to individuals exhibiting the trait and is not widespread elsewhere in the general population. In 1993, enormous media interest in genes purportedly linked to SSA was sparked by a paper from Dean Hamer's group (7). Their work was based on the observation that, in certain families with homosexual sons, the SSA trait appears to be inherited from the mother (with identifiable homosexual cousins or uncles only on the mother's side). The forty study families selected all showed this pattern and included at least two homosexual brothers. The genetic search was confined to the X chromosome, because sons (XY) always inherit their X chromosome from their XX mothers. Using densely spaced PMMs, a common variant region of this chromosome (Xq28) was identified in around 64% of the pairs of brothers tested. It was suggested that this Xq28 region could contain a variant gene (or allele) that might contribute to the development of SSA, at least for this narrowly defined subtype of male homosexuality showing maternal influence. Some later studies have failed to confirm any association between SSA and this Xq28 variant, even in families where two brothers are both homosexual (8), though the reasons for this disparity remain unclear. Very recently, the same type of analysis has been applied across the entire set of human chromosomes (genome), again focussing on families with two or more homosexual brothers (9). None of the variant regions identified in this study shows the degree of tight association needed to reach statistical significance in such a genome-wide study, but several fell only slightly short of this. Two such variant regions (7q36 and 8p12) were derived equally from fathers or mothers, while another at 10q26 was inherited only from the mother. Curiously, no significant association was recorded for the Xq28 site previously identified by Hamer's group (7). Since Hamer is one of the authors of this new study, and since his original sample was included within the larger study group, his 1993 sample was reanalysed using both the new (genome-wide) and old (X-chromosome only) sets of markers. The association for Xq28 was significant using the denser PMM set for the X-chromosome, but not using the sparser genome-wide set (9). This

suggests that Xq28 may still contain important genes contributing to SSA, but that mapping with more closely spaced markers will be needed to confirm its significance. This argument could equally apply to the other variant regions identified; more detailed mapping studies will either confirm them as being significantly associated with male SSA, or else refute any such link - perhaps because of statistical flukes in the initial study (9). It is possible that other variant regions will also show significant associations with male SSA. Overall, it seems likely that multiple DNA regions contribute to male SSA, and that varying combinations of these might influence SSA in different individuals. It seems clear that there is not **one** "gay gene", but probably many genes that contribute towards this behaviour - and these may not necessarily be the same in male and female homosexuals. The candidate regions identified so far contain an intriguing array of genes (9):- 7q36 contains a key patterning gene (*SHH*) and a hormone receptor gene (*VIPR2*) essential for development of the hypothalamus, a brain area that acts to co-ordinate the hormonal control of sexual development and behaviour; 8p12 also contains two interesting genes affecting sex-hormone production (*GNRH1*, *STAR*). This could, of course, be merely coincidental, and in any case, it remains to be established that the variant regions found commonly in SSA males do in fact contain unusual versions of these particular genes or that they play any role in SSA. Much work still remains to be done, and the current data remain ambiguous.

IS THERE AN EVOLUTIONARY RATIONALE FOR SSA?

If variant genes (known as alleles) persist at significant frequencies in the population, then Darwinian evolution would suggest that they must confer some kind of selective advantage. For instance, the sickle-cell variant of the haemoglobin b-chain occurs at high frequency in many Afro-Caribbean populations, despite the fact that people with two copies of the mutant gene (bS, bS) suffer from severe and often fatal sickle-cell anaemia. People with one normal and one mutant copy of this gene (bA, bS) do not suffer from anaemia, but this sickle-cell trait confers significant resistance to infection by the malaria parasite, in comparison to people with two normal copies of this gene (bA, bA). Thus, the prevalence of malaria in tropical Africa provides a **selective pressure** to maintain the mutant bS allele at higher frequency in those populations. A number of other genes have also been identified that protect against malaria and provide benefits for other conditions as well (10). However, this case is a simple one - involving just one mutant gene and one environmental factor (malaria). For homosexuality, an individual's genetic constitution (involving multiple genes) may provide only a **predisposition** to SSA, which also requires the participation of additional environmental factors before homosexual behaviour is actually expressed. Not everyone who is genetically predisposed to SSA will necessarily become homosexual, because essential environmental triggers may be missing. Conversely, a full array of environmental factors will not result in homosexuality amongst those who do not have the requisite genetic predisposition. This general kind of situation is familiar from many common conditions such as diabetes or heart disease - both of which involve complex interactions between multiple genes and environmental factors (diet, lifestyle, even viral infections).

It is not immediately apparent what selective pressures might help to maintain gene-variants for SSA in human populations. A variety of evolutionary rationales have been suggested, and the following summarises only one of these, albeit one that is unusually fully argued (11). The starting hypothesis is that both males and females display overlapping ranges of gender-related stereotypic behaviours; males are generally more aggressive, females more nurturing, and so on. SSA is suggested to arise at the female end of the male spectrum ("effeminate" or feminised) and at the male end of the female spectrum ("tom-boy" or masculinised). The fact that these stereotypes are demeaning caricatures (and clearly wrong in many individual cases) is essentially irrelevant to the argument here. If we accept both generalisations as containing some modicum of reality, then we are seeking an evolutionary rationale for the presence of both "feminised" males and "masculinised" females within the human population. It is suggested that homosexual men with a strongly developed

capacity for nurturing or empathy may promote social stability, for instance by helping to raise children or resolve disputes - as compared to more aggressively heterosexual men, who might instead provoke disputes or abandon existing families for new partners. Among empathetic and nurturing men, some (though clearly not all) are likely to manifest homosexuality - a likelihood which churches should recognise among male clergy, who are oftentimes selected on the basis of these selfsame nurturing personality traits. Analogously, more aggressive and independent women may be at an advantage when conflict or disaster faces a community. In both cases, such personality types will only prove advantageous within larger social groups - such as extended families or clans. In evolutionary terms, it may pay to have a broad spread of both "male" and "female" traits present in both sexes (11). One curious but consistent observation is that birth order strongly influences the incidence of male SSA, which is more prevalent among later-born than first-born brothers. This trend is influenced by the number of older brothers, but not sisters (12). The probability of a subsequent son developing SSA increases by roughly one third for each older brother (but note that the starting probability here is only 3%!). Yet how could birth order possibly influence the complex array of genetic and environmental factors associated with SSA (as discussed above)?

One speculative suggestion invokes unknown factors within the womb environment that would act selectively on developing male embryos, as suggested by the DZ twin studies cited above. This could involve male cell-surface proteins (HY antigens) encoded by genes on the Y chromosome. Because such proteins are foreign to the mother (who has 2 X chromosomes but no Y), the HY antigens might provoke an immune response in the mother (11). This would become stronger with each successive pregnancy involving a male foetus (on the same principle as booster shots for immunisation). Maternal antibodies recognising the HY antigens could act to suppress or stimulate cellular processes (possibly involving sex hormones) in the male embryo, which could in turn promote SSA. This would only become apparent if an appropriate combination of genetic variants were already present in the foetus, therefore not all younger brothers will necessarily show the SSA trait. The reality may turn out to be far more complicated even than this. Like most Darwinian explanations, this model can be dismissed as a *post hoc* rationalisation and over-simplification, but at the least it could suggest evolutionary reasons why human diversity is to be prized - over and above the aesthetic and theological value that we place on such diversity.

OTHER CORRELATES OF SAA:

(i) Neuroanatomy. Apart from the SSA-associated genetic variants (candidate gene alleles) discussed above, there are several neuroanatomical reports describing the relative sizes of specific brain regions (nuclei) in heterosexual males and females, as well as in homosexuals (usually men). In general, male homosexuals display characteristics associated with heterosexual females rather than those of heterosexual males (see 13, but note caveats in 14). In part, these differences help to underpin the "feminisation" hypothesis discussed above in relation to homosexual men.

Unfortunately, neuroanatomy is notoriously subjective and different laboratories have either failed to confirm these differences between the sexes, or else have ascribed them to different brain structures! Overall, there is no clear consensus as to the validity of these differences (14). But even if the brains of males and females cannot be differentiated by any consistent structural criteria, there are clearly **functional** differences between them, at least in terms of sexual attractiveness among the heterosexual majority. Heterosexual males are attracted to heterosexual females and *vice versa*, whereas homosexuals are attracted to people of the same gender. (ii) Pheromones. Like heterosexual attraction, SSA relies in large part on visual stimuli, but scent may also play a crucial role in both. Extrapolating from a great many studies in animals (from insects to vertebrates), specific sexual attractants known as **pheromones** are likely to be important, even in humans who often go to great lengths to cover up their natural odours! It should be acknowledged at the outset that there are no definitive human pheromones known as yet, but several candidates await further confirmation. Two compounds of particular interest are a derivative of the male sex hormone

testosterone (AND), and a steroid related to oestrogen (EST). In a recent study using advanced functional imaging techniques to study brain function, specific regions of the brain associated with sexual arousal were activated by AND in heterosexual females (but not in males), and a similar response was evoked by EST in heterosexual males (15). Interestingly, homosexual males did not respond in this way to EST, but did respond to AND (the converse response of homosexual females to EST was not tested in this study). Thus homosexual males showed evidence of arousal by male but not female candidate pheromones. All three groups showed general olfactory responses (in other brain areas) to both EST and AND, confirming that both chemicals are perceived as odorants. Non-pheromone odours also activated these general olfactory responses, but had no effect on the brain areas associated with sexual arousal. This study may be more relevant to the identification of human pheromones than to the causes of SSA. We already know that homosexual males are attracted to other men but not to women, so if AND and EST are indeed genuine human pheromones, then any pattern of results other than that reported would have been most surprising. However, this study opens up several interesting questions, not least the likely response pattern among homosexual females. When do these differential responses to AND or EST first become established during development? Are they fixed immutably, or can they be changed? Do bisexual people respond to both pheromones? It would doubtless be unethical to test putative sex pheromones on infants or pre-pubertal children, so the answers to some of these questions may have to be extrapolated from animal data. One central question remains unanswered: are male homosexuals born with an innate responsiveness to AND rather than EST, or is this a learned response?

(iii) Hormones in foetal development. An influential paper dating back to 1987 (16) suggests that abnormal exposure to male sex hormones (androgens such as testosterone) during foetal development can affect a person's later sexual orientation. Broadly speaking, male foetuses exposed to abnormally low levels of androgen in the womb may develop a predisposition towards homosexuality, and the same might be true for female foetuses exposed to abnormally high androgen levels. This model remains controversial, with evidence both for and against (reviewed at length in 17), but it clearly provides a rationale for the "feminised male"/"masculinised female" interpretation of homosexuality discussed above (11). Perhaps the best support comes from studies of two rare genetic diseases (reviewed in 17). In congenital adrenal hyperplasia (CAH), androgen is produced instead of cortisol, resulting in masculinisation of female babies; this can be corrected by surgery and later controlled by postnatal cortisol treatment. Despite being genetically and anatomically female, such CAH patients display a ~5-fold higher incidence of SSA and lesbian behaviour as compared to the general population. Conversely, androgen insensitivity syndrome (AIS) – an inherited inability to respond to androgens – has precisely the opposite effect on female foetuses, since SSA and lesbian behaviour are very much rarer among these patients. Since these two conditions represent opposite ends of the spectrum of androgen influence during foetal development (high in CAH, low in AIS), this suggests that prenatal androgen exposure strongly influences female SSA. No comparably strong evidence links male SSA with sex hormones.

(iv) Gene expression in early development. Correlates of human sexuality are much wider than just the contributions of the sex chromosomes (X and Y) and hormone exposure of the foetus during development. Scientists studying mice now realize that gene expression necessary for sexual differentiation of the brain begins much earlier in foetal development than had been previously thought. They have demonstrated that expression of certain genes occurs at a time **before** there is any significant contribution of endogenous sex hormones (18). While much of this research is aimed at understanding gender ambiguity (19), it is also likely to contribute to understanding all types of sexual behaviour in humans.

(v) Epigenetics. One interesting speculation is the possible role of genetic imprinting. This includes selective methylation of regions of DNA, or acetylation of the associated histone proteins, both of which act to repress gene expression. Repressed genes can also be activated through demethylation of DNA, or deacetylation of histones. In this context, it is worth recalling that studies of MZ twins show only 31-52% concordance for homosexuality, even though they share 100% of their genes. Women have two X chromosomes, one of which is normally inactivated by methylation to prevent over-expression of potentially deleterious

genes, hence the X chromosome is a prime candidate for differential methylation. It is possible that the expression or repression of a gene or gene-region on the X chromosome could contribute to the manifestation of male SSA. It is pertinent to ask whether there is any indication of imprinting arising in MZ twins that could help explain cases of non-concordance. One recent report has described discordant imprinting and manifestation of Beckwith-Wiedemann syndrome in MZ twins (20). Recently, it has also been reported that epigenetic differences arise between MZ twins over their lifetime (21). These examples reveal potential differences between genetically identical (MZ) twins through differential patterns of gene expression (via epigenetic means) that are **not** dependent on differences in DNA sequence.

SUMMARY:

It should be apparent from the information presented in this paper that there is no simple scientific explanation for SSA and homosexuality in humans – despite the fact that homosexuality has been present within the fabric of human sexuality for thousands of years. Identical and fraternal twin studies imply a role for both genes and prenatal (within-womb) influences, but neither of these is sufficient, either alone or in combination. The search for genetic variants associated with SSA is still at an early stage, with several candidate DNA regions awaiting confirmation and further characterisation. Although evolutionary arguments for the prevalence of SSA can be suggested, they remain purely speculative in the absence of more definitive evidence. Even the preliminary work on human pheromones does little more than confirm what we already know. Science cannot as yet provide us with unambiguous answers as to why certain people become homosexual. **Nevertheless, the available scientific evidence does suggest a possible biological basis for SSA, perhaps involving several different pathways and/or factors that together create a predisposition to SSA in certain individuals. Furthermore, the candidate mechanisms identified to date are not subject to individual choice, nor can they be readily changed. Therefore homosexuality should not be regarded as an elective lifestyle choice.** The possibility of overriding these tendencies (e.g. by aversion therapy) is not extensively addressed here. However, the American Psychological Association (APA) has issued a strong statement that homosexuality is **not** an illness that requires treatment, nor is it a choice. Rather, homosexuality is a biological condition that is not subject to change by therapeutic modalities that seek to reverse a homosexual identity to heterosexuality. Further, the APA has expressed concerns that these therapies could potentially harm patients (22). On the other hand, both anecdotal and historical evidence suggests that SSA tendencies can be consciously or subconsciously repressed in many people living in societies where homosexuality is still seen as unacceptable or deemed a criminal behaviour. There remains a fundamental ethical question as to whether scientists should even seek to investigate the genetic basis of homosexuality (23, 24), because the most likely end result is that such knowledge will lead to demands for a genetic test to reveal the presence or absence of those genes, not only in adults but also prenatally. For example, if such testing were offered during pregnancy, parents might choose to abort an affected foetus – in the absence of any effective “therapeutic” alternative. Leaving aside the vexed question of whether that person (who would inherit at most a predisposition to SSA) would ever become homosexual, such developments would inevitably diminish human diversity, should any children be aborted as a result of the availability of such a test. Benjamin Britten’s towering contribution to twentieth-century sacred music should remind us of the losses we might thereby unwittingly incur. Recently, twenty-three theologians and biblical scholars within the Anglican tradition have responded to the aforementioned Windsor Report (25) because it expressed a greater concern for the nature of biblical authority and protecting the institutional Anglican Communion, rather than dealing with the underlying issues surrounding the inclusion of homosexuals (and their sexuality) into the life of the Church. The report is criticised for its failure to address substantive issues, favouring instead Church unity. The ripple effect of what the Anglican Communion is experiencing is now being felt throughout Christendom. Homosexuality in the church is no longer “under the

table”, because virtually all religious traditions are having to deal with homosexuality in their own context. The Church must begin to speak to the homosexual community as brothers and sisters, rather than merely to speak about them. In the last analysis, it is always possible to hide behind the Bible, or indeed behind the science (however inconclusive), when considering homosexuality. But there is really only one question from which no one can hide. How will each of us deal with a teenage son or daughter who comes home one day and states: “Dad and Mum, I’m gay”? And perhaps we should also ask, how would Jesus deal with that infinitely precious child of God?

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APPENDIX: WHAT DO DNA SEQUENCES TELL US?

This brief note is meant for readers unfamiliar with the basic biology of DNA or genes. The inherited genetic material of all organisms (apart from some viruses) is held in the form of DNA – long double-stranded helical molecules (either circular or linear) comprising thousands to billions of linked bases (either A, T, G or C). The discrete pieces of DNA are termed chromosomes. The arrangement of bases along a piece of DNA (e.g. AAAGCTTCCCGA) is termed its **sequence**; but because A pairs with T and G with C in the double helix, each single DNA strand specifies its complementary second strand (here TTTCGAAGGGCT). These DNA sequences are copied very accurately during cell division and reproduction, but occasional errors can occur, resulting in **mutations** (e.g. AACGCTTCCCGA). We now know the entire DNA sequence (genome) for an ever larger number of organisms. For humans, there is increasing information about DNA sequence variants (polymorphisms) that are common in the population, or that are associated with disease (e.g. PMMs). Only a small proportion (1-2%) of human DNA comprises the **genes** that specify all the proteins from which our cells and their vital functions are built. Thus many DNA sequence changes may be functionally unimportant, but a minority will affect the structure and/or expression of a protein. Regions of DNA can be activated (i.e. **transcribed** into an RNA strand which is then **translated** into a protein) or inactivated without changing the underlying DNA sequence information. These epigenetic processes involve either methylation/demethylation of C residues in the DNA, or else acetylation/deacetylation of the histone proteins around which the long strands of DNA are wound. These epigenetic features do not show the simple inheritance patterns of DNA.

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Human chromosomes each have two arms, p and q , that represent the short arm and long arm respectively. Distances from the centromere (which divides these two arms) are numbered such that higher numbers relate to a longer distance from the centromere. Thus $Xq28$ would be on the distal end of the long arm of the X chromosome. Other somatic chromosomes are numbered from 1-22 to differentiate them from the X and Y chromosomes that determine the sex of the individual.