

The Y chromosome as a battle ground for sexual selection

E.R.S. Roldan and Montserrat Gomendio

The concept of sexual selection was developed to explain the evolution of traits that confer advantages in competition over mates, often at the expense of survival rates¹. Such traits enhance reproductive success, either by improving a male's ability to win intrasexual contests or by making a male attractive to females. Male-male competition was intuitively accepted as an important process, and perhaps for this reason has not been the focus of much empirical research. Traits assumed to be important in male contests include weapons (such as horns, antlers and enlarged canines) and large body size.

In contrast, female choice has been a controversial subject. Reluctance to accept the theory has prompted not only numerous empirical studies, but also an intense effort to develop mathematical models. Although the available evidence supports the view that females actively choose their sexual partners, the implications of such choices remain to be explained. In some cases, females gain direct benefits, such as food, paternal care and access to high quality territories. However, the benefits in other cases remain obscure, particularly when females receive little more than sperm from males.

One particularly controversial hypothesis proposes that females look for 'good genes', but the nature of such genetic quality remains elusive. Whereas some authors argue that males in a population will differ in their intrinsic genetic quality and that females should aim to be fertilized by superior males²⁻⁸, other authors believe that it is the degree of genetic compatibility between a male and a female that matters^{9,10}. These issues are of particular interest to behavioural ecologists, who often have to infer differences in genetic quality between males from data on offspring survival rates, with little knowledge of the genetic basis underlying such differences.

Understanding the genetic mechanisms of sexual selection therefore remains a major challenge. There are currently two major theories¹: (1) runaway selection, which requires genetic coupling between trait (in males) and preference (in females), such that females choosing to mate with males possessing an attractive trait produce both attractive sons and choosy daughters; (2) indicator mechanisms, which suggest that females choose to mate with males with conspicuous traits because they indicate high viability, which will be inherited by their offspring. A further elaboration of this idea suggests that such conspicuous

The Y chromosome was once thought to be devoid of genetic information. However, recent work shows that it contains numerous genes related to sperm production and dimorphic traits (such as body size and tooth development). Among mammals, these traits influence a male's competitive ability in male-male contests and in sperm competition. Therefore, sexual selection could have favoured genes on the Y chromosome that enhance male fertilization success because they spread unaltered through the male line. In contrast, female heterogamety among birds makes it possible for genes that benefit females to spread through the female line, a mechanism that could explain the prevalence of female choice.

E.R.S. Roldan is at the Instituto de Bioquímica (Centro Mixto CSIC-UCM), Facultad de Farmacia, Universidad Complutense, 28040 Madrid, Spain (roldane@eucmos.sim.ucm.es); Montserrat Gomendio is at the Departamento de Ecología Evolutiva, Museo Nacional de Ciencias Naturales, CSIC, c/J. Gutierrez Abascal 2, 28006 Madrid, Spain (montseg@mncn.csic.es).

traits act as handicaps, which are costly for the male and therefore signal high genetic quality. Genetic models based on this idea have encountered a recurrent problem: females mating with males with the handicap will produce daughters that will benefit from their father's high genetic quality, but will also produce sons that will inherit the handicap trait, which might reduce their fitness.

It has been suggested that understanding the genetic basis of sexual selection needs to take into account the mechanisms of sex determination (Box 1, Fig. 1). A recent model¹¹ proposes that, when females are the heterogametic sex (e.g. in birds – the focus of much sexual selection research), choice genes present on the sex-determining chromosome (W) will be selected because daughters will benefit from the overall high genetic quality of the chosen male, even if such female preferences favour 'handicap' traits in males that are deleterious. Because such choice genes are

restricted to female offspring, they never incur the disadvantage of being associated with the deleterious male phenotype. This model overcomes the problems of previous models by assuming that choice genes can be present on the sex-determining chromosome and can thus spread exclusively through the female line.

The genetic mechanisms of sex determination in mammals are different from those of birds because males are the heterogametic sex. In this taxonomic group, therefore, the implications of the nature of sex-determining mechanisms for sexual selection require further scrutiny. In mammals, the sex-determining chromosome (Y) is inherited exclusively through the male line. Theory predicts that genes encoding traits that benefit males, even if they are detrimental to females, will accumulate on the Y chromosome¹².

New genes on the Y chromosome

For many years, it was assumed that the Y chromosome was a wasteland carrying no genetic information apart from the sex-determining gene *SRY* (Fig. 2a). Recent work^{13,14} has revealed that there are actually more than 20 genes or gene families in the nonrecombining region of the human Y chromosome (Fig. 2b). This region, which constitutes about 95% of the human Y chromosome, does not exchange genetic material with the X chromosome, in contrast to the recombination seen between homologous autosomes and between the pseudoautosomal regions of the X and Y chromosomes. The genes that have been identified in this

region of the Y chromosome fall into two categories (Fig. 2b): (1) genes that are ubiquitously expressed (many of them having 'housekeeping' functions), and with a high degree of homology to genes present on the X chromosome; and (2) genes expressed only in the testis (thought to be involved in the control of spermatogenesis), and that are only present on the Y chromosome^{13,15-17}. The existence of these Y-specific genes involved in the control of spermatogenesis indicate a unique functional coherence of the Y chromosome not seen in autosomes, which carry genes with no common function¹³.

There are ten genes on the human Y chromosome expressed exclusively in the testes¹³. These genes probably play a crucial role in the control of proliferation and/or differentiation of the male germ line to form spermatozoa. Their involvement in spermatogenesis is deduced from two sets of data. First, patients with a defective sperm production and, therefore, a reduced sperm number (severe oligospermia) or a total absence of spermatozoa in the ejaculate (azoospermia) have microdeletions in regions of the Y chromosome carrying these genes^{16,17}. Second, products of these genes have been found in germ cells at crucial stages of spermatogenesis¹⁸⁻²¹ or in mature spermatozoa²¹.

One important characteristic common to all the testis-specific genes on the Y chromosome is that they occur in multiple copies and can be polymorphic in their sequences²². This has three implications. First, gene copies might act together, increasing the 'efficiency' of spermatogenesis. Second, deletions or mutations in one copy would not completely block spermatogenesis²³. Third, different males in a population would have Y chromosomes with a different number of copies of each gene, or with different sequence 'repeats', or even different combinations of copy numbers and sequences (*RBM*, Ref. 24; *DAZ*, Ref. 25). This leads to the following question: is there a relationship between the number of copies of genes controlling spermatogenesis in different individuals and the number of sperm, or sperm abnormalities, in those individuals?

Other genes on the Y chromosome

Other genes on the Y chromosome, whose presence has been either inferred or actually demonstrated, include several that might also be important in sexual selection, such as those related to the control of embryonic growth, stature and the development of teeth.

In several mammalian species, XY embryos grow faster than XX ones²⁶. The XY individuals are also developmentally more advanced than their XX counterparts at the same gestational age (reflected in the number of blastomeres or somites, weight, body length and head size)^{26,27}. This Growth Factor Y-effect has been shown to persist to mid-gestation.

It has been proposed that the Y chromosome has a region controlling stature²⁸. A putative gene named Growth Control Y (*GCY*), with a marked effect on stature, has been identified in the most proximal region of the long arm of the human Y chromosome²⁹. It is likely that this *GCY* gene is different from the gene determining the Y-effect on embryo growth (Growth factor Y) described above²⁶.

Measurements of enamel and dentine thickness of permanent incisors and canines in normal females and males, and in individuals with sex-chromosome abnormalities, have suggested that the Y chromosome influences dental growth by promoting both amelogenesis (i.e. the growth of enamel) and dentinogenesis (i.e. the growth of dentine)³⁰. The effect of the Y chromosome on tooth growth could explain the expression of sexual dimorphism in size, shape

Box 1. Origin and evolution of sex chromosomes in vertebrates

Distinct sex chromosomes have evolved independently in many taxa. In amphibians and reptiles both XX–XY (male heterogamety) and ZZ–ZW (female heterogamety) systems are found. In birds, only the ZZ–ZW system is seen, whereas mammals only have the XX–XY system. Sex chromosomes in birds and mammals have no homology because they evolved independently from different autosomal pairs present in a common ancestor (Fig. 1).

How did sex chromosomes originate and evolve? A likely sequence⁴² involves the acquisition of a sex-determining role by one chromosome, followed by suppression of recombination (crossing-over restriction), structural rearrangement, and degradation of the chromosome carrying the sex-determining gene. The key to this evolutionary process seems to be the lack of crossing-over between the X and the Y (or Z and W), with the X (or Z) keeping essential genes, and the Y (or W) experiencing loss of nonessential genes. Crossing-over restriction is seen in the heterogametic sex of amphibians, reptiles, birds and mammals. It is a primitive phenomenon occurring before sex-chromosome differentiation, given that it is observed in homomorphic (e.g. the fish *Oryzias latipes*) or almost homomorphic chromosomes (e.g. newts, *Triturus* spp.). Sex-chromosome heteromorphism is thus a consequence of sex-chromosome specialization⁴³.

The existence of a nonrecombining region in the Y or W chromosome implies that genes present in this region would be transmitted 'clonally' (i.e. unaltered) to the descendants of the same heterogametic sex. In mammals, genes in the non-recombining region of the Y would be transmitted to sons, whereas in birds putative genes in the nonrecombining region of the W would be transmitted to daughters.

Fisher⁴⁴ pointed out that a mutant gene that is advantageous in the heterogametic sex, but disadvantageous in the homogametic sex, would be much more likely to spread within the population if linked to the sex-determining region of the Y (or W) chromosome, because this would enable it to be predominantly associated with the sex in which it is advantageous⁴⁵.

In addition to genes present in the ancestral autosome (e.g. proto-Y), genes can end up in the Y chromosome by successive additions (to the X and Y) via the pseudoautosomal region, and/or additions directly to the nonrecombining region of the Y chromosome^{32,42}. Alternatively, genes in the Y chromosome can be eliminated by attrition (because of the accumulation of deleterious mutations in the absence of recombination between the X and the Y), unless they have a positive benefit to the male^{12,45}.

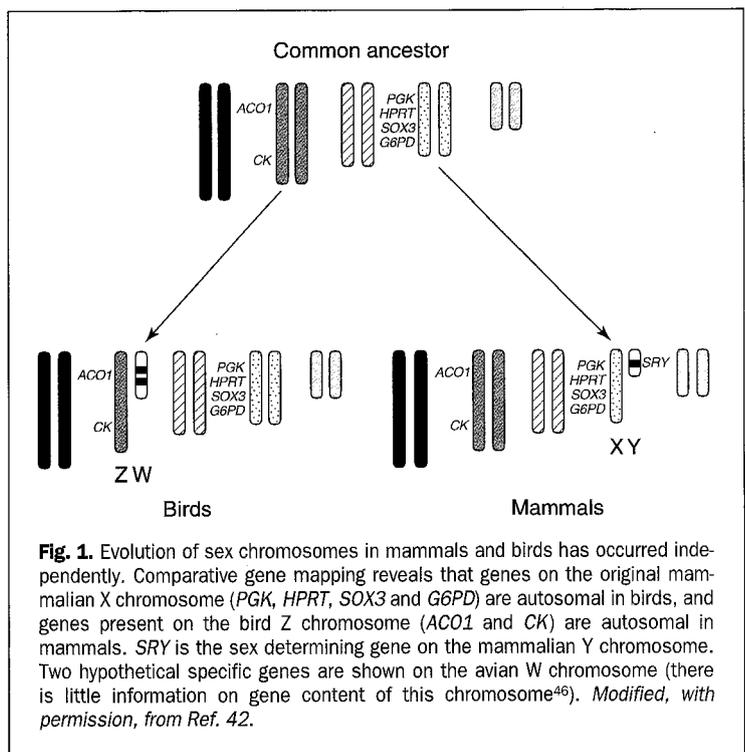


Fig. 1. Evolution of sex chromosomes in mammals and birds has occurred independently. Comparative gene mapping reveals that genes on the original mammalian X chromosome (*PGK*, *HPRT*, *SOX3* and *G6PD*) are autosomal in birds, and genes present on the bird Z chromosome (*ACO1* and *CK*) are autosomal in mammals. *SRY* is the sex determining gene on the mammalian Y chromosome. Two hypothetical specific genes are shown on the avian W chromosome (there is little information on gene content of this chromosome⁴⁶). Modified, with permission, from Ref. 42.

and number of teeth³⁰. Growth of tooth buds during human development is promoted by a gene on the Y chromosome (*AMELY*) and an homologous gene (*AMELX*) on the X chromosome³¹. *AMELX* transcripts are more abundant than the *AMELY* ones. However, the *AMELX-AMELY* genes are functionally

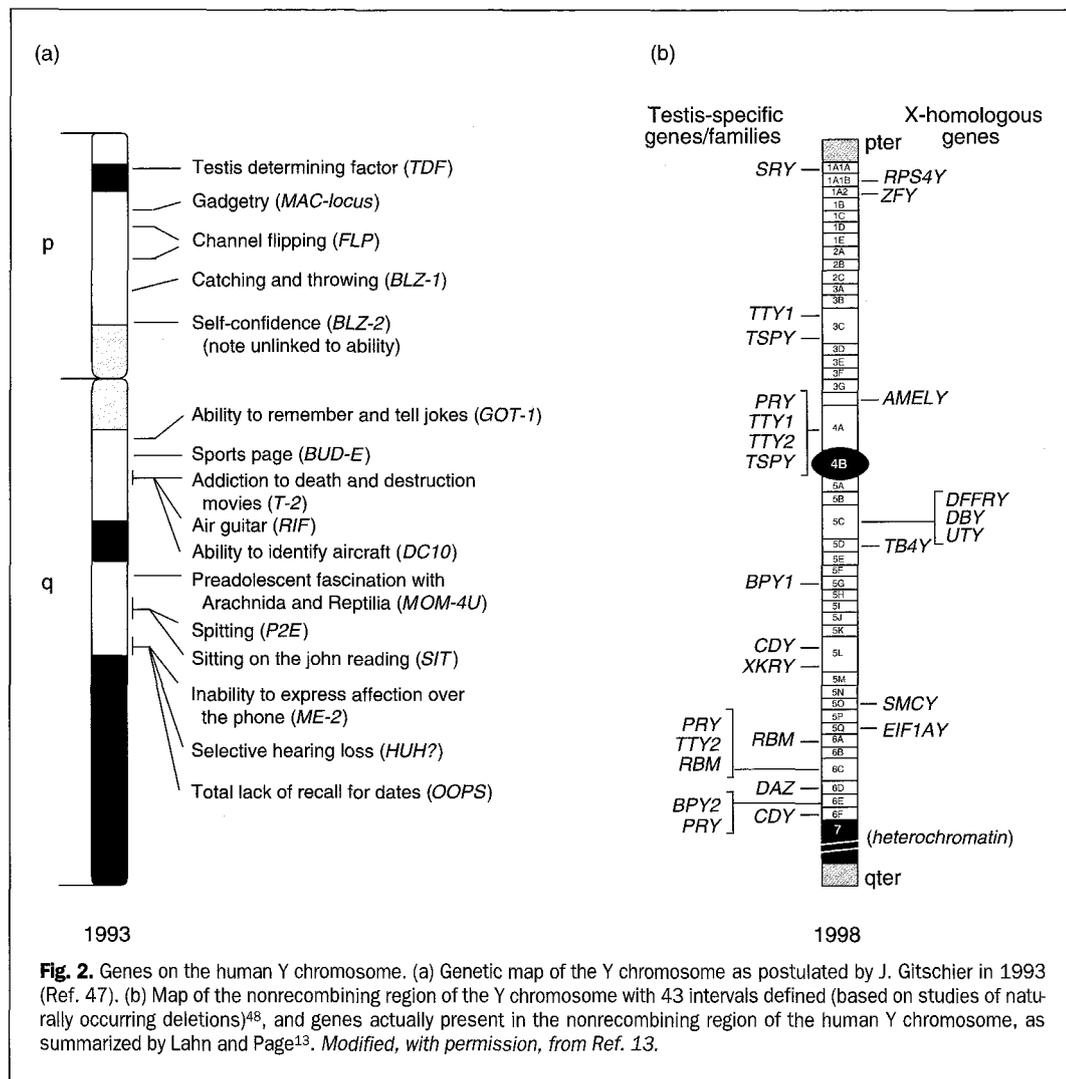


Fig. 2. Genes on the human Y chromosome. (a) Genetic map of the Y chromosome as postulated by J. Gitschier in 1993 (Ref. 47). (b) Map of the nonrecombining region of the Y chromosome with 43 intervals defined (based on studies of naturally occurring deletions)⁴⁸, and genes actually present in the nonrecombining region of the human Y chromosome, as summarized by Lahn and Page¹³. Modified, with permission, from Ref. 13.

unique because X inactivation can act on the X locus so that males have potentially more gene product than do females³¹. It is possible that *GCY* is also involved in tooth development, because individuals with deletions in the region containing this putative gene also have smaller teeth²⁹.

The search for products of gene expression has been guided by the assumption that they would be found preferentially in the testis¹³. More genes might be discovered on the Y chromosome if expression products are looked for in other tissues, such as brain³². Further searches would benefit from clear predictions as to which genes are likely to be found on the Y chromosome.

Evolution of genes on the mammalian Y chromosome

One important feature of the genes on the Y chromosome is that they show a high degree of conservation across mammals, including marsupials (Fig. 3). The best example is the testis-determining gene *SRY* that, with the exception of two species (having no Y chromosome)³³, is present in all mammalian species studied so far¹⁴, although, interestingly, its sequence is not highly conserved.

Genes on the Y chromosome that are expressed exclusively in the testis and are thought to control sperm production, are also present in a wide range of mammals. Two genes, *RBM* and *TSPY*, are present in all mammalian lineages examined (*RBM*: Refs 15,34,35; *TSPY*: Ref. 14). Interestingly, other genes expressed exclusively in the testis (the gene family *DAZ-SPGY*) are only present in the lineage of Old World monkeys (Cercopithecoidea) and the Great Apes (Hominoidea),

including humans, although within Hominoidea they are very conserved^{36,37}.

For those genes on the Y chromosome that have homologues on the X chromosome, it is still not obvious whether there are any genes that are conserved across all mammals, because results are still fragmentary (Fig. 3). However, it is evident that genes such as *ZFY* are present in all eutherian ('placental') mammals, and that *AMELY* is present in all eutherians with the exception of rodents. Another good example of conservation is *Ube1y*, which is found in marsupials, rodents, rabbits, dogs, horses and various species of Artiodactyla, although it is absent in Old World and hominoid Primates¹⁴ (Fig. 3). It will be very interesting to see what degree of conservation exists for the genes recently identified on the human Y chromosome¹³.

Some of the genes now found on the Y chromosome, such as *DAZ* of Old World monkeys and hominoids, were originally autosomal genes (Box 1). An autosomal homologue (*DAZL1*, formerly

called *DAZH*, *DAZLA* and *SPGYLA*) exists in these species and is also found on autosomes of rodents, artiodactyls and New World primates (Cebioidea). The autosomal *DAZL1* also has a role in the control of spermatogenesis³⁸, and is homologous to genes regulating spermatogenesis in *Drosophila* (*boule* gene)³⁹ and *Xenopus* (*Xdazl* gene)⁴⁰. It has been postulated that the primate *DAZ* was duplicated at some point and transferred to the Y chromosome³⁶, and it is thought that this could have been a recurrent theme in evolution with autosomal-to-Y transfer of genes resulting in genes being present exclusively in males (Box 1).

Genes on the Y chromosome and sexual selection

Although not all genes are conserved in all species, the common theme is that a group of genes involved in the control of spermatogenesis is present in the nonrecombining region of the Y chromosome in mammals. In addition, genes present on the Y are also related to rapid growth rates, adult body size and tooth development.

What are the implications of these findings for sexual selection theory? In mammals, given that males are the heterogametic sex (XY) and that there is no recombination of most of the Y chromosome, traits encoded by genes present on the Y chromosome will pass on unaltered from father to son. The evidence reviewed here indicates that such traits are mainly related to sperm production. Traits that ensure a male's reproductive capacity will be under strong selection. Such traits will also enhance a male's ability to win in sperm competition, a widespread phenomenon among

mammals⁴¹. Genes present on the Y chromosome are also related to traits that are known to be important in male contests, such as large body size and tooth development. Thus, genes on the Y chromosome code for traits that make males more competitive in intrasexual contests. The spread of such traits might have negative effects on females, who will incur greater costs in raising competitive sons.

These findings support the theoretical prediction that mutations beneficial to males, but detrimental to females, will accumulate on the sex-determining chromosome. This is because such genes will only be transmitted to sons (where they are favoured) and not to daughters (where they would be selected against). In this respect, the Y chromosome behaves very differently from autosomes. Traits coded by autosomes will only spread when they proffer a net selective advantage, either because they are beneficial to both sexes or, in the case of sexually antagonistic alleles, because the benefit to one sex is greater than the disadvantage to the other. Consequently, the mechanism of sex determination in mammals allows sexual selection to favour the evolution of traits that are beneficial to males in terms of male-male competition. This might explain why male-male contests seem more common in mammals than female choice.

In birds, where females are the heterogametic sex, sexual selection has the opportunity to favour traits present in the sex-determining chromosome that, because they spread exclusively through the female line, need only be beneficial to females. This could be the reason why female preference for costly male traits seems to be particularly common in birds.

Future directions

In conclusion, mechanisms of chromosomal sex determination provide an opportunity for the heterogametic sex to accumulate genes that are beneficial to that sex, even at the expense of the homogametic sex. In mammals, such genes spread through the male line and are related to ejaculate quality and competitive ability, whereas in birds such genes pass on through the female line and are predicted to be related to the ability to choose male handicaps. In this battle of the sexes, there seems to be little the homogametic sex can do.

We hope that this review will stimulate behavioural ecologists and molecular biologists to interact more closely in attempts to understand how genes on the Y chromosome have evolved in relation to sexual selection. Researchers working on sexual selection should pay more attention to genetic mechanisms – the evidence reviewed here suggests that a better understanding of sex-determining mechanisms could reveal ways in which sexual selection can operate. In addition, recent advances in molecular biology could help behavioural biologists clarify what ‘genetic quality’ means, as well as the consequences of sexual selection processes for males and females.

Moreover, molecular biologists could become more efficient in their search for genes if they use the framework of sexual selection to make more explicit predictions as to which genes they expect to find in sex-determining chromosomes. As we have seen, these are likely to differ markedly between taxa.

Note added in proof

Syntentic homology has been recently shown between gene dense regions of the Y chromosomes of the mouse and man⁵².

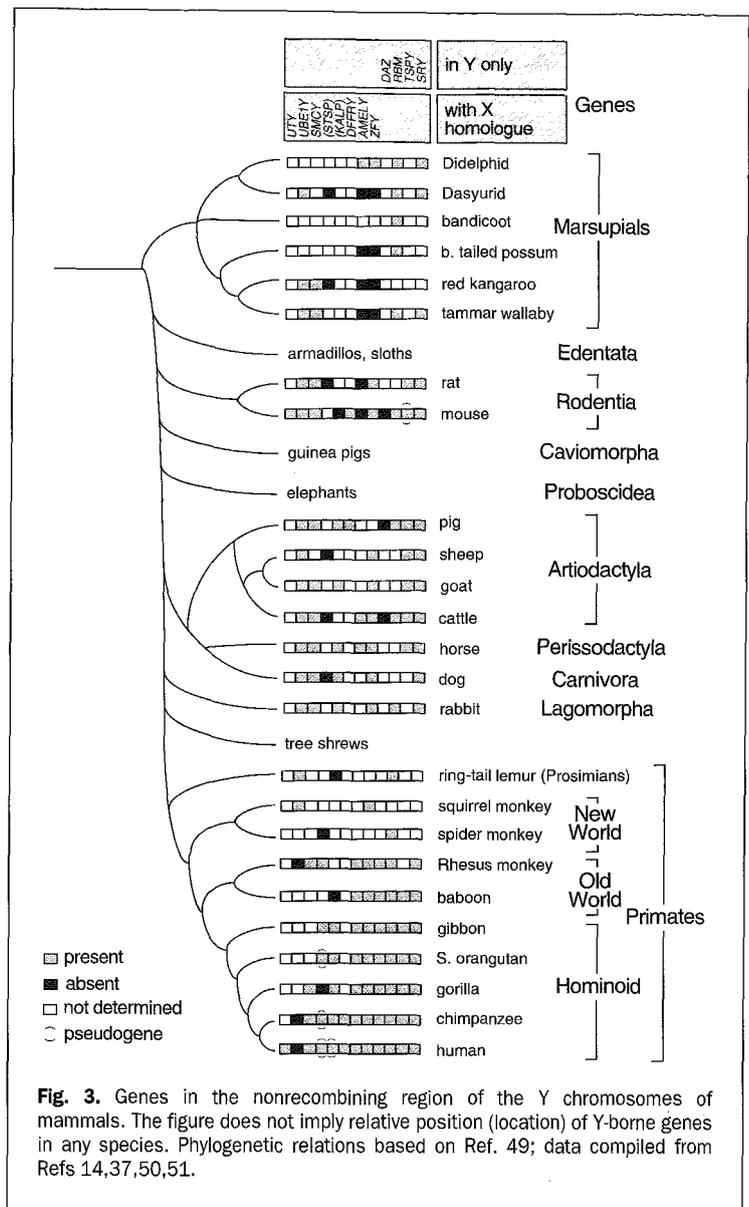


Fig. 3. Genes in the nonrecombining region of the Y chromosomes of mammals. The figure does not imply relative position (location) of Y-borne genes in any species. Phylogenetic relations based on Ref. 49; data compiled from Refs 14,37,50,51.

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