

## Outline of lectures 27-29

### Evolution of Genetic Systems

1. Features of our genetic systems are explained to various degrees by evolutionary biology. Starting with the best-explained and proceeding to the least well-explained, they are:
  - Sex ratios
  - Degeneration of Y chromosomes,
  - anisogamy (unequal-sized male and female gametes)
  - outcrossing with recombination (“sex”)
  - Diploidy
  - mutation rates.
2. **Sex determination** mechanisms vary widely. They include (see the overhead for more): XX-XY, ZW-ZZ, XX-XO, XX-XY<sub>1</sub>Y<sub>2</sub>, arrhenotoky (haplo-diploidy), environmental sex determination, self-sterility systems, and sequential hermaphroditism.
  - (a) XX-XY. Like us, males are XY and females are XX. Angiosperms having two sexes, most vertebrates.
  - (b) ZW-ZZ. The reverse of us. Males are ZZ and females are ZW. Most notably, birds, butterflies, moths. Some flatworms, some dipterans, some amphibians, some lizards.
  - (c) XX-XO. Like XX-XY except that there is no Y chromosome: males are those who are haploid for the X chromosome. Many insects.
  - (d) XX-XY<sub>1</sub>Y<sub>2</sub>. Two Y chromosomes pair with the X in males and both segregate to the same pole in the first division of meiosis. The Indian Muntjak, a small deer with fangs, is one example.
  - (e) Arrhenotoky. Haplo-diploid sex determination, which I mentioned in the discussion of kin selection explanations for social behavior in insects. Ants, bees, wasps (i.e., Hymenoptera), thrips, mites and ticks, many rotifers.
  - (f) Environmental sex determination. You’re female if you have: better nutrition (nematode roundworms), colder temperatures during development (lizards and crocodiles), hotter temperatures during development (most turtles), more extreme temperatures during development (snapping turtles and alligators).

- (g) Sequential hermaphroditism. Being transgendered, naturally. Start life as a male, later in life change to a female. Oysters, shrimp, some fish (including clownfish, the *Finding Nemo* fish).
  - (h) Self-sterility. In plants, have a particular multi-allele locus for which you can mate if the two individuals do not have any allele in common, or if the female plant tissue does not share any allele with the pollen that fertilizes it. This leads to many alleles at roughly equal gene frequencies. Are these “sexes”? Depends on your definition.
3. **Sex ratios** are often near 1:1, even when there is no good ecological reason why that would help the species. Often a species would produce more offspring if there were more females and fewer males. Nevertheless the sex ratio stays near 50% males.
  4. Darwin, in the first edition of *The Descent of Man*, and the Prussian biologist Carl Düsing in 1883-4 gave an explanation of this which is the standard modern theory. It is usually attributed to R. A. Fisher, in 1930, who mentioned it.
  5. It depends fundamentally on the fact that each offspring has one female and one male parent. If there is an excess of one sex (say females), then a gene that causes its bearers to become males more often will have an advantage. It will have it because the males as a whole are having as many offspring as the females as a whole have. So in that case the average male has more offspring than the average female. Therefore by producing more males, the gene will be more represented in the next generation.
  6. As an example suppose there are 10 times as many males as females. If there are 10,000 females and 100,000 males, a parent having a child would get more of its genes into the next generation if it has a female offspring. As the females as a whole contribute equally to the next generation as do the males as a whole, it pays to have an offspring be a female. For then it comprises 1/10,001 of all female genetic material but if it were male it would comprise only 1/100,001 of all male genetic material.
  7. This is not just an impressionistic verbal argument – it can be quantitated by working out the gene frequencies in males and females in future generations. The gene will increase as long as there is a deficiency of males, and as long as it produces more males. If a gene produces more than 50% males in its bearers, it will increase until it reaches a frequency where the population has a 50:50 sex ratio.
  8. The same argument works in haplo-diploids, and even in environmental sex determination systems.
  9. It also works even in sequential hermaphrodites, where it predicts equal effort put into being a male and into being a female.

10. It works even when there is only one sex, in monoecy, in simultaneous hermaphrodites such as many flowers, where it predicts equal reproductive investment in ovules and pollen (spending as much total energy producing pollen as ovules). If pollen are cheap, you get a reward for making many pollen grains instead of a few ovules – unless the total numbers around are already at the point of equal allocation of resources. Later we will see that this becomes important to the evolution of “sex” (recombination with outcrossing).
11. There are some weird phenomena possible involving Y chromosomes. For instance, if one has a “driven” Y which gets into gametes more than 50% of the time, it will increase in frequency. If a Y gets into 100% of gametes, it will increase to fixation *even though as it does so the population goes extinct!* Has this happened? It is hard to know, as what is left are the species that do not have such a driven Y. In any case modifiers of the X or autosomes that resist this will be favored too.
12. **The degeneration of Y chromosomes.** Y chromosomes commonly have few functional loci. This degeneration of the Y was finally explained in 1978 by Brian Charlesworth, using the phenomenon of “Muller’s Ratchet”, which was invented by H. J. Muller (and explained more in 1974 by me, too).
13. Y chromosomes have no recombination through most of their length, for a simple reason: to avoid producing chromosomes that have some but not all of the essential male-determining signals.
14. H. J. Muller pointed out that such recombinationless chromosomes would degenerate. They pick up mutants, at various loci. If the population gets into a state where each copy of the chromosome has at least one mutant, then without recombination it can never get a chromosome which has no mutants. The ratchet has moved one notch. This process continues, and mutations pile up on the chromosome, though the rate at which this happens may be slow if selection against the mutants is strong, mutation weak, and/or population size large.
15. Back mutation can actually reverse the ratchet, but it is a rare phenomenon and will not do so until it is far advanced. Recombination could produce mutant-free copies of the chromosome, but it is absent.
16. As more mutations pile up on the chromosome, the ratchet moves further – all of them have 2 mutants, all have 3, and so on. Ultimately these (mostly recessive) mutants knock out all loci except the sex-determining ones.
17. As this happens there comes to be a dosage-compensation problem (of producing the same phenotype with one X in males, and with two in females). Such systems would be strongly selected for at this point.

18. Other regions of the genome that have little recombination, such as the regions near centromeres, are subject to the same loss of functional loci.
19. **The evolution of recombination** with outcrossing is also to some extent explained by Muller's Ratchet. This is often called the "evolution of sex" to attract more attention, although it has nothing to do with the evolution of the differentiation between the sexes. Outcrossing allows genes from different parents to be recombined into the same offspring. This is not in and of itself an advantage (contrary to a lot of ill-considered statements).
20. Outcrossing with recombination allows the Ratchet to be unsprung. If we did not outcross all our chromosomes would be subject to the Ratchet.
21. (Fully self-fertilizing plant species should be subject to the Ratchet too).
22. An alternative explanation, due to Fisher (1930) and Muller (1932), is that an advantage of recombination is that it allows advantageous mutants that arise in different parents to both be fixed in the same population. (In a sense the Ratchet is a similar phenomenon, except that the advantageous alleles start out at high frequency instead of as mutants; recombination allows the favored nonmutant alleles to be combined).
23. A second major class of explanations for recombination is the theory of Sturtevant and Mather (1942) who imagined a case in which in some generations selection favors gene combinations  $AB$  and  $ab$ , but in others the combinations  $Ab$  and  $aB$ . The presence of recombination helps the population rapidly switch between these states, and can be used as the basis of an argument that modifier genes that encourage recombination will be favored.
24. The late W. D. Hamilton (of kin selection fame) pushed the idea that the evolutionary pressure caused by rapidly evolving parasites could select for some gene combinations in some generations, and different ones only a few generations later. This biological scenario uses the Sturtevant/Mather explanation. It is usually called the "sex and parasites" explanation.
25. Interestingly, the most common explanation for "the evolution of sex" (i.e. recombination and outcrossing) is that "it creates variability". This is ill-defined, and in most models it does nothing of the sort.
26. **The Cost of Meiosis.** The problem with all of these explanations is that John Maynard Smith has shown that there is a Cost of Meiosis, which is the 50% loss of fertility of the species due to wasting effort on making males (it is really a Cost of Males). This will select strongly against sexual lineages.

27. That 50% of the effort will be spent on males is actually a consequence of Düsing's sex ratio argument. It means that the Cost of Meiosis is just as serious in, say, hermaphrodite plants, where it is a cost of pollen production.
28. A clonally-reproducing organism will have a twofold advantage. The existing arguments for the evolution of recombination are hard-pressed to overcome this.
29. There are other explanations (the sex-and-parasites argument and an argument that recombination is needed only for repairing double-stranded DNA breaks are the main ones). At the moment we have lots of explanations and too few ways of telling which might be important. We also have the pesky Cost of Meiosis.
30. It is not that evolutionary biology cannot explain "sex" (as is sometimes implied) but rather that it has too many explanations and not enough relevant ways to choose among them.