# Evolution of genetic systems

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# How well can we explain the genetic system?


Sex ratios of 1/2 (C. Düsing, 1884, W. D. Hamilton, 1967)

Degeneration of Y chromosomes (B. Charlesworth, 1978; Orr, 1998)

Anisogamy and sexual dimorphism (Parker, Baker, and Smith, 1972)

Recombination
(Fisher, 1930; Muller, 1932; Sturtevant and Mather, 1938)

Diploidy

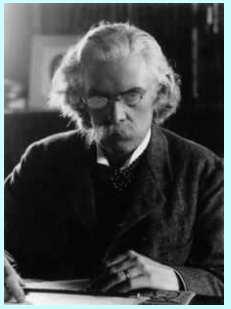
Mutation rates

# Sex determination systems

1. XX-XY  Like us: XX is female, XY is male
   Who: Many of the dioecious angiosperms (flowering plants), many animal species, including most vertebrates.

Sophia Kovalevskaya

| ♀ \ ♂ | X | Y |
|---|---|---|
| X | XX ♀ | XY ♂ |
| X | XX ♀ | XY ♂ |



Gösta Mittag-Leffler

2. ZW-ZZ.  The reverse of us: ZW is female, ZZ is male.
   Who: some flatworms, some crustaceans, some insects, especially lepidopterans and some dipterans, some fish, some amphibians, some lizards, most birds.

| ♀ \ ♂ | Z | Z |
|---|---|---|
| Z | ZZ ♂ | ZZ ♂ |
| W | ZW ♀ | ZW ♀ |

# More sex determination systems

3. XX-XO Like XX-XY but with no Y chromosome at all. In males half the gametes get the X, half get no sex chromosome. (Not the same as haplo-diploidy: the rest of the chromosomes are normal).
   Who: Many insects

| ♀ \ ♂ | X | O |
|---|---|---|
| X | XX ♀ | XO ♂ |
| X | XX ♀ | XO ♂ |

4. XX-X$Y_1Y_2$ An XX-XY system but with two Y chromosomes that pair with the X (each with one end of it). They segregate together, with the X going to the other pole.
   Who: most notable example is the Indian Muntjak Deer.

| ♀ \ ♂ | X | $Y_1Y_2$ |
|---|---|---|
| X | XX ♀ | X$Y_1Y_2$ ♂ |
| X | XX ♀ | X$Y_1Y_2$ ♂ |

# Yet more sex determination systems

5. Arrhenotoky Haplo-diploid sex determination. Males are haploid, females are diploid. Unfertilized eggs are haploid. There are no sex chromosomes (or else you could say they all are).
   Who: Hymenoptera (ants, bees, wasps), thysanoptera (thrips), mites and ticks, many rotifers

| | ♂ haploid sperm | (no sperm) |
|---|---|---|
| ♀ haploid egg | diploid ♀ | haploid ♂ |

6. Environmental sex determination. No sex chromosomes. The sex of an individuals is determined by the environment when it is developing.

| | | |
|---|---|---|
| More ♀♀ if: | better nutrition | nematodes (roundworms) |
| " | colder temperatures | lizards, alligators |
| " | hotter temperatures | most turtles |
| " | extreme temperatures | snapping turtles, crocodiles |

# And yet more

7. Sequential hermaphroditism.
   Start life as one sex (usually male)
   and switch later.

   Who: oysters, shrimp, and some fish.

8. Self-sterility systems. In some angiosperms (flowering plants) there are multiple alleles at a locus, that allow pollen to succeed in fertilizing only if it
   - it does not contain the same allele at that locus as the female ovule (gametophytic self-sterility)
   - it does not contain the same allele as either found in the female (sporophytic self-sterility)


## The evolution of the sex ratio

In a work (usually mistakenly attributed to R. A. Fisher, 1930 and called "Fisher's theory of the sex ratio"), Charles Darwin (*Descent of Man* (1871, 1st edition only) and Carl Düsing (1883 and 1884) put forward the modern theory of why sex ratios tend to be 1:1 :

> The females as a whole and the males as a whole contribute equally to the next generation, and to the ancestry of all future generations.
>
> If one sex is in short supply, an individual will contribute more to the future gene pool if it is of that sex (as then it is a bigger fraction of that half of the gene pool).

Düsing, C. 1884. *Die Regulierung des Geschlectsverhältnisses bei der Vermehrung der Menschen, Tiere und Pflanzen.* Fischer, Jena.

# Should one “want" to be a member of the minority sex?

Consider an allele that affects the probability that its bearer is a female.

| number of parents | genotype | offspring females | offspring males |
|---|---|---|---|
| 75,000 | aa | 100,000 | 50,000 |
| 100 | Aa | 100 | 100 |

Frequency of A among parents $= \frac{100}{150{,}200} = 0.00066578$

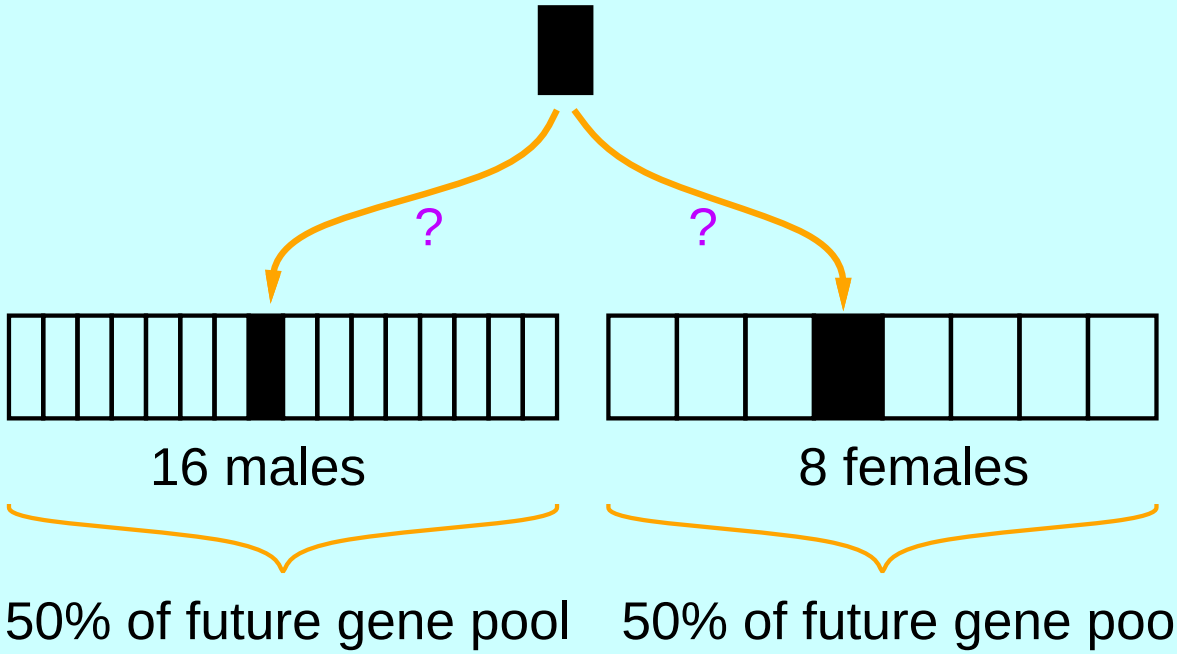
Frequency of the A allele, counting copies of genes

among females: $= \frac{100}{200{,}200} = 0.0004955$

among males: $= \frac{100}{100{,}200} = 0.000998$

The frequency of A among all offspring in the next generation is the average of these two numbers, which is $0.00074875,$ an increase.

## Visually, the Düsing theory:




Should you want your offspring to be in the majority sex or the minority sex, given that each of those sexes contributes, as a whole, half of the future gene pool? (In the above case, there are currently twice as many males as females).

# Monoecious organisms use the same system

Monoecious organisms such as many flowering plants face the same logic when they allocate energy among production of pollen and ovules.

- Should they devote more energy to producing ovules, or pollen?
- It can be shown that they should allocate energy equally to both.
- (For example, if pollen is ten times cheaper than ovules, by allocating to pollen, you have ten times as many offspring).
- The choice becomes equal when the total energy allocated to each in the species is equal (I will not explain the details here).
- This results in there being far more pollen around than the species “needs”.

We will see this having an important effect on the argument for evolution of “sex” later on.

# A driven Y chromosome – rigging the Mendelian rules

Suppose there is a Y chromosome that causes all offspring of a mating to be Y-bearing males, without reducing the total number of offspring. We then expect, if $p$ of the males have this Y* chromosome, in the next generation, the total fraction of offspring will be:

males: $\frac{1}{2}(1-p)+p$

females: $\frac{1}{2}(1-p)$

(since the Y*'s from the driven males will all go to male offspring, and there will be twice as many of these from those parents) and the frequency of the Y* chromosome among Y's should follow the equation:

$$p_{t+1} = \frac{p_t}{\frac{1}{2}(1-p_t)+p_t}$$

while the fraction of females among the offspring in generation $t$ will be

$$\frac{1}{2}(1-p_t)$$

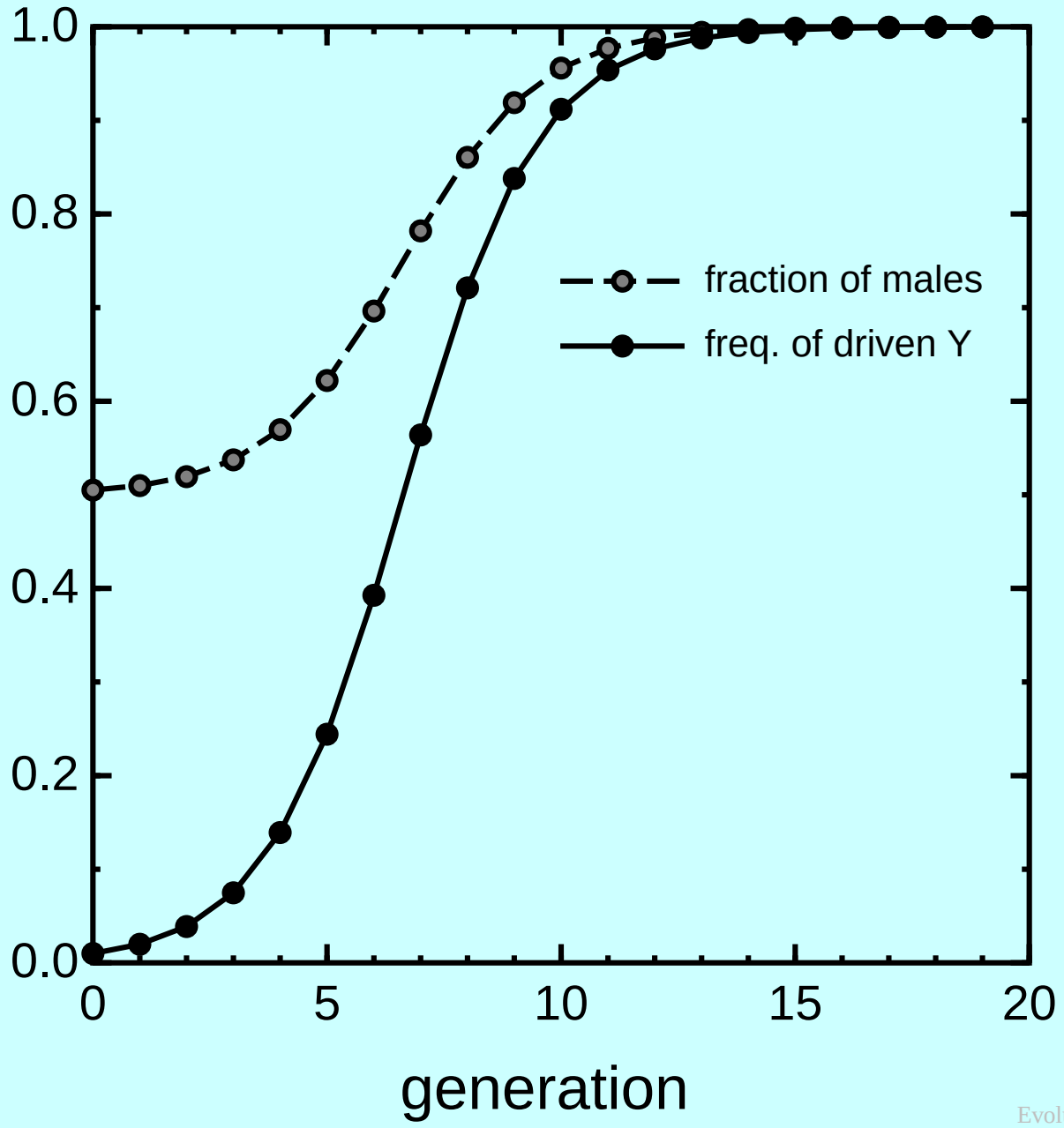
# Frequency of a meiotically driven Y chromosome

Here are values, starting at 0.01 frequency of Y* among Y's:

| generation | p | fraction of females |
|---|---|---|
| 0 | 0.01 | 0.5 |
| 1 | 0.0198 | 0.495 |
| 2 | 0.03883 | 0.4901 |
| 3 | 0.07476 | 0.4805 |
| 4 | 0.13913 | 0.4626 |
| 5 | 0.24427 | 0.4304 |
| 6 | 0.39364 | 0.3779 |
| 7 | 0.56387 | 0.3037 |
| 8 | 0.72113 | 0.2181 |
| 9 | 0.83797 | 0.1394 |
| 10 | 0.91184 | 0.0810 |
| 11 | 0.95389 | 0.0441 |
| 12 | 0.97640 | 0.0231 |
| 13 | 0.98806 | 0.0118 |
| 14 | 0.99399 | 0.0059 |

The population is evolving its way to extinction!

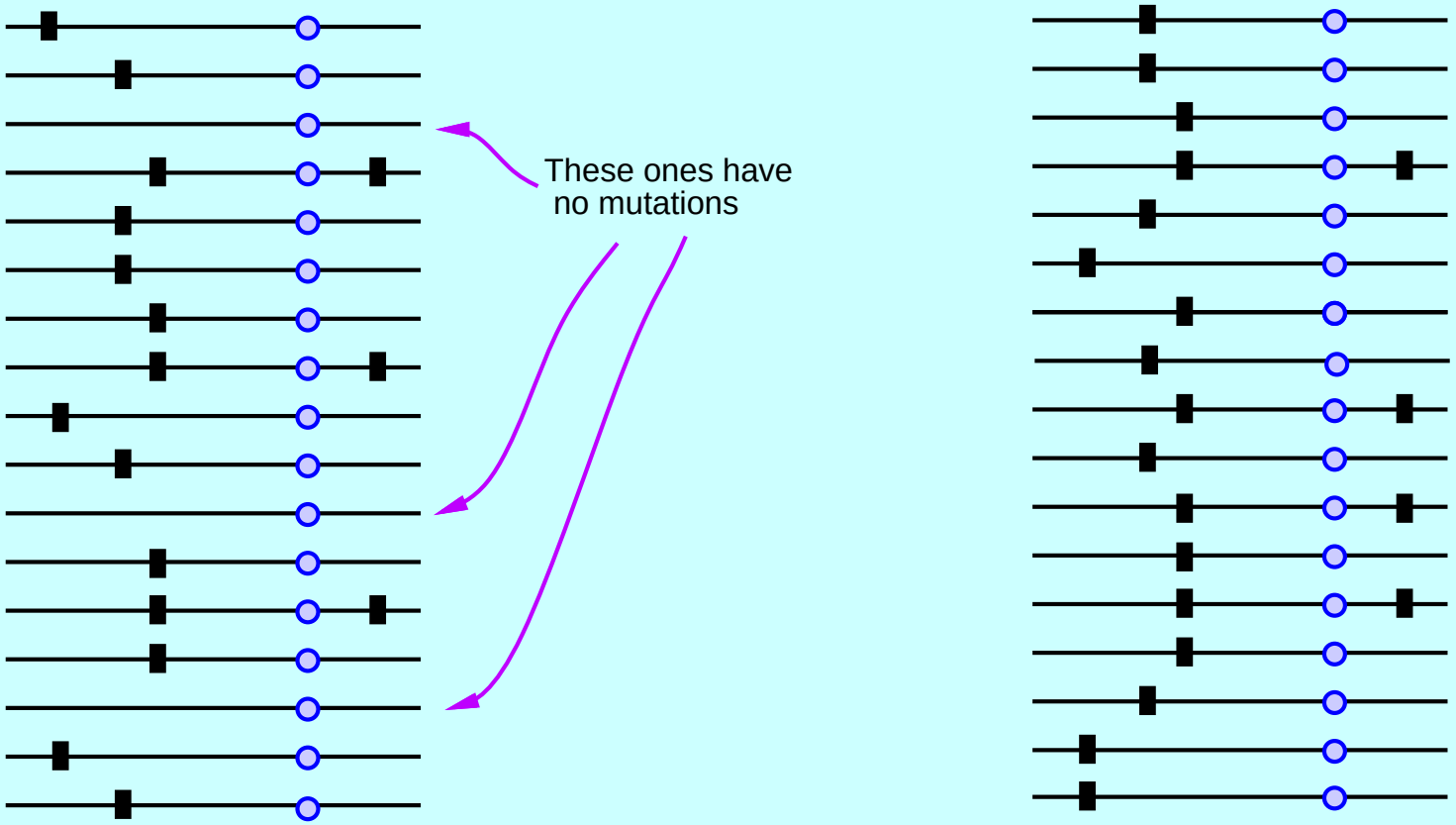
# A meiotically driven Y evolves to extinction



# Muller's ratchet

Suppose we have a population in which chromosome copies have deleterious mutations

Suppose genetic drift loses the chromosome(s) with no deleterious mutations:




The population can recover "wild−type" chromosomes by recombination.

Otherwise it has to wait for reverse mutation. The ratchet has moved one notch.

Gradually the mutations accumulate.

# Degeneration of the Y chromosome: Muller's Ratchet?

Brian Charlesworth (*Proc. Natl. Acad. Sci.*, 1978) has argued that the lack of recombination between X and Y chromosomes makes the Y subject to Muller's Ratchet. (I leave it to you to figure out why the X *isn't* subject to the same phenomenon). This was the first good explanation of why the Y chromosome has lost most genes aside from some needed for maleness.

A similar phenomenon occurs in regions of the chromosomes near centromeres and near telomeres, where recombination is reduced. Tandem repeated noncoding sequences seem to accumulate in the regions, and there are few functional genes.

# Major explanations for the evolution of recombination

1. It creates variation (East and Jones, 1919). Unfortunately it is easy to show that it destroys just as much variation, so this one doesn't even work: For example (in a haploid)

| Creates variation | | | Destroys variation | | |
|---|---|---|---|---|---|
| Phenotype | Parents | Offspring | Phenotype | Parents | Offspring |
| 2 | | AB | 2 | AB | AB |
| 1 | Ab $\times$ aB | Ab, aB | 1 | $\times$ | Ab, aB |
| 0 | | ab | 0 | ab | ab |

2. It breaks down random linkage disequilbrium which slows down response to selection (Fisher, 1930; Muller, 1932; Muller, 1958, 1964) Major variants:
   - Fisher and Muller's argument that recombination allows advantageous mutants to get into the same descendant.
   - "Muller's Ratchet", that recombination allows deleterious mutants at many loci to be eliminated even when haplotypes that have no deleterious mutants have been lost by genetic drift.

## more theories of evolution of recombination

3. (continued:)

   - Sturtevant and Mather's (1938) argument that recombination helps the pattern of linkage disequilibrium change rapidly in response to changes in the pattern of multi-locus selection. This has been the basis of Hamilton's "parasites and sex" explanation.

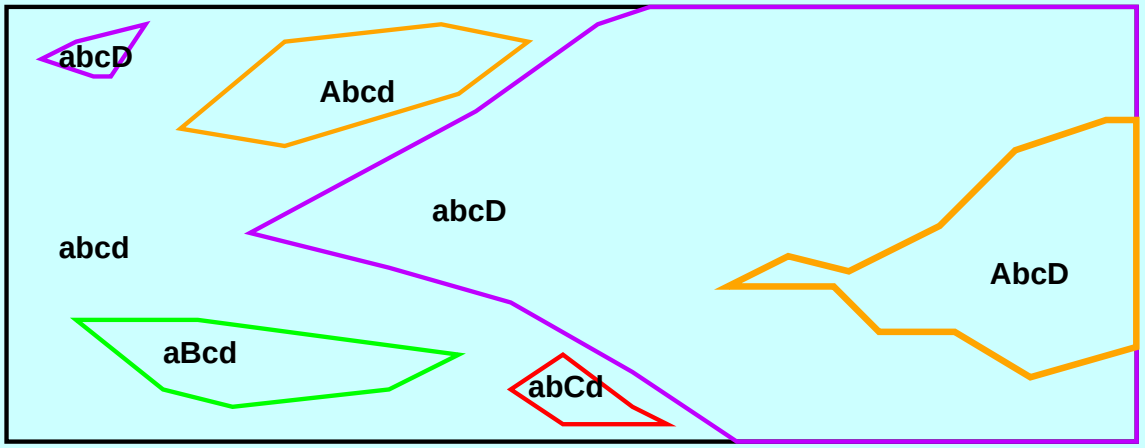
A. H. Sturtevant

4. Michod and Bernstein's argument that recombination is not needed for long-term evolutionary reasons, but is a byproduct of a system for repairing double-stranded breaks in DNA.

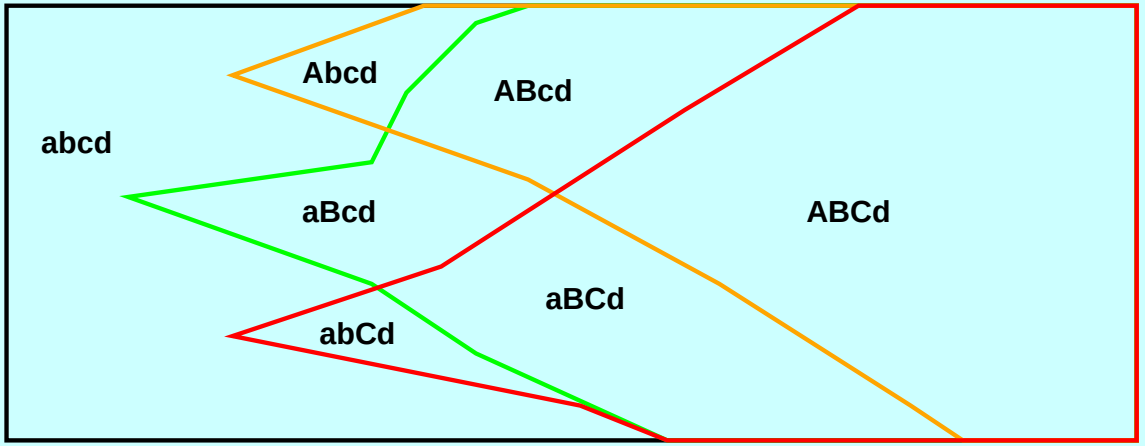
Many other explanations reduce to one or another of these (e.g. Williams's "sibling competition" scenario or Bell's "tangled bank" scenario). They are in effect biological scenarios in which these combinations of evolutionary forces act.

# The Fisher-Muller theory

no recombination




with recombination



# The Sturtevant-Mather argument

**Suppose that in one period the population favors haploid genotypes AB and ab:**

| | |
|---|---|
| **AB** | **1.0** |
| **Ab** | **0.9** |
| **aB** | **0.9** |
| **ab** | **1.0** |

**Then the population will, if there is no recombination, become composed almost exclusively of AB and ab genotypes:**


**AB and ab haplotypes are eliminated by natural selection**

**but will not become so well−adapted if there is recombination:**


**Ab and aB are continually produced by recombination in this case**

# (cont'd)

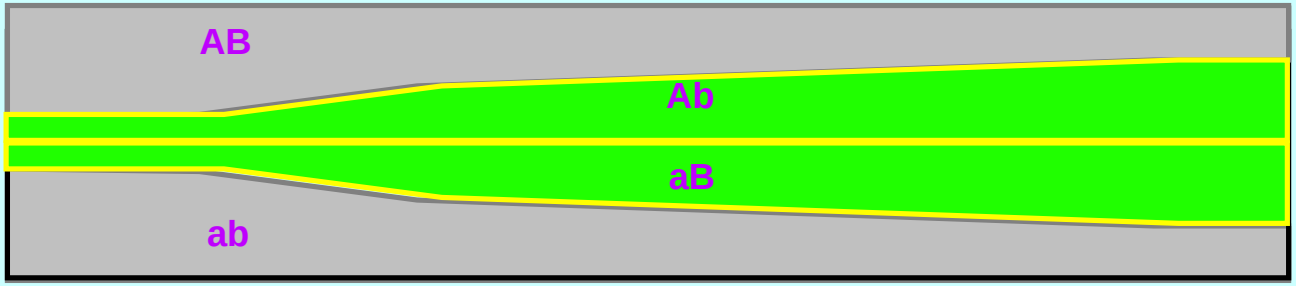
**... but in another period soon after, selection favors Ab and aB:**

| | |
|---|---|
| AB | 0.9 |
| Ab | 1.0 |
| aB | 1.0 |
| ab | 0.9 |

**the population without recombination will have a hard time getting Ab and aB**

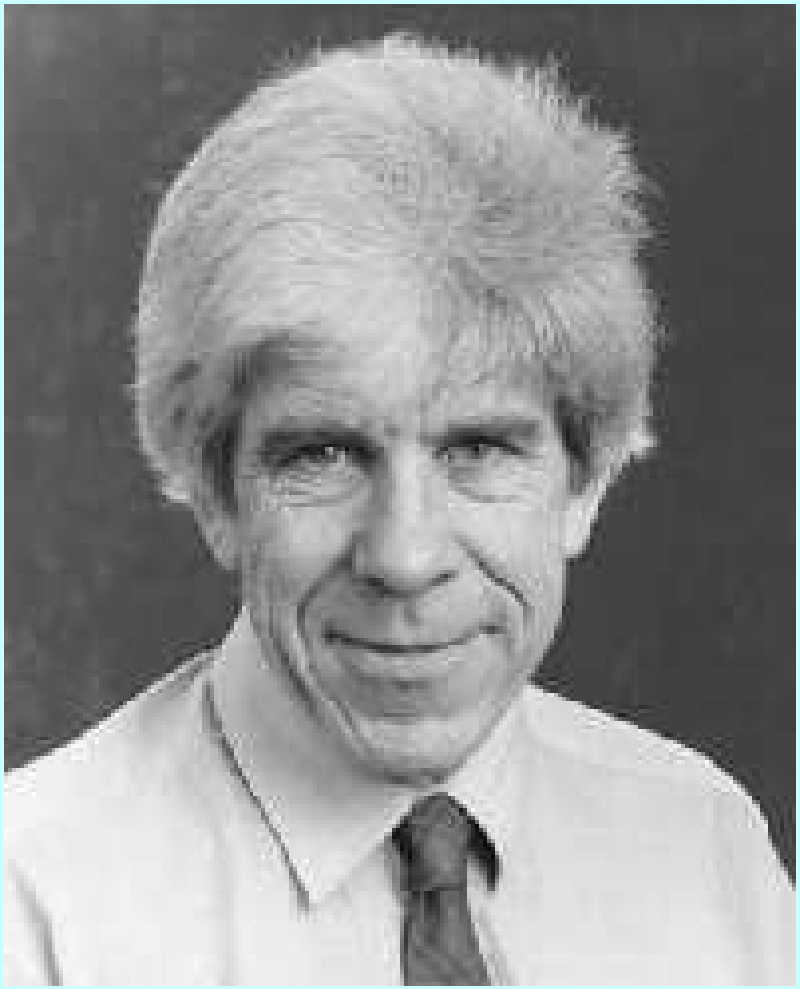

**Ab and ab are not created by recombination in this case**

**but the case with recombination is better adapted during this period**




**Ab and aB are re−introduced by recombination in this case**

# W. D. Hamilton (1936-2000)



# Hamilton's scenario for the Sturtevant-Mather mechanism

or: "Sex and parasites"

Suppose there are two kinds of parasites:

| Genotype | Parasite #1 | Parasite #2 |
|---|---|---|
| AB | grows | can't grow |
| Ab | can't grow | grows |
| aB | can't grow | grows |
| ab | grows | can't grow |

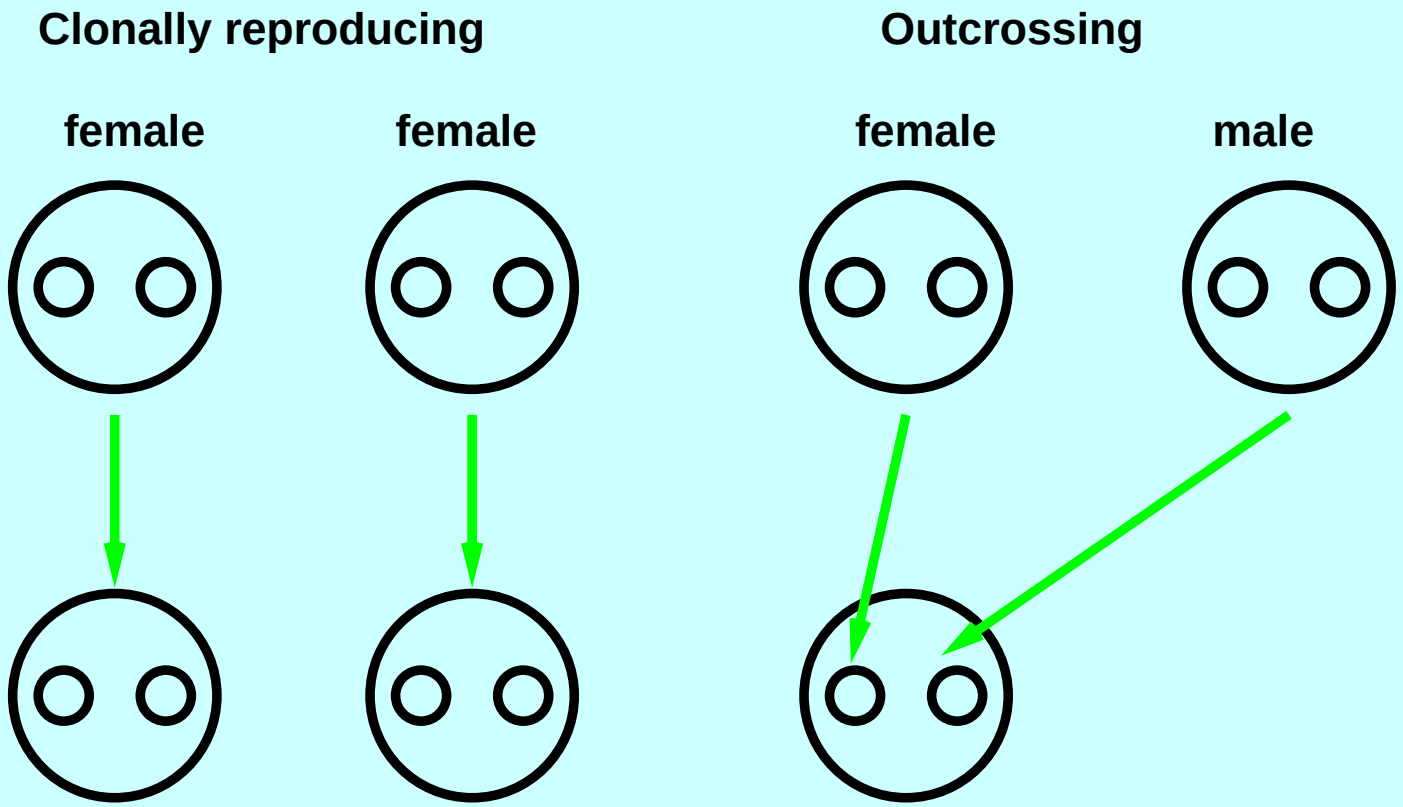
Then when parasite #1 is widespread and Parasite #2 is rare, AB and ab are favored. Once they become common, Parasite #2 spreads and Parasite #1 declines.

Then Ab and aB are favored. As they become common Parasite #2 declines and Parasite #1 spreads.

This provides a biological scenario for the Sturtevant-Mather mechanism.

# John Maynard Smith (1920-2004)

# The cost of sex




Note what has happened: where there were equal numbers of the two kinds of individuals before, now there are 2/3 of the clonal reproducers. Basically the outcrossers are wasting half their offspring producing males.

(If they are monoecious, the 50:50 sex ratio principle still means that they waste half their reproductive resources on male gametes).