Evolution of genetic systems

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How well can we explain the genetic system?

Very well

- Sex ratios of 1/2 (C. Duesing, 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)

Poorly

- Diploidy
- Mutation rates
Sex determination systems

1) XX − XY

- Male (♂): XY
- Female (♀): XX

- many dioecious angiosperms
- many animal species
- most vertebrates

2) ZW − ZZ

- Male (♂): ZZ
- Female (♀): ZW
- some flatworms
  - crustaceans
  - insects especially lepidopterans
diptera (some)
  - some fish, amphibians, lizards
  - most birds
More sex determination systems

3) $XX - XO$

<table>
<thead>
<tr>
<th></th>
<th>X</th>
<th>O</th>
</tr>
</thead>
<tbody>
<tr>
<td>♀</td>
<td>XX ♀</td>
<td>XO ♂</td>
</tr>
<tr>
<td>X</td>
<td>XX ♀</td>
<td>XO ♂</td>
</tr>
</tbody>
</table>

many insects

4) $XX - XY_1 Y_2$

<table>
<thead>
<tr>
<th></th>
<th>X</th>
<th>$XY_1 Y_2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>♀</td>
<td>XX ♀</td>
<td>$XY_1 Y_2$ ♂</td>
</tr>
<tr>
<td>X</td>
<td>XX ♀</td>
<td>$XY_1 Y_2$ ♂</td>
</tr>
</tbody>
</table>

e.g. the Muntjak deer
Yet more sex determination systems

5) Arrhenotoky (haplo–diploid sex determination)

\[ \begin{align*}
\varnothing & \quad \text{haploid no sperm} & \text{hymenoptera (ants, bees, wasps)} \\
\varnothing & \quad \text{haploid gametes} & \text{thysanoptera (thrips)} \\
\& & \quad \text{diploid } & \text{mites and ticks} \\
\& & \quad \text{haploid } & \text{rotifers}
\end{align*} \]

6) Environmental sex determination

more \( \varnothing \varnothing \) if better nutrition nematodes

colder temperature lizards, alligators

hotter temperature most turtles

extreme temperatures snapping turtles, crocodiles
7) Sequential hermaphroditism
(start life as one sex, usually male, and switch later)

    oysters, shrimp, some fish

8) Self-sterility systems
In some angiosperm plants, multiple alleles which allow pollen to succeed only
if it does not contain any allele at that locus which is found in the
female parent (gametophytic self-incompatibility) or the male does not
contain any allele at that locus found in the female
(sporophytic self-incompatibility)
The evolution of the sex ratio

In a work usually mistakenly attributed to R. A. Fisher (1930), 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).

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

16 males

8 females

50% of future gene pool

50% of future gene pool
Numerical example

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

<table>
<thead>
<tr>
<th></th>
<th>females</th>
<th>males</th>
</tr>
</thead>
<tbody>
<tr>
<td>aa</td>
<td>100,000</td>
<td>50,000</td>
</tr>
<tr>
<td>Aa</td>
<td>100</td>
<td>100</td>
</tr>
</tbody>
</table>

Frequency of A among everybody = \( \frac{200}{300,400} \approx 0.00066578 \)

Frequency of the A allele (counting copies of genes)

- among females = \( \frac{100}{200,200} = 0.0004995 \) increase!
- among males = \( \frac{100}{100,200} = 0.000998 \)

The frequency in the next generation is the average of
the frequency among males and the frequency among females: \( 0.00074875 \)

When males are rare, a male offspring will have more descendants
When females are rare, a female offspring will have more descendants
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 males 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:

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

The population is evolving its way to extinction!
A meiotically driven way evolves to extinction

![Graph showing the evolution of genetic systems](image-url)
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:

This one has no 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.
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.

2. It breaks down random linkage disequilibrium 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.

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.
John Maynard Smith
The cost of sex

Clonally reproducing

Outcrossing

Evolution of genetic systems – p.17/23
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:

\[
\begin{align*}
AB & : 1.0 \\
Ab & : 0.9 \\
aB & : 0.9 \\
ab & : 1.0
\end{align*}
\]

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

but will not become so well-adapted if there is recombination:
... but in another period soon after, selection favors Ab and aB:

\[
\begin{align*}
AB & \quad 0.9 \\
Ab & \quad 1.0 \\
aB & \quad 1.0 \\
ab & \quad 0.9
\end{align*}
\]

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:

<table>
<thead>
<tr>
<th>Genotype</th>
<th>Parasite #1</th>
<th>Parasite #2</th>
</tr>
</thead>
<tbody>
<tr>
<td>AB</td>
<td>grows</td>
<td>can’t grow</td>
</tr>
<tr>
<td>Ab</td>
<td>can’t grow</td>
<td>grows</td>
</tr>
<tr>
<td>aB</td>
<td>can’t grow</td>
<td>grows</td>
</tr>
<tr>
<td>ab</td>
<td>grows</td>
<td>can’t grow</td>
</tr>
</tbody>
</table>

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.
How it was done

This projection produced
- using the \texttt{prosper} style in \LaTeX{},
- using \LaTeX{} to make a \texttt{.dvi} file,
- using \texttt{dvips} to turn this into a Postscript file,
- using \texttt{ps2pdf} to mill a PDF file, and
- displaying the slides in Adobe Acrobat Reader.

Result: nice slides using freeware.