Asexual reproduction by parthenogenesis is efficient
Summary

- costs of sexual reproduction
- benefits of sexual reproduction
  - reminder about levels of selection
- population benefits
  - group selection
  - evolvability
  - Muller's ratchet
  - mutational deterministic hypothesis
- individual benefits
  - the red queen
- selfish gene benefits
- a pluralist approach

Costs of sexual reproduction

- the twofold cost of sex
Costs of sexual reproduction

- asexual reproduction by parthenogenesis is extremely efficient
  - if parthenogenetic offspring have the same fitness as sexual offspring, then parthenogens replace sexuals within a few generations
  - but apparently that does not happen, since sex is clearly widespread throughout the animal and plant kingdoms
  - so there must be some benefits of sexual reproduction that are able to compensate these costs

The fate of a sexual population with $10^6$ individuals into which one parthenogenetic female is introduced

Costs of sexual reproduction

- sex is actually costly in more than one way (Lewis 1987)
  - recombination (disrupts adapted gene combinations)
  - cellular-mechanical costs (time costs of meiosis, syngamy, and karyogamy)
  - fertilisation (many wasted gametes and increased predation risks)
  - genome dilution (cost of males, twofold cost of sex)
  - sexual selection (wasteful competition and specialisation)

See also Lehtonen et al. 2012 for a more recent review
Asexual reproduction by budding is also highly efficient

A polychaete of the species *Myrianida pachycera*, Photo by Greg Rouse

Benefits of sexual reproduction

Table 7.1 Theories of the evolution of sex

1. Selection favours sexual populations
   (a) Sexual populations can evolve more rapidly
   (b) Asexual populations accumulate deleterious mutations

2. Selection favours individuals that reproduce sexually
   (a) It pays an individual to produce a variable progeny (the lottery model)
   (b) Sex makes repair of damaged DNA easier
   (c) Even within a population, selection may favour sexual individuals for the reasons under (1) above: in a changing environment, their offspring may be better adapted to the new circumstances, or, in an unchanging environment, their offspring may have fewer harmful mutations

3. Selection favours genes that cause individuals to undergo sexual fusion (or to produce gametes that fuse) because then a gene present in one of the fusing cells can transfer to the other.

from Maynard Smith & Szathmáry 1999
Reminder: levels of selection

- selection can in theory act on many different levels
  - for example, at the level of the gene, the individual, and/or the group
- gene-level selection
  - although sex could well have originated as a result of the action of a selfish gene, this is unlikely to explain sex in extant organisms
- individual-level selection
  - we have already seen that selection often appears to act primarily on the individual level; why should sex be an exception?
- group-level selection
  - can occur if the migration rates are low and if there are frequent extinctions of local populations
    - low migration rate allows for differences between groups to emerge
    - local extinction allows more productive groups to spread to new populations
  - however, this type of selection is only effective over relatively long time periods

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Population benefit: group selection

- since there usually is no gene flow, the competition between sexuals and parthenogens is similar to the competition between species
  - but this is not true for parthenogenetic hermaphrodites that may still be able to donate sperm ('infectious parthenogenesis')
- to maintain sexuals, imagine a metapopulation structure where
  - locally originating parthenogens tend to out-compete the ancestral sexuals in the short-term
  - but such parthenogenetic populations will have a higher long-term risk of population extinction
  - empty patches are preferentially colonised by individuals from neighbouring sexual populations (but parthenogens may actually be good at colonising!)
- for this scenario to maintain sexual reproduction we require long-term reasons why sexual populations tend to die out less often than parthenogenetic populations

Population benefit: evolvability

- if the environment changes rapidly, sex allows for faster evolution
  - because it makes it easier to bring independent beneficial mutations (such as $a \rightarrow A$, $b \rightarrow B$, and $c \rightarrow C$) together in the same individual ($ABC$)
Population benefit: evolvability

- but does the environment actually change sufficiently rapidly?
- the abiotic environment does not usually change that rapidly
  - probably more often over timeframes of hundreds to thousands of generations, rather than tens of generations
  - moreover, migration and range shifts allows populations to follow slow trends in, for example, the climate conditions
- but the biotic environment can change extremely rapidly
  - due to coevolutionary arms-races with competitors, predators or parasites
  - so it is probably these environmental changes that are particularly important

Population benefit: removing mutations

- sexual reproduction can help to avoid the accumulation of deleterious mutations
  - assume that at the A locus individual 1 carries the deleterious mutation $a$ and at the B locus individual 2 carries deleterious mutation $b$
  - sexual reproduction allows these two individuals to produce some offspring that carry no deleterious mutations at either locus ($AB$)
  - this is sometimes called the ‘engine-and-gearbox’ model
  - however, this benefit comes at a cost of also producing an equal amount of offspring that carry both of the deleterious mutations ($ab$)

- so we need to think more deeply about the potential role of deleterious mutations, and how selection can act to remove them from a population
Reminder: the distribution of mutational classes

• individuals can be expected to fall into distinct mutational classes that follow a Poisson distribution
• the mean (and variance) of the expected distribution at mutation-selection balance is \( \mu = u/s \)
  - \( u \) is the mutation rate per genome and generation
  - \( s \) is the selection acting against a deleterious mutation
  - a large \( u \) pushes the distribution to the right and a large \( s \) pushes it to the left
• the fitness of an individual with \( i \) mutations therefore is \( (1-s)^i \)
  - here we initially assume that each deleterious mutation has the same effect, but we later relax this assumption (e.g. by permitting so-called epistasis)
• the probability for the least mutated class is \( P_0 = e^{-\mu} \)
  - this determines the probability to sample from the least mutated class
  - if \( u \) is large and \( s \) is small, then the probability for the least mutated class is small, and so we expect very few individuals to be in this least-mutated class

Reminder: the distribution of mutational classes

• the mutational classes are expected to follow a Poisson distribution

- \( u \) mutation rate
- \( s \) selection strength
- \( \mu \) mean and variance
- \( N \) population size
- \( P_0 \) probability for least mutated class
- \( n_0 \) individuals in least mutated class

example 1:
\( u=0.5, s=0.25, \mu=0.5/0.25=2 \)
\( P_0=e^{-\mu}=e^{-2}=0.135 \)
with \( N=1000 \)
\( n_0=NP_0=135 \)

example 2:
\( u=0.5, s=0.1, \mu=0.5/0.1=5 \)
\( P_0=e^{-\mu}=e^{-5}=0.0067 \)
with \( N=1000 \)
\( n_0=NP_0=6.7 \)

but, with \( N=20000 \)
\( n_0=NP_0=134 \)
Population benefit: avoiding Muller’s ratchet

- sexual reproduction can prevent the stochastic loss of the least-mutated class of individuals (i.e. of the currently ‘best’ or least mutated genotype)
  - in asexuals such a genotype is lost forever unless the exact back-mutation occurs (which is highly unlikely)
  - Muller’s ratchet acts more rapidly in small populations (if $n_0 < 10$, then the ratchet operates very quickly)

![Image](image_url)

Population benefit: mutational deterministic hypothesis

- but can deleterious mutations also lead to an advantage of sexual reproduction in very large populations?
- this only works if such mutations have worse effects when they occur in combination, compared to what would be expected given the sum of their individual effects
  - i.e. these mutations need to act synergistically (they need to show so-called synergistic epistasis)
- the basic idea of this hypothesis is that each (mutation-linked) death of an individual tends to remove more mutations than one would expect probabilistically
Reminder: epistasis

- epistasis occurs when the effect of an allele at one locus depends on an allele at another locus in the genome
- so the genetic effects behave as follows
  - $G_{AB} = G_{Ab} + G_{aB}$ if the alleles act additively
  - $G_{AB} > G_{Ab} + G_{aB}$ if the alleles show synergistic epistasis
  - $G_{AB} < G_{Ab} + G_{aB}$ if the alleles show antagonistic epistasis
- for example
  - the fitness difference between genotype $G_{ab}$ and $G_{Ab}$ is +2 units
  - the fitness difference between genotype $G_{ab}$ and $G_{aB}$ is +3 units
  - when $G_{Ab} + G_{aB} = +5$ units, then the alleles act additively
  - when $G_{Ab} + G_{aB} > +5$ units, then the alleles show synergistic epistasis
  - when $G_{Ab} + G_{aB} < +5$ units, then the alleles show antagonistic epistasis

Population benefit: mutational deterministic hypothesis

- so do deleterious mutations actually show synergistic epistasis?
  - note that we consider fitness effects of deleterious mutations, so in this case synergistic epistasis means the effects are more negative than expected

Hypothetical data

![Hypothetical data graph](image1)

Antagonistic/positive
Linear/additive
Synergistic/negative

Actual data

![Actual data graph](image2)

Figure 1 | Types of directional epistasis for deleterious mutations. Three hypothetical relationships between fitness (log scale) and number of deleterious mutations are plotted. All relationships depict have the same mutational robustness ($W_{1} = 0.78$) but different directions of epistasis: negative epistasis (plain line, concave downwards; $1 - \beta < 0$), no directional epistasis (bold, straight line; $1 - \beta = 0$) and positive epistasis (dashed line, concave upwards; $1 - \beta > 0$).

Figure 2 | Observed effect of increasing the number of deleterious mutations on fitness in the bacterium, E. coli. Each point represents the average fitness value for 75 different genotypes carrying one, two or three insertion mutations. Error bars indicate standard errors, using the jack-knife method. The solid line shows the best fit of a log-linear (multiplicative) model to the data. A model with synergistic epistasis, which includes an extra quadratic term, does not yield a significant improvement in the fit to the data.

from Azevedo & al. 2006  from Elena & Lenski 1997
Population benefit: mutational deterministic hypothesis

- with recombination
  - individuals on average have 3 mutations
    - which are randomly distributed
  - truncation selection at ≥5 mutations
    - kills 15% of the individuals
    - leads to an average of 2.45 mutations
  - individuals acquire new mutations
    - probability of 0.55 (new distribution)
  - recombination redistributes mutations
    - reestablishes the original distribution

- without recombination
  - truncation selection at ≥5 mutations
    - kills 55% of the individuals
    - all remaining have 4 mutations
    - probability of 0.55 to get a new mutation
    - reestablishes the original distribution

Figure 12.9 Number of mutations per individual in populations with (left) and without (right) recombination. From Maynard Smith 1998
Population benefit: summary

- group-level selection may have been important for the maintenance of sex
- the phylogenetic distribution of parthenogenesis largely supports this
- an exception are the bdelloid rotifers
  - called ‘ancient asexual scandals’ by Judson & Normark (1996)
- however, this scenario requires that the origin of parthenogenesis is a rare event
  - which it probably is not
- and species that exhibit facultative or cyclical parthenogenesis (or other forms involving asexual proliferation) cannot be explained in this way

Benefits of sexual reproduction

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from Maynard Smith & Szathmáry 1999
Individual benefit: the lottery model

- sex may be favoured if the environment is highly unpredictable
- asexual reproduction is like buying 100 identical lottery tickets, whereas with sexual reproduction you can buy 50 different tickets
- can explain the benefits of sex in rather exceptional circumstances (i.e. it is possible to build a model that works)

Individual benefit: DNA repair

- sex may help to repair DNA damage
  - DNA damage is not the same as DNA mutation
  - DNA mutations often cannot be detected by the cell, while DNA damage can
- repair of single-strand damage is often easy (depends on tdamage)
- repair of double-strand damage requires diploidy, but not sex
Individual benefit: the Red Queen

- sex may be favoured in a rapidly changing environment, but could it change fast enough to favour a different genotype every generation?
- coevolving parasites could lead to negative frequency-dependent selection, where initially rare resistant host genotypes spread
- as they spread, they may be tracked by initially rare parasite genotypes, reverting rare and frequent, and leading to cycling

Individual benefit: the Red Queen

- so this process is expected to involve mutual coevolution of host and parasite

following Lively & Jokela 1995
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     fewer harmful mutations

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   ✓ gametes that fuse) because then a gene present in one of the fusing cells can transfer to
   the other.

A pluralist approach

- all of these hypotheses have some problems
- Muller’s ratchet only works very slowly in large populations
  - and populations are often very large
- the mutational deterministic hypothesis requires both a high
  mutation rate and synergistic epistasis
  - considerably more than one mutation per genome and generation, which seems
    more than what is found in many species
  - moreover, evidence for synergistic epistasis is not very strong
- the Red Queen only works well if parasites have very severe effects
  on host fitness and if the cycling occurs over the correct timeframe
  - some parasites clearly do have strong effects, but are they strong enough, and do
    the timeframes match?
  - can clonal diversity in a population possibly offer the same benefits as sex?
- how do these hypothesis interact? do they support or hinder each
  others effects?
A pluralist approach: the MDH and Muller's ratchet

- the mutational deterministic hypothesis (MDH) can slow down or even halt Muller's ratchet, by removing the right tail of the distribution, leading to more individuals in least mutated class

\( u \) mutation rate
\( s \) selection strength
\( \mu \) mean and variance
\( N \) population size
\( P_0 \) probability for least mutated class
\( n_0 \) number in least mutated class

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A pluralist approach: parasites and Muller’s ratchet

- Muller's ratchet can help the Red Queen
  - clonal diversity can potentially offer similar advantages under the Red Queen as sexual reproduction
  - but co-adapting parasites can lead to fluctuations in (clonal) population size, thereby greatly speeding up Muller’s ratchet

FIG. 3. Population dynamics for monotypic asexual lineages in competition with sexual populations in the presence of either parasites or mutation, or both. a. Parameters for these runs included a mutation rate \( U \) of 0.5 per genome per generation, selection coefficient \( (s) = 0.025 \), probability of parasite transmission \( (f) = 0.5 \), and effect of parasites on host fitness \( (E) = 0.5 \). Note that for mutation only, the clone fixes in a population of 1,000 individuals in less than 50 generations; the clone then undergoes “mutational meltdown” (in the sense of Lynch and Gabriel\(^{14}\)), beginning at about generation 480. For parasites only, the clone also fixes in about 50 generations, but it does not undergo the meltdown. Finally, for mutations combined with parasites, the clonal population does not replace the sexual population, but rather it oscillates: Each oscillation increases the mutational load through the action of Muller’s ratchet, and there is a tendency for the lowest point of the oscillation to decline with time. Finally, the clone begins the mutational meltdown (see ref. 16) at about generation 220, and it is quickly eliminated from the population. b. As for a, except that the mutation rate \( U \) has been increased from 0.5 to 1.0. Note the more rapid extinction of the asexual lineage as compared with a.

from Howard & Lively 1994; note that panels a and b were swapped in the original article
A pluralist approach: parasites and the MDH

- the Red Queen can help the mutational deterministic hypothesis
  - one problem is that recently emerged parthenogenetic lineages are likely to have a relatively low mutational load, because they have just originated from sexuals
  - so it may take many generations for mutations to accumulate (and for mutation-selection balance to be reached), giving an initial advantage to the parthenogens
  - but frequency-dependent selection can slow down invasion of parthenogens, giving more time to achieve mutation-selection balance
  - and coevolving parasites may reduce the number of mutations that are required to make asexuals less competitive than sexuals

![Graphs showing number of mutations per individual in populations with and without recombination.](image)

Summary

- costs of sexual reproduction
- benefits of sexual reproduction
  - reminder about levels of selection

- population benefits
  - group selection
  - evolvability
  - Muller’s ratchet
  - mutational deterministic hypothesis

- individual benefits
  - the red queen

- selfish gene benefits

- a pluralist approach

from Maynard Smith 1998
Literature

- **Mandatory Reading**

- **Suggested Reading**

- **Books**