Why reproduce sexually if you don't have to?

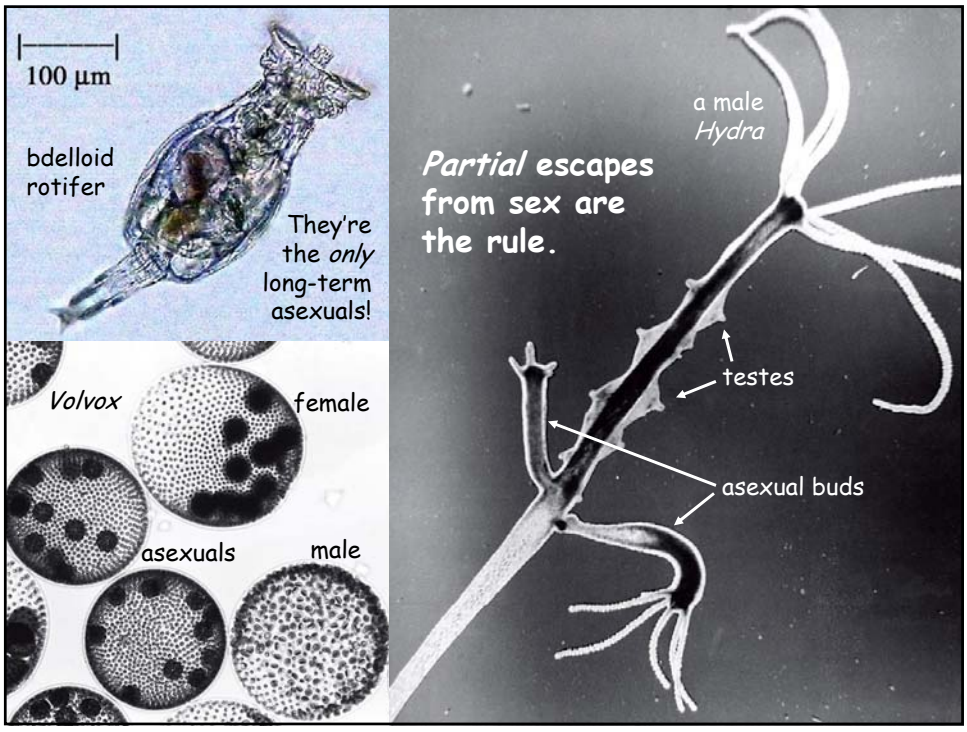
This female aphid is having babies parthenogenically.

But later in the season, the asexual females will make sexual males and females.

Why?

Anthro/Biol 5221, 8 December 2008


But they haven't been so for long.
(Look at those showy flowers!


100 µm
bdelloid rotifer
They're the *only* long-term asexuals!
Volvox
female
asexuals
male
Partial escapes from sex are the rule.
a male *Hydra*
testes
asexual buds

The cost of sex I: mom's genes are diluted by dad's

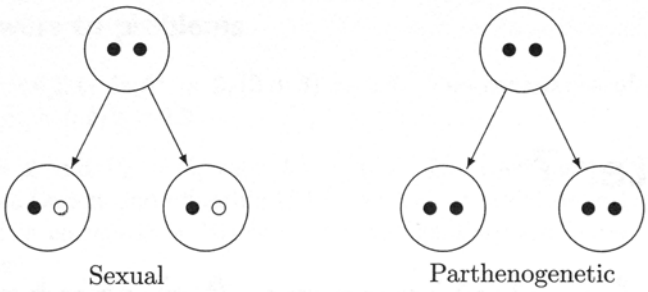
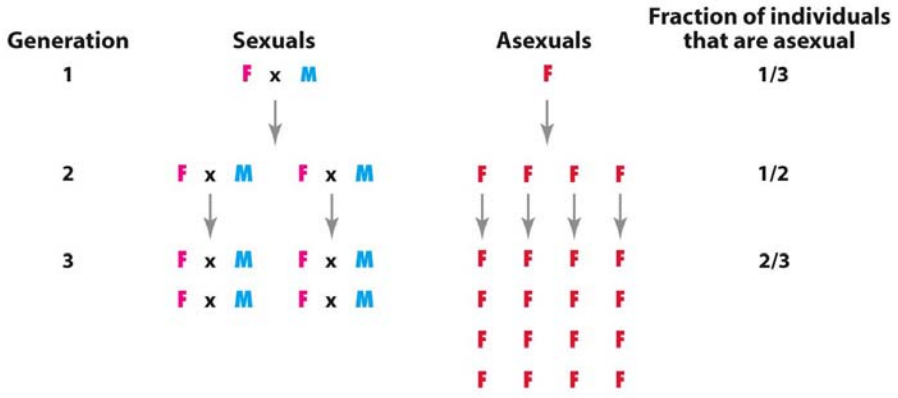
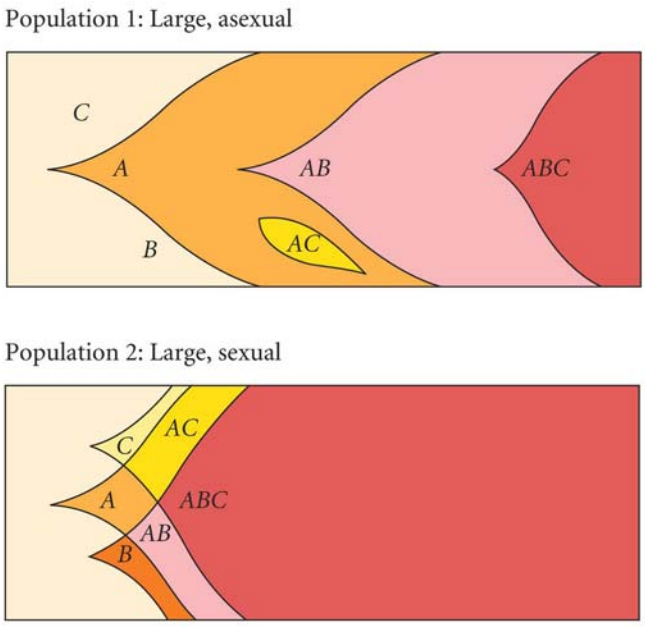


Figure 7.1: A comparison of the consequences of sexual versus parthenogenetic reproduction. The female parent is illustrated in both cases. The filled circles in the offspring come from the mother; the father's genetic contribution is indicated by the open circles in the sexual species.

The cost of sex II: parthenogens out-reproduce sexuals!



Benefit I (Crow & Kimura): sexual populations adapt more rapidly



But not if they're small!

(because the first fixation will be finished before the second one begins).

Population 3: Small, asexual

Population 4: Small, sexual

Asexual diploids adapt slowly (needing *two* mutations/substitution)

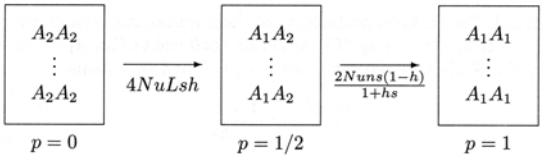


Figure 7.2: Evolution in parthenogens.

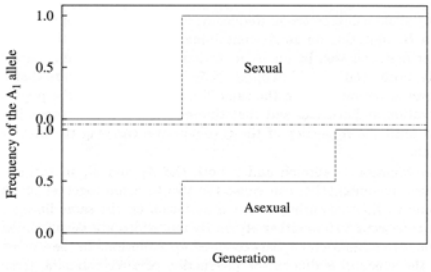
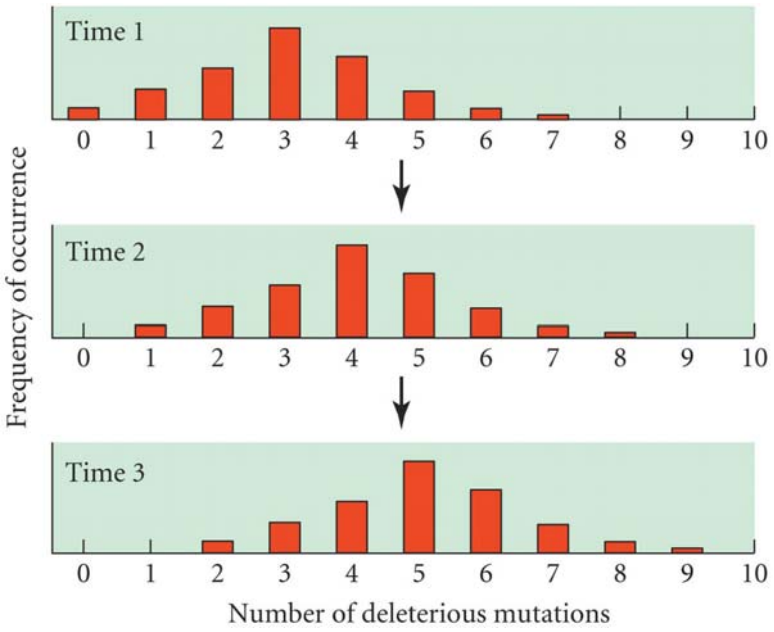


Figure 7.3: The trajectory of allele frequencies under directional selection in asexual and sexual species. The vertical dashed lines indicate transient dynamics that are not part of the model.

But there's no need to be diploid, and many organisms aren't!

ALL asexuals will suffer the effects of "Muller's Ratchet"



Why does the ratchet turn?

Remember the mutation-selection balance for an infinite population?

We assumed that an individual with i mutations has fitness $W_i = (1-hs)^i$.

Then at equilibrium, $W\text{-}bar = e^{-U}$ and $Load = 1-e^{-U}$, for any $s!$ (G., p. 72)

The Muller's Ratchet model also assumes multiplicative fitnesses.

Without sex, the distribution of i at equilibrium is Poisson, with mean

$m = U/hs$. (Gillespie, p. 177)

If m is large, there will be very few individuals with zero mutations:

$p_0 = e^{-m}$

Thus in a *finite* population, the class of "unloaded" individuals will disappear, and then the next-least-loaded, and so on.

In general, the least-loaded class will be *rare*, hence vulnerable to loss.

Thus it's *another* form of drift that's potent even in huge populations!

(because the least-loaded chromosomal class is *rare*).

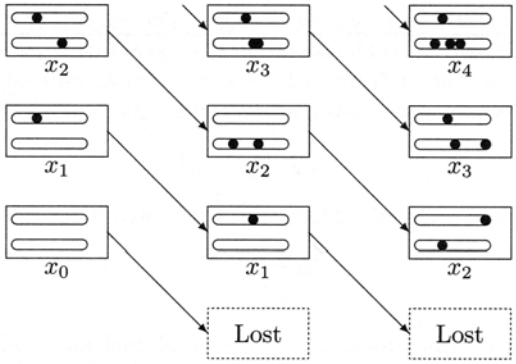


Figure 7.4: An illustration of two clicks of Muller's ratchet. The boxes represent the class of individuals with i deleterious mutations. The two chromosomes within each box represent typical individuals. The number of generations between each column of boxes depends on the efficacy of genetic drift.

Without sex, things just keep getting worse, and worse, and worse…

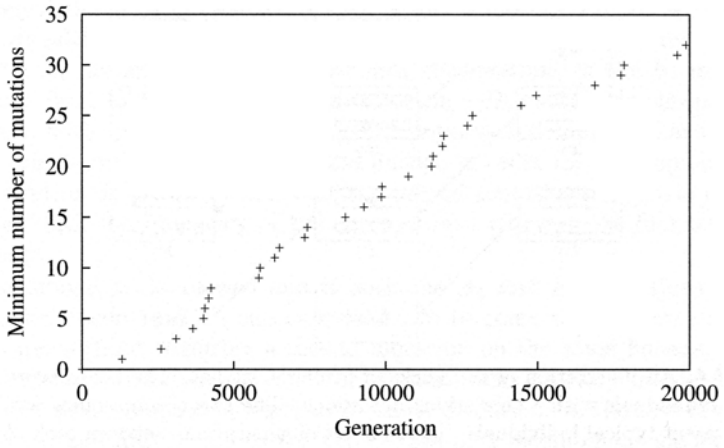


Figure 7.5: Simulation of Muller's ratchet with parameters $U = 0.1$, $hs = 0.1$ and $N = 100$.

But recombination provides an escape from the ratchet.

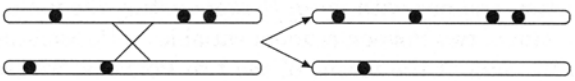


Figure 7.6: An illustration of the production of a chromosome with fewer deleterious mutations than in either parental chromosome because of crossing-over. The filled circles represent deleterious mutations.

Chromosomes better (and worse) than any in the current population can be made by crossing over in meiosis.

Thus sex promotes both improvement and purging!

But is the effect strong enough *in the short term* to prevent the spread of an asexual mutant with a 2x reproductive advantage?

Kondrashov's Hatchet:

Sex is more efficient if "synergistic epistasis" amplifies the penalty for having more than an average number of bad mutations.

In theory, this could provide a two-fold benefit of sex.

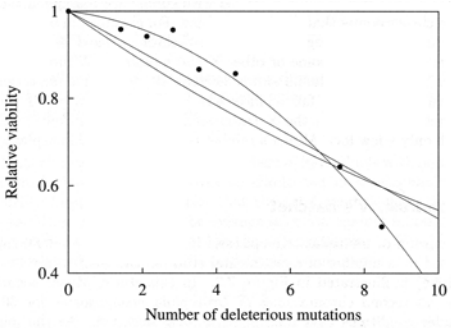
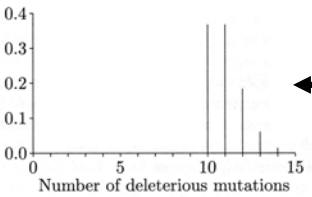


Figure 7.7: The relative viability as a function of the inferred number of homozygous deleterious mutations in *Drosophila melanogaster*. The upper concave curve is a quadratic synergistic model, the middle straight line is an additive model, and the lower convex curve corresponds to multiplicative epistasis. The data are from Mukai (1968).

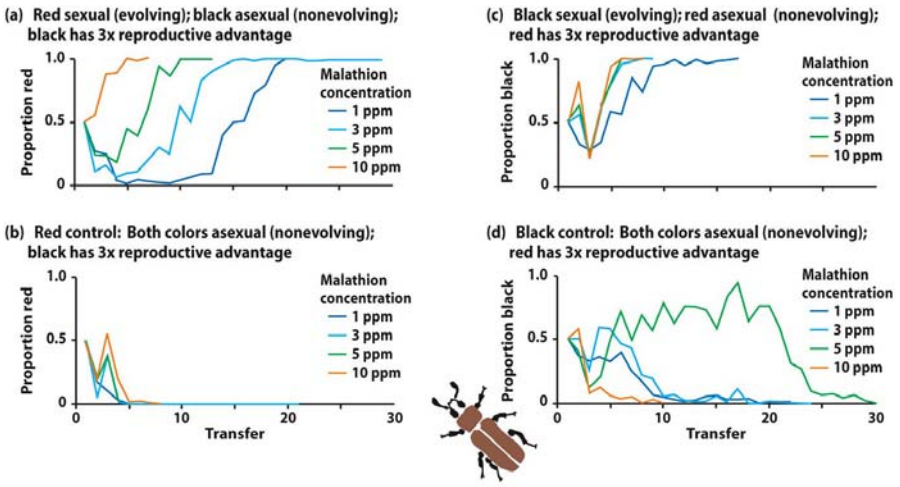


Truncation selection is the ultimate form of "synergistic epistasis". In this model, offspring with 11 or more mutations (63% of the total) die. So the asexual population is only 37% (e^{-U}) as efficient as a sexual population that keeps itself well to the left of the fitness "cliff" at k=10 bad mutations.

Figure 7.8: The frequency of asexual individuals just after reproduction and just before selection when $k = 10$ and $U = 1$.

Adaptation to changing environments also seems likely to be important

An experiment with flour beetles that simulates sex versus asex:

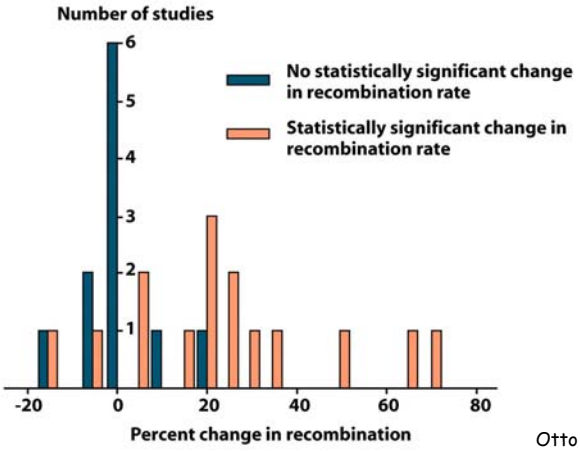


Q: What does sex do to populations of genes?

A: It reduces linkage disequilibrium.

Selection can be stalled by disequilibrium between favored and disfavored alleles (interference).

Does selection favor higher rates of recombination?

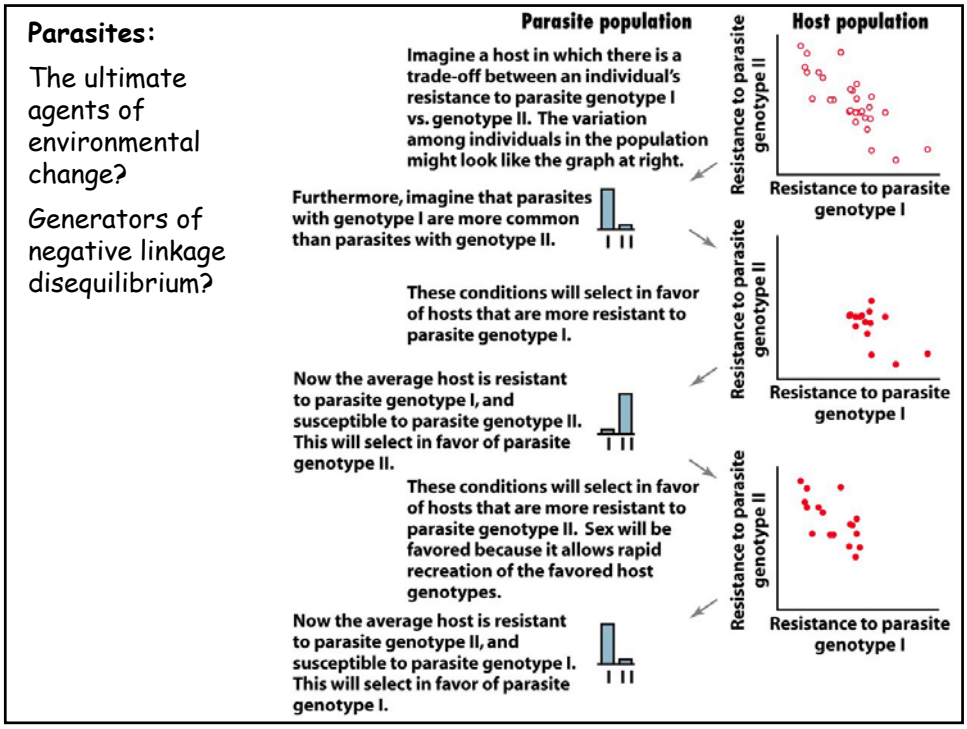


Otto & Lenormond 2002

Parasites:

The ultimate agents of environmental change?

Generators of negative linkage disequilibrium?

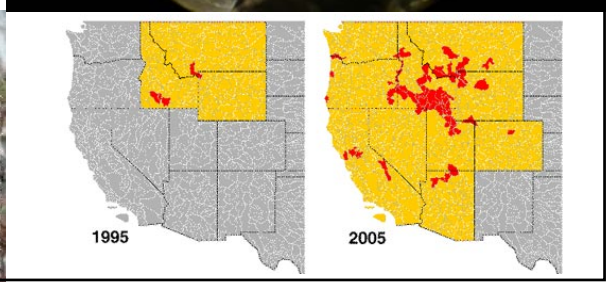


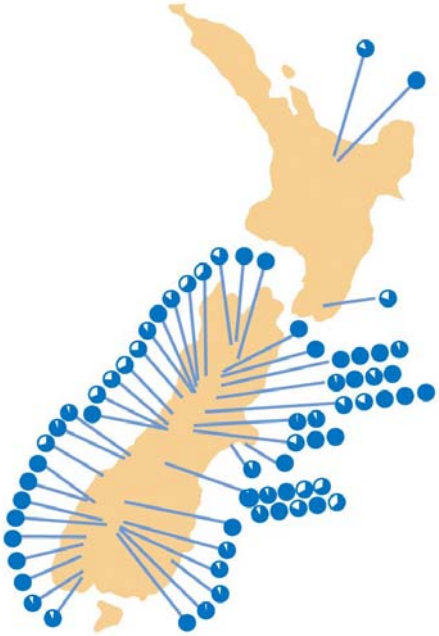
The New Zealand mud snail (*Potamopyrgus antipodarum*)

Plagued by trematodes

Sexual and asexual strains (genetically determined).

And now your neighbor.

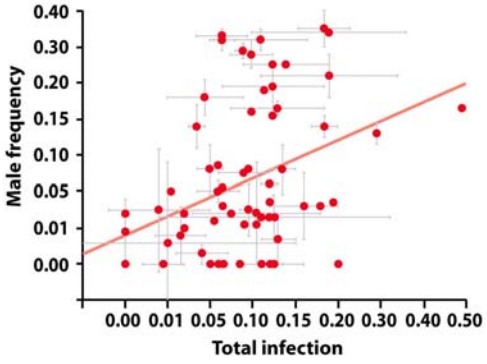




A lake with *no* males contains 100% asexual snails.

A lake with ~50% males would contain ~100% sexual snails (but none do).

The frequency of males varies greatly among lakes, as does the rate of infection by trematodes.



And sexuals more rapidly evolve resistance to their local trematodes than do asexuals.

Summary

In species without male parental care, there is a 2-fold *cost of sex*. (Or to put it the other way around, a 2-fold reproductive *advantage* of *asex*.)

Many species of multicellular eukaryotes have asexual capabilities (*e.g.*, parthenogenic life-history phases, or the ability to self-fertilize).

Given the enormous and immediate advantage of parthenogenesis, it is puzzling that more species aren't taken over completely by forms (*e.g.*, mutations) that simply delete the sexual phases of the life history!

Thus to explain the prevalence of sex we need to identify *short-term* benefits of sex that are *large enough* to offset the 2-fold cost.

Sex reduces linkage disequilibrium and creates new combinations of alleles at different loci.

This may permit faster rates of adaptation, but the benefit is unlikely to be large enough unless species are *strongly* challenged, *much of the time!*

Rapidly coevolving parasites could supply such challenges in principle, and there is evidence that they do so in at least some species.

Unconditionally deleterious mutations could also be important, or even sufficient, if they interact "synergistically" rather than multiplicatively.