

The last lecture ended with an overview of the ecological genetic models of sexual reproduction and recombination.

Here are some of the key points that I tried to make.

1. William's lottery model was the favored model from 1975 until 1982. The basic idea of the lottery model is that sexual reproduction should be favored because it increases geometric mean fitness in a temporally variable environment. Selection was assumed to be frequency and density independent (hard selection).

2. Graham Bell reasoned that under the lottery model, more Asexual reproduction should be favored in the tropics and other physically stable environments. But instead he found that sexual reproduction was most common in the tropics, and asexual reproduction, when found, was more common at high latitudes, high altitudes and other physically unstable environments (Bell 1982). The lottery model faded; it was a good idea, but it did not hold up to the data. The Tangled Bank model replaced the lottery model, because it predicted that sexual reproduction would be favored in stable habitats, like the tropics, where competition for resources would be very intense. However, parasites might also be more common in such environments, making it difficult to separate the predictions of the tangled bank and the Red Queen.

3. The Burt and Bell paper from 1987 (on recombination in mammals) was inconsistent with the tangled back, but consistent with the parasite Red Queen. Other studies got similar results (Antonovics and Ellstrand 1984; Schmitt and Antonovics 1986; Lively 1987; Kelley et al. 1988). Presently, the RQ is the most favored idea, based on the data, but it has theoretical difficulties.

#### 4. Theoretical difficulties of the Red Queen

A. Parasites have to be very virulent (kill or sterilize) in order to prevent the fixation of an asexual clone in the short-term (10s of generations). The conventional wisdom is that most parasites are not this virulent. (But note the logical error in the conventional wisdom: the theory is not about what most parasites do).

B. Parasites are simply a source of frequency-dependent selection. They could select against clones when the clones are common, but they would not select against the clones when they are rare. In fact, parasites would favor the spread of a rare clone. As such, parasite-host coevolution could result in the accumulations of clones with different genotypes, driving the sexual population to extinction.

Regarding 4A. Hamilton et al (1990) reasoned that parasites might not be sufficiently virulent by themselves, but in combination with intra-specific (within species) competition for resources they could become very virulent (Hamilton et al. 1990). They imagined a kind of rank-order truncation selection against the hosts that were infected by the most parasites. Specifically, the most-infected 7% we assumed to be unable to compete for resources and would fail to reproduce. The failure was driven by competition. These individuals would have been able to reproduce in a non-competitive world. The main point is that parasites in combination with other factors, like competition, could be very virulent, as apparently required for the theory to work. Note the combination of ideas here (infection, competition and rank-order truncation selection.)

Regarding 4B. Parasites are indeed a source of frequency-dependent selection, but under theory they should be able to drive clones through regular bottlenecks (periods of low density). Hence, parasites could speed up Muller's ratchet. Remember, we rejected the ratchet previously, as it was too slow. But if parasites could prevent fixation of a clone in the short-term, the ratchet would be given time to work. Simulation models showed that the combination of the Ratchet and the Red Queen could prevent the fixation and accumulation of clones (Howard and Lively 1994). Here again we see a combination of ideas. None of the ideas may work alone, but some in combination might do the trick (West et al. 1999). As such, the strong inference program (Platt 1964) might need some tweaking.

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Now, moving onto a new topic...

Epistasis, linkage disequilibrium, and recombination: E, D & Rec.

In the Burt and Bell (1987) paper, recombination increased with age to maturity in mammals. But, overall, recombination was highest (for a given age to maturity) in domesticated animals.

WHY?

Apparently REC was indirectly selection by breeders. REC may have helped create the combinations of traits that breeders favored, and selection for these traits indirectly selected for modifiers increasing the recombination rate.

But why is there recombination in natural populations. Remember what recombination creates it also takes away (As we will see).

This is the PARADOX OF RECOMBINATION. Why break up a good thing?

An example of favorable trait combinations...

1. Let there be a haploid host and haploid parasite
2. Let there be two loci, each with two alleles.
3. One host locus has either **A** or **a**. The other host locus has either **B** or **b**. (Remember the host is haploid).
4. One parasite locus has **A** or **a**. The other parasite locus has either **B** or **b**.

### Infection matrix

		<u>Host genotype</u>			
		<b>AB</b>	<b>Ab</b>	<b>aB</b>	<b>ab</b>
<u>Parasite Genotype</u>	<b>AB</b>	Inf.	---	---	---
	<b>Ab</b>	---	Inf.	---	---
	<b>aB</b>	---	---	Inf.	---
	<b>ab</b>	---	---	---	Inf.

This is the matching alleles model of infection. The outcome of having **A** depends strongly on whether it is in the presence of **B** or **b**. Hence, there is strong *epistasis*.

Epistasis is the interaction between alleles at different loci.

1. How is that similar to dominance?
2. How do you think epistasis would affect heritability?

Here is a formal definition of epistasis for fitness.

$$E = \ln \left[ \frac{W_{AB} W_{ab}}{W_{Ab} W_{aB}} \right]$$

$$E = \ln(W_{ab}) - \ln(W_a W_b)$$

$$E = \ln(\text{observed}) - \ln(\text{expected})$$

Now for the most ridiculous term in population genetics, **linkage disequilibrium**.

Linkage disequilibrium, D, is a statistical association between alleles at different loci, which may or may not be on the same chromosome. Specifically, a population has linkage disequilibrium if two (or more) alleles are together more often than expected by chance alone.

$$D = \text{freq}(ab) - \text{freq}(a) * \text{freq}(b)$$

D = observed freq of the ab genotype minus the expected freq of the ab genotype based on allele frequencies.

So, if there is epistasis for fitness, selection is favoring a particular combination of alleles at different loci. After selection, this genotype will be more common in the population than expected by chance alone.

Epistasis for fitness (as in the matching allele model) generates linkage disequilibrium.

(But linkage disequilibrium can also be generated by drift...)

Now... What is the effect of recombination?

In terms of linkage disequilibrium, the effect of recombination is to reduce linkage disequilibrium. In other words, recombination reduces the statistical association between alleles at different loci.

This has prompted some prominent population geneticists to suggest that the only effect of recombination is to reduce linkage disequilibrium.

Specifically, if  $D'$  is the linkage disequilibrium after recombination, then

$$D' = D(1 - r),$$

Where  $r$  is the rate of recombination.

From web site, see: Excel worksheet: the effect of selection and recombination on linkage disequilibrium

Literature Cited

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