

Note: If you draw diagrams as part of your answers, make them LARGE.

Short Problems:

1. It is intuitively reasonable that a species in which most males do not reproduce would evolve to have fewer males, as the excess males appear to be “wasted”: but this does not happen. Briefly explain why not.
2. Considering inversions which do not span the centromere, is a big inversion (in a heterozygote) worse for fertility than a small one? Why or why not?
3. Inversion heterozygotes have reduced fertility in most cases; thus, inversions are generally underdominant. Give three ways in which an inversion could none the less become fixed in a population.
4. The red/green color vision loci in humans appear to be right on the border between independent evolution and concerted evolution. Suggest one manipulation (either genetic engineering or changing the environment) that would make independent evolution more likely, and one that would make concerted evolution more likely. (Your suggestions do not need to be feasible in practice.)
5. All other things being equal, are transposable elements more dangerous to a sexually reproducing organism or an asexually reproducing (cloning) one? Explain briefly.

Long Problem:

Sex determination in a (fictional) alligator species is driven by temperature: there are no sex chromosomes. At current temperatures, 80% of offspring are female.

A new mutation  $T$  causes all offspring carrying it to be male. Recessive homozygotes  $tt$  still determine their sex by temperature as usual (80% females, 20% males). We start a large population with 800  $tt$  females, 200  $tt$  males, and 200  $Tt$  males.

1. Why didn't I specify the gender of  $TT$  individuals?
2. What is the one-generation outcome of the population described above?
3. What is the equilibrium frequency of the  $T$  allele, assuming that the effort to make male and female offspring, and the survival of male and female offspring, are both equal?
4. In a population at the equilibrium, a dominant mutation arises at the  $H$  locus, which is closely linked to  $T/t$ ; the mutation arises on a haplotype bearing  $T$ . In males, this mutation gives a 10% fitness advantage. It does nothing in females. Assuming no recombination separates  $H$  from  $T$ , what will happen to this new allele? Specifically, what is its equilibrium allele frequency, and what will the sex ratio of the population be? (This question is challenging; I'd probably write a program.)
5. The chromosome bearing the  $T/t$  locus can be considered a brand new sex chromosome. Why didn't we arrive at the standard  $XY$  system?