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

Short Problems:

1. (3 pts) 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. *There is strong selection to increase the number of whichever sex is rarer, because that sex gets a disproportionately high share of alleles into the next generation. This pushes the ratio to 50/50. The species does not calculate what would be "good for it".*

2. (3 pts) 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? *A big one is worse, because the inversion will cause fertility problems (in a heterozygote) if there is a crossover, and the chance of a crossover inside the inversion is roughly proportional to its length.*

3. (3 pts) 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. *Genetic drift could allow it to fix by chance, especially in a small population. If the inversion creates a favorable trait, selection on that trait could fix the inversion; this could also happen if by chance the inversion was linked to a favorable trait. Strong inbreeding could reduce the disadvantage by decreasing heterozygotes. The inversion could hold favorable alleles together by suppressing recombination. Or you could be in a species with adaptations that reduce the fitness cost of inversions, like Drosophila.*

4. (3 pts) 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.) *The question I was trying to ask is: how could we stop this pair of loci from evolving in concert? We could move them further apart or to other chromosomes, to reduce the chance of recombination or gene conversion. We could modify flanking sequences to make the recombination target smaller (might not work if the coding sequence is the main problem). We could invert one relative to the other, as there is less concerted evolution of inverted repeats than direct repeats. We could impose stronger selection against colorblindness, which would weed out copies that have become homogenized. How could we encourage them to evolve in concert? We could make them more similar, make more copies in the genome, or reduce selection for color vision.*

5. (3 pts) All other things being equal, are transposable elements more dangerous to a sexually reproducing organism or an asexually reproduing (cloning) one? Explain briefly.

   Plausible sounding arguments can be made either way. A sexual organism can get rid of transposons by recombining two copies of a chromosome with transposons in different places to produce a daughter chromosome with fewer transposons. It is generally diploid so an insertion is less likely to be immediately fatal by knocking out an essential gene.

   A counterargument would be that in a sexual, transposons can be targets of mispaired recombination leading to harmful chromosomal rearrangements, but asexuals do not have this problem. Also, transposons are a sexually transmitted disease and you can avoid "catching" them by not having sex; and without sex you do not risk making an offspring that inherits your transposons but not your transposon silencing genes.

   Bdelloid rotifers have very few transposons, suggesting that the first set of arguments are more important than the second. I gave credit for either. We do not have very many cases to look at so I think the question is still open.

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. (2 pts) Why didn't I specify the gender of TT individuals? *There aren't any, as both parents would have had to contribute T, but then both of them would have been males.*

2. (3 pts) What is the one-generation outcome of the population described above? *Females always transmit t, so we just have to consider the males, who are 0.5 Tt and 0.5 tt. This means that 0.25 of offspring will be Tt males. Of the remaining 0.75, 0.8 will be female (0.6) and the rest will be male (0.15). The next generation is therefore 0.25 Tt males, 0.15 tt males, and 0.6 tt females. The frequency of T went up from 0.083 to 0.125, a pretty dramatic increase. Males are rare, and T is in males, so it spreads.*

3. (3 pts) 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? *By arguments given in class, the sex ratio should be 50/50 at equilibrium, so we can just solve for the T frequency that will give this ratio. The 50% of the population that is female should be 80% of the tt population, which is therefore 0.625; the remaining 0.375 are Tt and are all males. This means the frequency of T is 0.1875. Watch out for that last step–it is tempting to think it's 0.375, but only half the alleles in Tt are T.*

   To check that this is truly the equilibrium, you can do the one-generation calculation as above and verify that the frequency does not change. Even in a weird system like this, the selection pressure is towards 50/50.

   You can also solve directly for the equilibrium by setting the difference between allele frequencies this generation and next generation to zero, and solving for the frequency of T, or by writing a program to iterate the one-generation calculation.

4. (5 pts) 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.) *I wrote a program (see end of key), calling the new form of T "R", and obtained the following frequencies at equilibrium. Note that I might have a bug– I have not tried to solve it analytically–though one student got exactly this answer analytically. (Most of the other answers involved integer numbers of animals or other approximations and aren't directly comparable, though all were similar.)*

Condition	*Prop. males*	*pT*	*pR*	*pt*
Pre-selection	*0.4889*	*0.0000*	*0.1944*	*0.8056*
Post-selection	*0.5294*	*0.0000*	*0.2059*	*0.7941*

   It is easy to see why T goes extinct as R is simply superior. It is not too surprising that the equilibrium frequency of R is higher than the one for T without R, as R has a relative advantage. But I was surprised by the behavior of the sex ratio; there are more females at birth and more males at adulthood. This has interesting implications for the biased sex ratio in humans.

   A huge booby-trap in this problem is thinking you can ignore females, but I don't think it's possible to get the selection right while doing so, as you need to divide through by the new size of the population....

5. (2 pts) 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? *Because tt are not all female, whereas in a standard XY system they should be. You could get to XY from here, though, by increasing the chance that tt are female either through environmental change, behavioral change, or genetic change.*

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# alligator.py
# probability an offspring is a male
chance_tt_male = 0.2
chance_tt_female = 1.0 - chance_tt_male

# initial population frequencies IN MALES
m_T = 0.3
m_R = 0.3
m_t = 1.0 - m_T - m_R
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# fitnesses
fit_Tt = 1.0
fit_Rt = 1.1
fit_tt = 1.0

for i in xrange(10000):

  # mate the males with females, who are 100% tt
  Tt = m_T
  Rt = m_R
  tt_male = m_t * chance_tt_male
  tt_female = m_t * chance_tt_female
  freq_of_males = (Tt + Rt + tt_male)/(Tt + Rt + tt_male + tt_female)

  # save them for later use
  preselect = [Tt, Rt, tt_male, tt_female, freq_of_males]

  # apply fitnesses
  Tt *= fit_Tt
  Rt *= fit_Rt
  tt_male *= fit_tt
  tt_female *= fit_tt

  # renormalize
  total = Tt + Rt + tt_male + tt_female
  Tt /= total
  Rt /= total
  tt_male /= total
  tt_female /= total

  freq_of_males = Tt + Rt + tt_male

  # new allele frequencies in males
  m_T = 0.5 * Tt / freq_of_males
  m_R = 0.5 * Rt / freq_of_males
  m_t = (0.5 * Tt + 0.5 * Rt + tt_male) / freq_of_males

# we are hopefully at equilibrium, so find frequencies
# these use the adult frequencies of genotypes post-selection

pop_T = 0.5 * Tt
pop_R = 0.5 * Rt
pop_t = 1.0 - pop_T - pop_R

print "Post-selection", freq_of_males, pop_T, pop_R, pop_t

# use saved values to get pre-selection frequencies

pop_T = 0.5 * preselect[0]
pop_R = 0.5 * preselect[1]
pop_t = 1.0 - pop_T - pop_R
freq_of_males = preselect[3]
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print "Pre-selection", freq_of_males, pop_T, pop_R, pop_t
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