This homework is primarily to see how much population genetics you remember from previous courses. If you find it difficult, please don't panic; we will cover all of this material in more detail during the course. Please pay particular attention to **boldface** terms as they are meant in a technical sense. Also, be sure not to round so much that the effect you are looking for disappears! (I recommend at least 4 digits after the decimal.)

Reminder: A **homozygote** has two copies of the same allele; a **heterozygote** has two different alleles.

1. Cystic fibrosis (CF) is a disease caused by a mutation at the CFTR gene. Mutant homozygotes are gravely ill and (prior to modern medical treatment) normally die before reproducing. Heterozygotes ("carriers") appear normal.

   A village was founded by 98 normal homozygotes and two heterozygotes.

   (a) What are the **allele frequencies** of the normal and disease alleles at the time of founding?

   (b) In the next generation, assuming random mating, what are the expected frequencies of the three **genotypes** (homozygous normal, heterozygous, homozygous mutant) at birth?

   (c) What are the frequencies in survivors of that generation at age 50 (assuming that all CF homozygotes die, and no one else dies)?

   (d) If no further mutations to create CF alleles occur, what would be the expected long-term fate of the CF allele?

   (e) If the village had a tiny population such as 20, would your predictions about changes in CF allele frequency be any different?

   (f) A particular village is found to have a very high frequency of CF. What are two possible explanations?

2. A salmon researcher found the following genotype frequencies at a protein-coding gene (it has two alleles, A and B):

Genotype	Frequency
AA	0.45
AB	0.05
BB	0.50

   (a) What are the **allele frequencies**?

   (b) What **genotype frequencies** would be expected in a randomly mating neutral population (i.e. what are the Hardy-Weinberg proportions)?

   (c) Suggest at least three reasons why this gene might not exhibit Hardy-Weinberg proportions.

   (d) Can you do a statistical test, based on the given data, of whether these deviations from H-W are significant? If so, how? If not, why not?

3. A mutant allele of the Duffy blood-type gene, DuffyO, grants complete immunity to one form of malaria, *Plasmodium vivax*. DuffyO has a frequency close to 100% in sub-Saharan Africa and is nearly unknown elsewhere.

   (a) Give a plausible explanation for the high frequency of DuffyO in Africa.

   (b) The area of the chromosome close to Duffy has almost no variation in sub-Saharan Africans; in non-Africans it is about as variable as the rest of the genome. As far as we can tell there are no functional genes or control elements in this area. Why is it so lacking in diversity in Africans?

   (c) *Plasmodium vivax* is currently found in Asia, Europe and the Americas, but NOT in most of Africa. Speculate on the past history of *P. vivax*. It may be helpful to know that human hosts are essential to its life cycle.