

These are for your own information only; I won't be collecting or grading them. A solution key will be available on the Web. Some are a little longer and more open-ended than actual exam questions but otherwise they should be comparable.

- As a teenager I attempted to show Mendelian segregation by crossing pure-breeding normal-wing fruit flies with pure-breeding vestigial-wing fruit flies (vestigial-wing flies have small, stubby wings and cannot fly). My first generation consisted of 100% normal-wing flies. In my second generation I saw something like this:

Phenotype	Number of flies
Normal	180
Vestigial	20

- Using the information that this is a single-gene Mendelian trait, calculate the expected flies in each category and perform a statistical test to see if the observed flies match expectations. (Note that this was a controlled cross, **not** a random population of flies.)
  - Assuming that offspring were conceived in the expected ratios, calculate the fitness of vestigial-winged flies in my experimental setup.
- Suppose that naked mole rats have one breeding female and about three breeding males per colony. A geographic region has 100 colonies of average size 80 individuals.
    - What is the census (headcount) population size of this region?
    - What is the effective population size? (It will be useful to know that if the sex ratio is unequal, the effective population size is  $4N_mN_f/(N_m + N_f)$ , where  $N_m$  is the count of breeding males and  $N_f$  is the count of breeding females.)
    - A related species of mole rat is solitary, with all individuals able to reproduce. If the two species had the same census population size, would you expect the solitary species to contain more, less, or the same amount of neutral genetic variability?
    - What advantage might naked mole rats gain by being eusocial (only a few individuals reproduce)? What disadvantage might they face?
  - In Hawaiian fruit flies, we sampled a gene of unknown function. 60% of our flies were homozygous GG; 40% were homozygous gg. Despite a lengthy search we never found any heterozygotes.
    - List as many possible explanations for this result as you can. Try to be specific.
    - Which of your explanations would still be plausible if we sampled newly laid eggs and found some heterozygotes?
  - An agricultural geneticist tried to select for resistance to leaf rust in tobacco. He started with a gene pool that clearly contained lots of genetic variability: some individuals were very susceptible, others were very resistant. He imposed strong artificial selection in favor of resistance for several generations, but there was no improvement in average resistance.
    - What are at least two possible explanations for this result?
    - For each of your reasons, is there something the experimenter can try in order to improve his results, or is it hopeless?
  - A cat breeder discovers several kittens with curled ears. She attempts to establish a new breed of curled-ear cats by selling off all normal-eared kittens and breeding only the curled-ear kittens. Ten generations later, she is frustrated to find that crosses between two of her curled-ear cats still produce 67% curled and 33% normal kittens. She measures  $V_E$  for the curled-ear trait and finds that it is nearly 0. Identical twin kittens invariably have the same kind of ears. She also notes that her breed is not as fertile as expected.
    - What is a likely explanation for her results?
    - Is there anything she could do to obtain pure-breeding curled-ear cats?

(This is a real situation. I am a bit surprised that cat breeders didn't figure it out right away.)

6. The well-known form of hemophilia is a recessive X-linked gene. Homozygous recessive females and hemizygous recessive males are gravely ill. However, there are also autosomal (not sex linked) genes which can cause hemophilia when defective.
- If we followed two human populations of the same size, one with a 10% frequency of the X-linked hemophilia allele, and one with a 10% frequency of the autosomal hemophilia allele, what would be the initial frequency of hemophiliac individuals in each population (assume H-W)? What proportion of these hemophiliac individuals would be males in each population?
  - Which population would tend to lose the harmful allele more rapidly? Why?
  - If a complete cure for hemophilia were discovered so that these alleles became selectively neutral, would there be any expected difference in the length of time the hemophilia allele would take to fix or be lost in the two populations? Why or why not? (Hint: count gene copies.)
7. In humans, BB and Bb individuals have brown eyes, and bb individuals have blue eyes. We survey 1000 Northern Europeans and find the following: 824 brown-eyed people, 176 blue-eyed people.
- If we can assume Hardy-Weinberg, what are the allele frequencies of B and b?
  - There are a number of reasons the HW assumption could be wrong. Give two possible reasons. For each one, say whether it would cause you to over-estimate the frequency of the blue allele, or under-estimate it.
8. In the social amoeba *Dictyostelium discoideum*, individual free-swimming amoebae, not necessarily related to each other, come together to form fruiting bodies with a stem and a tip. Only the amoebae in the tip reproduce. An individual amoeba can be "selfish" or "altruistic" depending on an allele at the gene *csA*. Selfish amoebae have a greater chance to end up in the tip, which increases their individual fitness. Altruistic amoebae have a greater chance to end up in the stem, which increases the fitness of the amoebae in the tip.
- Would you expect the altruism allele to be able to spread, if introduced into a population of mostly selfish alleles?
  - If fruiting bodies were made up of closely related amoebae, would this change your conclusion? Supposing that the cost of being altruistic is 50% and the benefit is 1% each to 1000 other amoebae, how closely related would the amoebae have to be, on average, to make altruism superior to selfishness? (The example is real, but these numbers are fictional.)
  - Remarkably, individual amoebae with the altruistic allele of *csA* can recognize each other (they literally stick together). This enables them to be less altruistic when surrounded by selfish amoebae, and more altruistic when surrounded by altruistic amoebae. Does this change your conclusion about whether altruism can spread?
9. In Japan, there is a strong correlation between a father's height and his sons' height. Heritability is high:  $h^2 = 0.8$ . We sampled a group of fathers and sons who were all of Japanese ancestry (with no European admixture). The fathers were all born and raised in Japan; some of the sons were raised in Japan, others were raised in California. In this study, heritability was low:  $h^2 = 0.1$ . The height of a father did not predict the height of his sons well. The mothers of these children were also Japanese with no European admixture. How can this result be explained?
10. Plants can be either zinc-sensitive or zinc-resistant based on alleles at the *z* locus. The sensitive allele, zS, is dominant over the resistant allele zR.
- On a zinc-contaminated mine site, we collect random seeds and find 17 zinc-sensitive seeds (these are either zS/zS or zS/zR) and 105 zinc-resistant seeds (these are zR/zR). Assuming that the seeds are in Hardy-Weinberg proportions, what are the allele frequencies of zS and zR?

Here are fitnesses on different types of soil. The death due to selection happens after seeds germinate but before they can flower.

Genotype	zS/zS	zS/zR	zR/zR
Fitness on zinc soil	0.5	0.5	1.0
Fitness on regular soil	1.0	1.0	0.9

- (b) If we plant these seeds on zinc soil, what genotype frequencies will we expect in the flowering adults?
  - (c) After one generation of selection, what will the new allele frequencies be?
  - (d) Suggest a hypothesis for why zS alleles are still present on this mine site.
11. Researchers find a gene for which mutant alleles in modern humans are associated with inability to speak. They wonder whether change in this gene was important in the development of human speech abilities.
- (a) One approach is to calculate  $\omega = D^N/D^S$  for this gene within modern humans. Supposing that the gene really is essential for human-like speech abilities, what general result would you expect? Why? (I.e. would  $\omega$  tend to be greater than 1, less than 1, or approximately equal to 1?)
  - (b) Another approach is to compare this gene between humans and chimpanzees via a Hudson-Kreitman-Aguade (HKA) test. Supposing that change in this gene partially explains the superior speech abilities of humans, would you expect it to show higher polymorphism or higher divergence, compared to a neutral control gene? Why?
  - (c) Suppose that when the gene sequences were compared, the human and chimpanzee alleles were found to code for exactly the same protein product. Would this rule out a role of this gene in development of human speech? Why or why not?