General Biology: Ecology and Evolution
Summer 2005 Session #2

Week #1 Workshop Answers

Questions:

1. The evidence supporting the pattern component of the theory of evolution can be criticized on the grounds that it is indirect. For example, no one has ever observed the formation of a vestigial trait over time. Because of the indirect nature of the evidence, it could be argued that structural and genetic homologies are coincidental and do not result from common ancestry. Is indirect evidence for a scientific theory legitimate? Are you persuaded that modification with descent is the best explanation available? Discuss this with your group.

Indirect evidence is legitimate for a scientific theory. In the case of processes that occur over long periods of time, no one can observe them directly. Indirect evidence cannot be used to prove a theory, only to provide evidence supporting the theory. Indirect evidence can stimulate further experimentation or analysis that can be used to either support or refute the theory, depending on the outcome. For example, many different genes can be sequenced to see how strong the genetic link is between two organisms that exhibit genetic homologies.

2. The average height of humans has increased steadily for the past 100 years in the industrialized nations. This trait has clearly changed over time. Most physicians and human geneticists believe that the change is due to better nutrition and less disease. What do you think – has human height evolved?

Height increase in humans over the past 100 years is more likely due to environmental changes in health care enabling each individual to more fully realize their height potential. There is no documented evidence that changes in the allele frequencies of genes that control height have occurred in the human population over the last 100 years, so there is no evidence of evolution. Height is a quantitative trait that is known to be affected by environmental conditions and probably by multiple genes. As a quantitative trait, an individual inherits a range of possible height

3. Genome sequencing projects may dramatically affect how biologists analyze evolutionary change for quantitative traits. For example, suppose the genomes of many living humans are sequenced and that genome could be sequenced for many people who lived 100 years ago (this might be possible with preserved tissue). If 20 genes have been shown to influence height, how could you use the sequence data from these genes to test the hypothesis that human height has evolved in response to natural selection?

Sequence the 20 genes from many samples of preserved human tissue and compare the sequences to those of the 20 genes from currently living humans. If evolution has occurred, there should be a significant increase in DNA changes correlated to taller individuals over the last 100 years—that is, a change in allele frequencies over time should be evident.

4. The text maintains that mutation has two roles as an evolutionary mechanism. It is too infrequent to produce significant changes in allele frequencies but it is the ultimate source of genetic variability. Explain.

Genetic variability can arise only by some type of mutation of the genome. It is the ultimate source of genetic variation, because a change in a gene must occur for the gene to specify a new product. Mutation occurs only relatively rarely each time gametes are formed, but the sum total of mutational events in a population provides the genetic variability of the population. Mutation, coupled with selection, can significantly affect genotype frequencies in populations.

5. In humans, albinism is caused by loss of function mutations in genes involved in the synthesis of melanin, the pigment in skin. Only people who are homozygous for a loss of function allele have the phenotype. In Americans of northern European ancestry, albino individuals are present at a frequency of about 1 in 10,000 (or 0.0001). Knowing this genotype frequency, we can calculate the frequency of the loss of function allele. If we let q stand for this frequency, we know that q^2 = 0.0001, so q = square root of 0.0001 which = 0.01.By subtraction, the frequency of normal alleles (p) is then 0.99. If these loci conform to the conditions required by the Hardy-Weinberg principle, what is the frequency of carriers (people who are heterozygous). Once you get an answer, you will know the percent of Caucasians in the US who are carry an allele for albinism.

The frequency of carriers of the albino allele in the population is 0.0198. If q = 0.01, and p = 0.99, then the frequency of the heterozygote is 2 pq, which equals 0.0099 + 0.0099, which equals 0.0198.

6. A certain strain of mice have 3 distinct coat colors: Black, gray and white. These coat colors are due to the following genotypes: BB = black; Bb = gray; and bb = white. Assume that the frequency of the black coat allele for mice in a population is 0.6, and that the population meets all of the expectations of the Hardy-Weinberg equilibrium theory. What are the expected genotype frequencies for the coat color locus in this population?

B = black; b = white; Bb = gray
Since allele frequency for B = 0.6, p = 0.6 Thus the allele frequency for b must = 0.4 since the total must equal 1.0. So, q = 0.4

P^2 = BB = 0.36
2pq = Bb = 0.48
Q^2 = bb = 0.16 All add up to 1 so there are no evolutionary pressures on this population.

7. If the population in question #6 continues to meet the assumptions of the Hardy- Weinberg Equilibrium theory, do you expect the allele and genotype frequencies at this locus to change drastically over time? Explain your answer.

No, there are no factors causing evolution (e.g., natural selection, drift, nonrandom mating, gene flow, or mutation)

8. Now let's investigate the coat color locus in another large population of this mouse species. In a sample of 100 mice from this population you find 60 with black coats, 10 with gray coats, and 30 with white coats. Calculate the allele and genotype frequencies in this sample.

Total mice = 100, The total # of alleles is therefore 200 (since each mouse has two copies of the gene)
B = black, b = white
Allelic Frequencies: p = freq of B = (60*2 + 10)/200 = 130/200 = 0.65
                         q = freq of b = (30*2 + 10)/200 = 70/200 = 0.35
                         Total = 1 – so it checks out
Genotypic frequencies: BB = 60/100 = 0.6 Bb = 10/100 = 0.1 Bb = 30/100 = 0.3 TOTAL = 1

9. The following frequencies are know from extensive research on a large population of PTC tasters and Non-Tasters: TT = 251 individuals; Tt = 250 individuals; tt = 334 individuals

    a. What are the allele frequencies of T and t?
    835 total individuals, 1670 total alleles
    p = freq of T = ((251*2)+250)/1670 = 0.45
    q = freq of t = ((334*2)+250)/1670 = 0.55

p + q = 1 – so it checks out.

    b. What are the expected genotype frequencies?

TT = p^2 = 0.45*0.45 = 0.202
Tt = 2pq = 2*0.45*0.55 = 0.495
tt = q^2 = 0.55*0.55 = .303
All add up to 1 – so they check out.

    c. What are the phenotype frequencies?

Can't really determine from information given. We don't know what's dominant. If we assume that T is dominant, then tasters = 0.202 + 0.495 = 0.697; t = 1-freq of T = 0.303

10. Out of 100 red oaks *(Quercus rubra)* in a population, the frequency of allele B is 0.45. The other allele at the locus, a recessive allele (b), was expressed in 20 individuals. Determine:
    a. observed and expected genotype frequencies
    b. whether the population is at Hardy-Weinberg Equilibrium.
    c. Challenge question: If you conclude its not in Hardy-Weinberg Equilibrium, can you design a study that would eliminate or confirm one evolutionary mechanisms that may be at work.

    a. Freq of B = p = 0.45
       Freq of b = q = 0.55
       Observed: bb = 0.2
       Expected: BB = 0.45*0.45 = 0.2025
                   Bb = 2*0.45*0.55 = 0.495
                   bb = 0.55*0.55 = 0.3025

b. Expected does not equal the observed, so it may not be in Hardy-Weinberg equilibrium.

c. too many to describe...