TEST FORM A

Evolution – PCB 4673
Exam # 2

Name _____

SSN _____

Multiple Choice: 3 points each

1. The horseshoe crab is a so-called "living fossil" because there are ancient species that looked very similar to the present-day species found in the fossil record. Is there any indication that the lack of phenotypic change in such lineages is the result of a lack of genetic variation?

   A. Yes, surveys of genetic markers indicate that such taxa are monomorphic (have no variation) at most loci surveyed.
   B. No, surveys of genetic markers indicate that such taxa have similar levels of heterozygosity at most loci surveyed as other taxa.
   C. Early surveys conducted on proteins suggested that there was not lack of variation, but more recent studies at the molecular level contradict the early results.
   D. Such taxa have been shown to be genetically uniform at key loci determining morphological variation.
   E. None of the above are correct.

ANSWER B

2. Based on current estimates of mutation rate, how many mutations in protein encoding genes are typical for each human?

   A. 6-10 per person per generation (about 5 deleterious)
   B. 60-100 per person per generation (about 5 deleterious)
   C. close to zero, any mutations that arise are typically lethal
   D. 4-5 per person per generation (about 2-3 deleterious)
   E. none of the above are correct

Answer D

Use the following data on numbers of individuals with each genotype to answer the next two questions.

	Genotypes		
Locus	A_1A_1	A_1A_2	A_2A_2
Alcohol dehydrogenase (Adh)	5	10	5
Esterase (Est)	1	6	13
glucophosphoisomerase (Gpi)	0	4	16
lactose dehydrogenase (Ldh)	8	1	11

3. What is the heterozygosity at the Ldh locus of an average individual in this population? Hint: one possible solution: $H = 1 - \sum p^2$

A) 0.49
B) 0.32
C) 0.43
D) 0.51
E) None of the above is correct

Answer A

4. Which of these loci is **least** consistent with Hardy-Weinberg expectations?

A) Adh
B) Est
C) Gpi
D) Ldh
E) All are equally consistent with Hardy-Weinberg predictions

Answer D

5. If the heritability of a trait is 0.1, it means:

A) that 10% of the phenotypic variance is from environmental effects on development.
B) that 0.1 map units separates the genes that determine this trait.
C) that 30% of the phenotypic variance of the trait is additive genetic variance.
D) that a major part of the phenotypic variance of the trait, up to 90%, is from environmental and non-additive genetic effects.
E) both C and D above are correct.

Answer E

6. If the norm of reaction for two genotypes cross, it indicates
   A) the presence of a genotype by environment interaction.
   B) that the heritability of the trait is different in different environments.
   C) That the difference between the two genotypes depends on the environment where they are compared.
   D) A and B are correct.
   E) A, B, and C are all correct.

Answer E

7. The offspring of two very different plants (one with leaves with smooth margins and another with very deeply indented margins) are raised in a common garden experiment. All the offspring of both plants display identical phenotypes, leaves with a smooth margin. What does this indicate?

A) the presence of a genotype by environment (GxE) interaction.
B) that leaf margin shape has a high heritability.
C) that leaf margin shape is largely or exclusively determine by the environment.
D) that both genes and environment determines the shape of the leaf margin.
E) none of the above can be determined from a common garden experiment alone.

Answer C

8. The number of ridges on the surface of eggs laid by females of a small intertidal fish, *Fundulus heteroclitus*, decreases gradually in populations along the eastern U.S. coast from northern New Jersey to Jacksonville, Florida. This pattern of variation is described as:

A) sympatric
B) allopatric
C) a hybrid zone
D) a cline
E) none of the above are correct.

Answer D

9. Imagine there are 100 populations with 10 diploid individuals each, and each has an initial allele frequencies of $A_1 = 0.2$, $A_2 = 0.6$, and $A_3 = 0.2$. After many generations, how many populations are expected to be fixed for A_2?

A) 20
B) 60
C) 80
D) 10
E) can't tell from the information provided.

Answer B

10. According to theory of gene coalescence,

A) all individuals in a finite population are identical by descent before they are identical in state.
B) all individuals in a finite population are identical in state before they are identical by descent.
C) all individuals in a finite population become identical by descent in haploid populations.
D) genetic drift leads to an increase in homozygosity within populations.
E) none of the above are correct.
Answer B

11. In an assemblage of 100 finite local populations of the same species,

A) each local population will lose heterozygotes from genetic drift and individually deviate from the Hardy-Weinberg predictions in a unique way.
B) each local population will accumulate an excess of heterozygotes and individually deviate from the Hardy-Weinberg predictions in a unique way.
C) each local population will lose allelic variation from genetic drift and ultimately one allele will be come fixed.
D) A and C are both correct.
E) B and C are both correct.
Answer C

12. In an assemblage of 100 finite populations of the same species with an initial allele frequency of allele $A_1 = 0.3$:

A) the frequency of A_1 for the total assemblage will still be 0.3 after 100 generations.
B) the frequency of A_1 for the total assemblage will drift to 0 after many generations.
C) the frequency of heterozygotes in the total assemblage will be 0.42 after many generations.
D) frequency of homozygotes in the total assemblage will be 0.58 after many generations.
E) C and D are both correct.
Answer A

13. In an assemblage of 100 finite populations of the same species:

A) the frequency of heterozygotes in individual populations will remain consistent with Hardy-Weinberg expectations even after 100 generations.
B) the frequency of heterozygotes for the total assemblage of populations will become fewer than Hardy-Weinberg expectations after many generations, dependent upon the actual population size in each.
C) the frequency of heterozygotes for the total assemblage of populations will remain consistent with Hardy-Weinberg expectations even after many generations, even if the size of each is small.
D) A and B are correct.
E) A and C are correct.
Answer D

14. Deviations between the effective population size and census population size

A) may be the result of genetic drift.
B) are generally in the direction that effective size is smaller than census size.
C) indicate that the real population is losing genetic variation at a different rate than the ideal population.
D) A and B are correct.
E) A, B, and C are correct.
Answer D

15. Which of the following is a subspecies:

A) a named geographic race
B) a geographic region where two genetically distinct populations meet.
C) Two forms that live in separate geographic locations.
D) Two forms that live in the same geographic location.
E) None of the above are correct.

Answer A

Short Answer Questions: Five points each.

1. Consider a population where all males have the genotype "aa" and all females have the genotype "AA" at a diploid, autosomal locus. Males and females are equally common. How many generations will elapse before the Hardy-Weinberg predicted equilibrium frequency of genotypes is reached at this locus if mating is random? (Show your work.)

Freq A = 50%; a = 50%; H-W predicted genotypic freq = AA 25%; Aa 50%; aa 25%

So, AA x aa = Aa 100%; Aa x Aa = 25% AA, 50% Aa, 25% aa; which will remain indefinitely if H-W conditions remain. Therefore, the correct answer is 2 generations of random mating are required.

2. List the assumptions of the Hardy-Weinberg Law as indicated on the class notes.

No mutation, migration, selection, drift (infinite population size), random mating, no meiotic drive.

3. Given the following allele frequencies, what are the expected frequencies of gamete types: A = 0.6; a = 0.4; B = 0.8; b = 0.2?

AB = 0.6 x 0.8 = 0.48; Ab = 0.6 x 0.2 = 0.12; aB = 0.4 x 0.8 = 0.32; ab = 0.4 x 0.2 = 0.08

4. Identify the two mechanisms that can yield linkage disequilibrium that were discussed in class. Explain how long each persists (one generation, a few generations, indefinitely) and why?

Physical linkage – lasts indefinitely, until recombination occurs between the genes, which is a function of the length of chromosome that separate the genes

Founder event – lasts from a few to many generations, arises because of sampling events in the founding of the new population (not all chromosome types are represented).

5. Explain what it means that mutations are not random at the molecular level AND give some mechanisms covered in lecture that cause mutation to be non-random.

This means that the likelihood of a mutation occurring is not equal across the genome; some features of the DNA strands increase the likelihood a mutation will occur nearby (or decrease the likelihood). Mechanisms include: transitions more likely than transversions; AT rich regions facilitate mutation by changing DNA secondary structure; sense and anitsense strands differ in rate; neighboring base pair affects rate, i.e., G in 3rd codon position increases chance of mutation relative to C in 3rd position; repair mechanisms vary in efficiency.

6. If the genotypes at two loci are each at Hardy-Weinberg equilibrium frequencies, can there be linkage disequilibrium between the loci? Provide an example to illustrate your answer.

Yes, they can be in H-W genotypic frequencies but not in linkage equilibrium if the assumptions of H-W are met.

Consider the example I gave in class where were started a population with only A1A1B1B1 and A2A2B2B2 genotypes. After one generation of random mating, the genotypic frequencies were: A1A1B1B1 = 0.25; A1A2B1B2 = 0.5; A2A2B2B2 = 0.25. This population is very much in linkage disequilibrium because many haplotypes are missing completely (e.g., A1A1B2B2), but the allele frequencies are A1 = 0.5 and B2 = 0.5 and both loci are in Hardy-Weinberg genotypic frequencies (e.g., A1A1 = 0.25; A1A2 = 0.5; A2A2 = 0.25).

7. Most of the differences between maize (=corn) and its wild progenitor, teosinte, can be attributed to the effects of only four genes. Nonetheless, these two plants have very different phenotypes. How might you resolve this paradox?

This is the result of interactions of genes. In particular, if one gene regulates the expression of some of the others, large differences could arise. Another possibility is that these genes play a role in early development. In class we discussed the homeotic genes in animals, that determine the timing and early steps of body segmentation. Mutations in any of these genes can have tremendous implications for phenotypes, for example "antennapedia" fruitflies.

Special Thought Questions: 10 points each

1. Describe the design of an experiment that tests the hypothesis that mutations are "directed" (arise in response to their potential benefit to the organism). What was found when such an experiment was conducted? Use a picture if you wish.

Luria and Delbruck experiment: Start several lineages of bacteria from a single cell founder. Maintain these for several generations, freeze a sample each generation, split culture into two new lineages after each generation to yield a collection of cultures with known pedigrees going back several generations (4 in class example). Expose fourth generation to a novel environmental challenge, lethal phage for example, and record the resistance to challenge for each culture. If resistance is random with respect to pedigree (Poisson distribution of resistant lines across lineages), then mutation arose in response to challenge. If resistance clumped in a subset of lineages, resistance arose in previous generation without regard to challenge. Also, can trace back in lineages to see where the mutation arose that provided resistance using frozen historical cultures.

2. In lecture, I claimed that mutation rates are sufficiently low, per locus per gamete per generation, not to be a strong force in producing evolution. Yet a close personal friend of yours claims that in fact mutations are very common in natural populations and therefore mutation rates play a significant role in evolution. Describe a study that could resolve this paradox. (hint: experimental evolution study)

Lenski and Benett study. Start several lineages of E. coli from a single cell (i.e., starting material all genetically identical). Maintain several lineages in each of several environmental conditions (four temperatures for example). Freeze samples from each lineage every 100 generations. After many generations (2000 in example), reconstitute the original lines and conduct competition experiments to see which cultures have highest fitness in experimental conditions (fitness is measures as the population growth rate r in mixed culture). To show that mutation alone is not adequate to explain adaptation, compare the performance of cultures maintained in the original conditions for the experiment to those in different environments. If mutation alone was adequate, there should be no difference in the fitness of the culture exposed to the new environment and the ones maintained under the original environment.

Considering mutation rates could make a second line of argument. Typical mutation rates for protein encoding genes are 10^{-4} per gene per generation or lower. In our experiment, imagine that 100 genes might affect population growth rate of E coli with respect to temperature (the real number may be much lower). In 2000 generations, the odds that any one of these had any mutation (deleterious or beneficial) would be $2000 \times 10^{-4} = 0.2$ and the odds that at least one of the 100 genes had any mutation is at best $0.2 \times 100 = 20$ (indicating that there were on average 20 mutations in the genetic system responsible for temperature tolerance over the course of our experiment). Since most mutations are neutral to mildly deleterious (the figure I gave in class would suggest over 90%… probably closer to 99.9%) and since the Lenski and Bennett study demonstrated an improvement in performance at the temperature they were reared for every experimental lineage, mutation alone is not a likely explain for these results.