Biology 3410, key to second midterm exam (9 March 2007) 11 March 2009

1. What are *quantitative traits?* Please explain *in general*, and give an *example.* [10 points]

They are continuously distributed phenotypic attributes such as lengths, masses, rates and the like, that can be measured on a numerical scale. Their variation is caused by genotypes at several or many different genetic loci that contribute more or less independently to the trait values of individuals, and by environmental experiences that also vary randomly. Examples include human height, the beak dimensions of birds, and fitness itself (among thousands of other possibilities).

2. Why are quantitative traits *normally distributed* (at least approximately, in most cases)? [10 points]

With several or many independent contributions from different genetic loci and the environment, there are many more ways to have near-average trait values than there are to have extreme trait values. This adding-together of many independent, random effects inevitably gives rise to normal or near-normal distributions.

3. In 1976 (before the drought of 1977), the average adult beak depth of Darwin's medium ground finches on Isla Daphne Major was 9.4 mm. In 1978 (after the drought) the average beak depth of the survivors was 10.1 mm. The survivors then mated at random with respect to beak depth, and produced offspring with an average beak depth of 9.7 mm. From these data, you can estimate the narrow-sense *heritability* of beak depth in this population. Please do so, and *show your work.* [15 points]

S = 10.1 – 9.4 = 0.7 mm
R = 9.7 – 9.4 = 0.3 mm
h^2 = R/S = 0.3/0.7 = 3/7 = 0.43 [3/7 is a perfectly acceptable answer]

4. Can a *change in the environment* create or destroy *heritable genetic variation* for a quantitative trait? Please answer yes or no, and then briefly *explain.* In your explanation, you might want to use an *example* like bristle number in *Drosophila* larvae, or myopia (near-sightedness) in human populations. [15 points]

Yes! Depending on how different genotypes in the population respond to various environmental variables, there may be some states of the environment in which the genotypes produce distinct phenotypes (hence much heritable variation), and other states of the environment in which the genotypes produce very similar phenotypes (hence little heritable variation). [The technical jargon for this effect refers to *norms of reaction* and *genotype-by-environment* (=GxE) *interaction*, but you didn't need to use these terms to get full credit.] Experimentally estimated relationships between bristle number and temperature for different genotypes of Drosophila were used in lecture to show how a change of rearing temperature could increase or decrease the heritable variation for larval bristle number, holding the mixture of genotypes constant. A real-world human example was discussed in the last lecture on human health. Near-sightedness is highly heritable in modern environments, where small children spend a lot of time reading and doing other close-focus work, but not in traditional environments where few people become near-sighted because they don't do long periods of close-focus work as children. The same underlying genetic variation was present in the traditional environments, but there was little heritable variation for near-sightedness because all genotypes produced normally-shaped eyes. But by demanding many hours of close-focus performance in childhood, modern environments reveal that these genotypes are *differentially susceptible* to developing myopia in response to the stress of extended periods of close-focus work early in life.

5. Many species of marine fish and shrimps have been heavily harvested for decades. Fisheries biologists have noticed that most of these species now mature at younger ages and smaller sizes than they did in the past, and that their maximum lifespans are now shorter. These evolutionary changes were caused by fishing, and they could have been *predicted*. Please explain why. [20 points]

Harvesting is a cause of increased *extrinsic mortality* (deaths that randomly strike all individuals equally without regard to their rate of senescence) When extrinsic mortality goes up, the evolutionary value of late reproduction goes down, because individuals become relatively unlikely to survive to older ages. Selection to survive to older ages therefore becomes weaker. And for the same reason, the value of *early* reproduction goes *up*. Individuals are therefore selected to reproduce more at early ages, even if this makes them less able to survive to older ages. This is a straightforward prediction of the evolutionary theory of senescence!

6. Suppose you wake up tomorrow morning and read in the *Deseret News* that a new species of South American songbird has been discovered, in which the females are larger and more brightly colored than the males. What would you predict about their breeding system? Does one sex build the nest and do most of the offspring care? If so, which sex? Or do both parents share equally in the work of parenting? *Explain your reasoning.* [15 points]

I would predict that *males* do most of the parenting. Males probably make nests, brood the eggs, and do most or all of the feeding and protection of young. Females probably mate with several males each season (if they can), while males typically mate with just one female. Females probably court males and defend them (and their nests) from other females. In a "sex-reversed" species such as this, male reproductive success is limited by access to resources (nesting sites, food for the young, etc.), while female reproductive success is limited by (mating) access to males. Thus males evolve to be "ecologically sensible" and females evolve to be good at competing for mates.

7. No one knows how many species there are on Earth. Fewer than two million have been described and named, but estimates of the total range from 10 to 100 million. Many experts believe that of this total, more than half (perhaps *way* more than half) will turn out to be parasites. This belief is based mainly on extrapolations from existing data, but it is also supported by theoretical considerations about the process of speciation. What is it about the biologies of parasites that would lead you to think that they might tend to *speciate* at relatively *high rates*? Hint: Parasites are often highly specialized for life on or in particular species of hosts (or groups of closely related host species). Briefly explain your reasoning. [15 points]

Speciation requires *reproductive isolation* of two populations. Otherwise migration (gene flow) will prevent them from diverging genetically. By shifting onto a new host species, a parasite population can easily become isolated from its "parent" population. This will obviously happen if the parasite lives its whole life on the host and typically transmits directly from host parents to their offspring. But it can also happen if the parasite has a free-living stage of its life cycle, or even if it has an alternate host that it still shares with its parent species, as long as it doesn't reproduce sexually when it's together with the parent species. As long as sexual reproduction occurs only on the new host, to which members of the "daughter" population are attracted but members of the parent population are not, then the daughter population will be reproductively isolated from the beginning and therefore able to speciate. This is the mechanism of "host-race formation", which was illustrated in lecture and in the textbook by the apple maggot (*Rhagoletis pomonella*), which recently shifted onto cultivated apples from its native North American host, wild hawthorn.

Biology 3410, key to the 3rd midterm exam (as given 6 April 2005) 11 March 2009

1. Humans are fond of recording their heights, so we have a great deal of information about the distribution of this simple quantitative trait. Body size measurements of all kinds are usually highly heritable. The heritability of height has been estimated many times in many populations, and the resulting values of h^2 almost always fall between 0.6 and 0.8. *What is heritability*, and what do numbers like 0.6-0.8 imply about the *causes of the observed variation in height?* [25 points]

$h^2 = V_A / V_P$. In words, heritability is the fraction of the total (phenotypic) variance that is contributed by the additive genetic variance (the part caused by the additive effects of genes). The heritability estimates for human height imply that 60%-80% of the phenotypic variance is caused by the additive effects of genes, within a given population at a given time in human history. The non-additive genetic variance and the environmental variance together account for much less than half of the total.

2. Average human heights have increased dramatically in many countries since World War II. The reasons are always said to be improved nutrition and medical care (*i.e.*, environmental change). We all believe this, *but how do we know?*

What if an overeducated friend of yours said "Nonsense! I think it's *selection!* Quantitative traits evolve like crazy in most natural populations. Haven't you heard of Darwin's finches? Why couldn't something like that happen with human height? It's *highly* heritable, so a large change in the mean trait value seems most likely to be an *evolutionary* change."

On the face of it there's nothing wrong with this argument, so you can't just assert that your friend is out of her mind. (Well you *could,* and you might be right, but doing so won't win the argument.) What you need to do is show that *in fact* these changes were *not* evolutionary. Assume you have access to a comprehensive, LDS-quality genealogical database for Kyoto, Japan, for the years 1935-1995, with information on peoples' adult *heights* as well as their complete family histories (who they married and who their children are). How would you analyze these data to test your friend's hypothesis, and what finding would refute it? *Be specific!* [25 points]

We know that height is highly heritable at any given time and place, so we don't need to prove that again. The key question is whether *selection* has favored increased height. I would plot offspring number against height for people in their 50s (an age when they have virtually all finished reproducing) in each 5- or 10-year period in the database. My friend's prediction is that taller people have more children. My prediction is that they don't – or at least that the association between height and reproductive success is *too weak* to explain the large increase in average height during this 60-year period.

3. In an ecological and evolutionary context, what is a *tradeoff?* Explain (in general) by reference to a *specific example.* [15 points]

A tradeoff exists when changing some phenotypic attribute causes both beneficial and harmful effects ("good news" and "bad news", with respect to fitness). In many cases, the optimum phenotype represents a compromise between these up-side and down-side effects. For example, a plant that makes larger, fitter seeds must make fewer of them because the resources available for reproduction are limited. Another good example is the size of galls made by a fly whose larvae develop in the stems of goldenrods. Small galls are more likely to be attacked by a parasitoid wasp, but large galls are more likely to be attacked by a woodpecker. The fittest gall size is near the midpoint of the observed distribution.

4. Given that reproductive altruism *can* evolve by kin selection, why do we expect it to occur mainly between very *closely related* individuals? Why not between *distantly* related individuals? Be sure to include *Hamilton's rule* in your explanation. [15 points]

$Br > C$, where B is benefit to the recipient of the altruism and C is cost to the altruist. As r (the coefficient of relatedness) becomes smaller, the altruism must become more efficient (B/C must become larger). For obvious ecological reasons, very large B/C ratios will seldom be attainable.