Biology 3410

Second midterm exam

13 March 2009 your name: Jon's key student ID:

1a. (24 pts) The variance of beak depth in a population of Darwin's finches is 1.0 mm^2, and the slope of the regression of mid-offspring values on mid-parent values is 0.80. Which of the following quantities can we estimate from these data, and for those that we *can* estimate, what are their *values*? Also please give the *name* of each quantity (in words), or briefly explain its meaning, if you forget the name.

Symbol	Name in words	Can we estimate it? (circle one)	If so, its *value*
V_P	phenotypic variance	[✓ Y] N	1.0
V_G	genetic variance (total genetic variance)	Y [✓ N]	
V_A	additive (genetic) variance	[✓ Y] N	0.8
V_D	dominance variance	Y [✓ N]	
V_E	environmental variance	Y [✓ N]	
h^2	narrow-sense heritability	[✓ Y] N	0.8

1b. (8 pts) The mean beak depth before a big drought was 9.5 mm. Afterward, the mean beak depth of the survivors was 10.0 mm. What do you predict the ***mean beak depth*** of the next generation (offspring of the survivors) will be? You will need to use one of your answers from above. Please show your work!

$S = 10.0 - 9.5 = 0.5$

$R = h^2 S = 0.8 * 0.5 = 0.4$

mean beak depth = 9.5 + 0.4 = 9.9 mm

1c. (8 pts) Suppose no similarly severe drought comes again for many years. All else equal, do you expect beak depth to maintain its post-drought mean value? Or will it probably change? If so, in which direction, and why? Please explain carefully. Hint: trade-offs!

I would expect mean beak depth to gradually decline back toward the pre-drought mean, on the assumption that 9.5 mm is closer to the long-term optimum under the trade-off between greater ability to crush large, hard seeds (associated with larger beaks) and greater efficiency in processing smaller, softer seeds (associated with smaller beaks).

2. (20 pts) A change in the environment often creates *heritable genetic variation*. How can this happen? As part of your explanation, you might want to draw some hypothetical norms of reaction (in which case be sure to label the axes and curves). Or you might want to talk about common medical conditions that seem to be associated with modern diets, pollutants, or other unique features of our current environment.

If different genotypes produce the *same* phenotype in a certain environment, then there is no heritable variation *in that environment*. But if the environment changes to a state in which those genotypes now produce different phenotypes, then there may be heritable variation for the trait in question, in that environment. We illustrated this idea in lecture with a thought-experiment based on real norms of reaction for larval bristle numbers in *Drosophila melanogaster*. You could have drawn any sort of similar example, where the norms cross at some values of the environmental variable, but have substantially different values of the phenotypic variable at other values of the environmental variable.

We also discussed evidence suggesting that modern human environments may be "uncovering" variation for sensitivities to particular foods, or diet types (e.g., unlimited calories from refined carbohydrates), or pollutants, which were basically never experienced by our ancestors. And we mentioned the hypothesis and evidence that myopia (near-sightedness) is largely a condition of modern industrialized societies, where young children engage in extended close-focus activities, uncovering abundant but previously "cryptic" variation for susceptibility to developing myopia when challenged in this way.

3. (16 pts) Selection is constantly "optimizing" quantitative (and other) traits, to maximize fitness under current environmental conditions. Nonetheless, phenotypes are usually less than optimal for most individuals in a population, and even on average. Please give (and briefly explain) at least *two* reasons why we expect this to be true, in general.

There are many reasons why phenotypes are usually less than optimal. One is that environments are constantly changing, and adaptation takes time, so populations are usually somewhat "behind the curve". Another is that traits are often correlated for genetic and/or developmental reasons, so that selection on one of the traits tends to change the other, which may therefore be pulled farther away from its optimum value. A special case of this is where selection on a certain trait in one sex (say, larger size in males) changes the same trait in the other sex, maladaptively for members of that sex.

Another, very general reason for non-optimality is that mutations of small effect are constantly occurring in every population, and many of these will rise to significant frequencies even if they are slightly deleterious. Quantitative traits provide an important special case of this principle. A trait subject to stabilizing selection might have a population mean value that was right on the optimum, but even so, individuals in the upper and lower tails of the distribution might have significantly suboptimal values of the trait. In such a case it could be true that *none* of the alleles contributing to heritable variation of the trait was "deleterious" in any absolute sense, but the effect of all of them, collectively, over all the loci contributing to variation in the trait, would be to make most individuals significantly suboptimal! Of course if selection against the tails of the distribution were very strong, then the variance would evolve downward to some extent.

Modern humans live a lot longer on average, and have much longer maximum life spans, than other mammals with similarly large body sizes. In particular, we live longer than any of the other great apes. Thus it's clear that our lineage evolved *increased* life spans, after the time of our last common ancestor with chimpanzees. To make sense of this, please carefully answer the following questions. (8 pts each)

4a. In general, how does selection shape the rate of senescence? What's the key *tradeoff*, and what determines the optimal balance point between the competing "goods" (one of which is longer life span)?

The tradeoff can be stated in several different ways, but the key idea is that reproduction (now) and survival (into the future) are in competition with each other, for inescapable physiological and developmental reasons. Thus reproduction now and reproduction in the future are somewhat incompatible. If the rate of "extrinsic" mortality (e.g., by predation) is high, then future reproduction is less valuable because it's less certain to happen at all, and individuals may do best to reproduce as much as possible, as early as possible, regardless of the consequences for future survival and reproduction. And conversely, if extrinsic mortality is low (as for adult birds), then it may be best to "save oneself" for future breeding seasons.

4b. How might the ecology of our ancestors have changed so as to tilt the balance in favor of a longer maximum life span? Your answer will be speculative, of course. But it should be a specific hypothesis that (together with the general principle in 4a) could explain what happened.

The rate of extrinsic mortality must have gone down, to favor a longer pre-reproductive juvenile period and a longer maximum life span. There are many reasons why life might have become safer for early hominids, including better nutrition, fewer deadly diseases (maybe the drier savanna habitat was healthier than the humid forests), weapons and fire for defense, stronger social groups, etc. In any case, if life expectancy started going up for these extrinsic reasons, then it might have become more profitable to reduce the effort put into early reproduction, so as to better maintain the body, slowing the rate of senescence and increasing the probability of successful reproduction later in life.

4c. And how can we possibly explain the long post-menopausal life spans of women? In no other primate (and probably in no other animal) do females remain healthy and strong for so many years after they cease reproducing! The general principle (4a) cannot explain this, by itself. What else do we need?

We need a way for females to have indirect or "vicarious" reproductive success (for example, through their children). For example, a grandmother can help her daughter have more children by feeding and otherwise caring for the daughter's previous children. The grandma is half as related to her (extra) grandkids as she would be to children of her own.

Another possibility is that selection on males to live longer (for example, to accumulate more wives and actual offspring) increased female life spans as a *side effect*. But if grandmothering were not a significant source of inclusive fitness for females, then I would expect the age of menopause to evolve *upward*, so as to allow women to capitalize directly on their increased longevity. This has not happened, suggesting that the indirect fitness gained through grandmothering has probably been substantial.