Study questions. First Exam. Evolution (L567) 2013

Note: the same question is sometimes asked in different ways.

Write the equations for variance and covariance. Then write a sentence in English that defines variance and covariance. Compare the two. In what way can a variance be seen as the covariance of a thing with itself? Write the equation for the slope of a regression line. How is it related to variance and covariance?

Why did Francis Galton (Darwin’s cousin) reject Darwin’s view of evolution by natural selection? Why did he reject both gradualism and the provisional theory of pangenesis? What did Galton mean by regression on mediocrity?

In class we showed (assuming one locus with two alleles, and infinite population size) that

\[ \Delta p = \frac{pq[p(W_{11} - W_{12}) + q(W_{12} - W_{22})]}{W} \]

There are “trivial” equilibria at \( p=0 \), and \( p=1 \). Why are those called trivial equilibria? There is also an equilibrium at \( W_{11}=W_{12}=W_{22} \). Why is that an equilibrium? This equilibrium is called the ____________ equilibrium (fill in the blank). If the population is perturbed away from the equilibrium, will the population return to the starting point? Is this kind of equilibrium also an attractor?

Now suppose there is variation in fitness, and that there is genetic variation. Solve for \( p \) at equilibrium ( \( \hat{p} \) ). Show the algebra.

For \( \hat{p} \) to be on the interval between zero and one requires that \( 1 > \hat{p} > 0 \). Show what this means algebraically with respect to your solution for \( p \) at equilibrium. What does it mean in words?

We also showed in class, using \( h \) as the coefficient of dominance and assuming weak selection (small \( s \)), that

\[ \Delta p \approx pq s [ph + q(1-h)] , \]

Assuming

\[ W_{11} = 1 \]
\[ W_{12} = 1 - hs \]
\[ W_{22} = 1 - s \]

If allele 1 is dominant, what is \( h \)? What is \( \Delta p \)?
If allele 1 is recessive, what is \( h \)? What is \( \Delta p \)?
If allele 1 is co-dominant, what is $h$? What is $\Delta p$?

Relatively speaking, compare the spread of allele 1 when rare for each of the three conditions above. Defend your reasoning (hint: your excel model should help here).

What are the consequences of assuming an infinite population size in the model with respect to the spread of a recessive beneficial allele?

What is meant by “mutation-selection” balance? In English words, what is going on at mutation selection balance? (Note: the solution for $\hat{q}$ is given on the course website.)

What is meant by “fixation” in population genetics?

What is the heritability of two ears on a population of mice, all having two ears?

Given our population genetic formulation to this point, what is the general long-term effect of directional selection on Additive genetic variance?

Will natural selection always drive population mean fitness up? Why, or why not?

When will genetic drift over-ride natural selection?

Can there be additive genetic variation in an asexual population composed of multiple, genetically different clones?

What is the difference between “dominance” and “dominance variation”?

Why was Darwin’s idea of gradual evolutionary change through the action of “natural selection” rejected by most of his contemporaries?

Is natural selection necessary and sufficient for evolutionary change? What is the difference between natural selection and evolution? Define both.

What is essentialism? What is typological thinking? How are these different from populational thinking?

What is meant by blending inheritance? Why was it thought to be a problem for natural selection?

Give a formal definition of an ESS. Contrast an evolutionarily stable strategy with an evolutionarily stable state. What is a mixed ESS? What is a pure strategy? What is a conditional strategy? Give examples.

In the hawk-dove game, when would hawk be an ESS (in general)? When would dove be an ESS (in general)?
Is it possible that, in a two-strategy game, both strategies would be evolutionarily stable? If, so explain how. Is it possible that neither strategy is an ESS? If so, what would you predict about the population?

What is meant by “frequency-dependent selection”?

What did the study by Nick Davies show regarding the defense of sunspots by butterflies?

What is the difference between dynamic equilibria (limit cycles) and static equilibria? Give examples.

What is the difference between discrete strategies and continuous strategies? How does the difference affect the modeling approach?

In the Hawk-Dove-Bourgeois game, why is the payoff for B vs. B equal to V/2? And why is this an ESS? It is the same payoff as for Dove vs. Dove, but Dove is not an ESS. How is that possible?

What was Fisher’s argument for the evolution of investment in the sexes? What were the assumptions, and how were these assumptions later relaxed by Hamilton?

How could restricted access to mates (Local Mate Competition) affect the evolution of gender allocation (male vs. female function) in hermaphrodites? Give an intuitive explanation.

How could restricted access to mates (LMC) affect sex ratio evolution in species with separate sexes (females and males)? How could you find the optimum investment in sons? How could you tell if the candidate ESS was actually stable to invasion (i.e., at a local maximum)?

What is the difference between an ESS and a CSS? Why is Fisher’s sex ratio problem a CSS and not an ESS?

How will natural selection operate on offspring size? What is a size-number compromise? How can a size-number compromise lead to parent-offspring conflict? Give a graphical model for the evolution of optimal allocation to each offspring. Is the size-number compromise an example of frequency-dependent selection? (Why or why not?)

What was the main result of the Salmon study (by Einum & Fleming) with respect to life-history evolution?

When might selection operate to reduce the variance in offspring survival over time? How would such a reduction in variance affect the geometric mean? How might it affect the amount of resources allocated to individual offspring? What, if anything, would the solution have to do with Bet Hedging?
Contrast the assumptions and predictions of Muller's Ratchet and Kondrashov's hatchet. How does Muller’s Ratchet work? How does Kondrashov's hatchet work?

If mutations act independently, how could you find fitness at mutation-selection balance? (You don’t need to do the math, just indicate what must be true at mutation selection balance.)

Write your own question, and briefly answer it (5 points for the brilliance of the question and 5 points for the answer; total score is the product of the two).

Write your own question, and briefly answer it (13 points for the brilliance of the question and 12 points for the answer; total score is the sum of the two).

Which of the above two questions is most scary? Why?

What three concepts would you insist on including in an undergraduate course in evolution?

What is meant by diminishing returns? Give an example. Exactly how do diminishing returns figure into Local Mate Competition? How do they figure into the size-number compromise?

What is Bateman’s principle? Can you think of ways in Bateman's principle it can be violated?

How would you determine “candidates” for a local maximum for fitness? How would you determine whether a “candidate” is an ESS? Advanced: how would you determine whether the “candidate” is convergence stable (a CSS)?

In our model of local mate competition: Why did mean fitness decline as the population evolved to increase male allocation (starting in a population where the resident’s allocation was 0.25)? Why was the plot of fitness against an individual's allocation to male function almost linear for large mating populations (e.g. K=100)? And why did it have a non-linear shape in small mating populations (e.g., K=4)? In other words, how are diminishing returns on male allocation manifested?

Assuming infinite, randomly mating populations, how could the investment in sons and daughters be equal, but the sex ratio be female biased? In Fisher's result, what is being equalized: the sex ratio or the allocation of resources to sons and daughters?

Compare the macroevolutionary models for the prevalence of sexual reproduction in higher plants and animals.

More on Population Genetics:

What was Darwin’s provisional theory of pangenesis?

What is blending inheritance? Why did it present a problem to the theory of evolution by natural selection?
Why did Huxley think that Darwin’s commitment to gradual change was an unnecessary burden?

For one locus with two alleles, write the equation for $\bar{W}$. What does it mean?

The equation for $p'$ is: $p' = p\frac{pW_{11} + qW_{12}}{\bar{W}}$. Give an intuitive feeling for why this should be so. What is true at the equilibrium value for $p$?

Is $\bar{W}$ always maximized by selection? Why or why not?

What is meant by mutation-selection balance?

What difference does it make if an allele is dominant or recessive on the spread of that allele when it confers a slight fitness advantage when expressed.

Remember that $\Delta p \approx pq^2s$ for a favored dominant and $\Delta p \approx p^2qs$ for a favored recessive. Give an intuitive feeling for why the equations are correct (or incorrect, if they seem wrong to you).

Quantitative Genetics:

Give an intuitive feeling for the equation: $R = h^2S$. How is $h^2$ related to Galton’s worry about regression on the mean?

What is the selection differential $(S)$? How is it similar/different to the selection gradient $(\beta_{w,z})$? How is it different from the selection coefficient $(s)$ in Population Genetics?

What is meant by additive genetic variance? Why is it so important? How is it affected by dominance? How is it affected by allele frequencies? How is it related to the rate of evolution?

Give an intuitive feeling for the equation: $h^2 = \frac{V_A}{V_P}$

$h^2 = \beta_{o,\bar{p}}$, where $\beta_{o,\bar{p}}$ is the regression coefficient for the regression of the mean of a brood for a quantitative trait against the mean of the parents for the same trait. Why? How is this relationship used to estimate narrow sense heritability? Why is $\beta_{o,\bar{p}} = \frac{\text{cov}(\bar{p}, o)}{\text{var}(\bar{p})}$? What does it mean, in words?

What is meant by $H^2$? This is the broad-sense heritability. How is it different from $h^2$? (I don’t remember if I explicitly covered this, but $H^2 = \frac{V_G}{V_P}$). In words, $H^2$ is the genetic variance
divided by the total phenotypic variance. So the numerators for $H^2$ and $h^2$ are different. Why would that matter?

"Unpack" $V_G$. Give graphical examples of Dominance, Additivity, and an interaction between two loci. Make a clear distinction between dominance and $V_D$.

Does complete dominance mean that there is no additive genetic variance? Does an interaction between loci imply that there is no additive genetic variance? Why or why not?

Give an intuitive feeling for why the additive genetic variance depends on allele frequencies. Connect your reasoning with the population genetic models.

Design a simple experiment to estimate the environmental variance, $V_E$.

Give an intuitive explanation (or graph) for why dominance might reduce the slope of the regression of offspring value on mid-parent value compared to the slope of the regression of the same slope under co-dominance.

Explain why heritability, $h^2$, is different from the probability of inheriting a trait. Design an undergraduate lab to get the difference across to the students.

What do you think the long-term effect of strong directional selection would be on $h^2$, $V_A$, and $V_F$.

What was the gist of Boag and Grant’s result regarding the effect of the 1977 drought on Darwin’s ground finch?

What are breeding values? How are they involved in the calculation of Additive Genetic Variance? How are breeding values calculated?

Assuming we start with a rare, dominant mutation that is favored by natural selection. Why does the additive genetic variation change over time as the mutation spreads to fixation? Why does the dominance variation also change over time? Why does heritability change over time?

Across modeling strategies.

In your opinion, what are the strengths and weaknesses of using the following kinds of models to study evolution by natural selection: population genetic models (1-locus, 2 allele); quantitative genetic models; discrete games models (e.g. the Hawk-Dove game), and continuous strategy models (e.g. sex ratio models). Which would you use to study the evolution of anisogamy (unequal sized gametes)?