You've got over a week to answer this exam. It's due no later than Friday, April 2nd. We do not have class that day, so if you want to turn in a paper copy, please do so by Thursday afternoon.

Be as clear as possible. I recommend writing a full and thorough response to the question, then editting it down to meet the word limits. Because of the time and resources available to you, vague, rambling answers with incorrect statements contained within garner basically no credit. I interpret each answer you give as the best version you can. So **edit** your responses to be as clear as possible. It's easiest to write in your own speaking voice. I recommend reading your answers aloud to yourself and, ideally, to others.

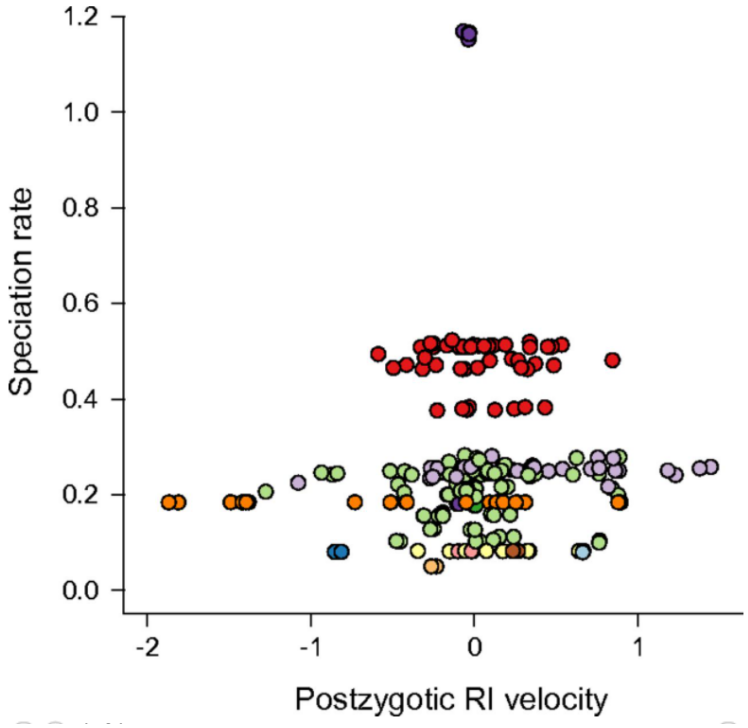
Don't get things wrong. In your answer, don't say untrue things. This is harder to do than it seems–I recommend avoiding jargon, as it's easy to misunderstand what a technical term means, resulting in a factually incorrect statement within the answer. Unless you're feeling really confident, try to be circumspect. I assume that anything you choose to write down is something you've decided to provide as evidence to me that you understand the material.

Correlation does not equal causation. It's a lot easier to show that two things both change in the same way than it is to show that changes in one thing *cause* changes in another. This relates back to the above advice about not getting things wrong. Be circumspect! Be skeptical!

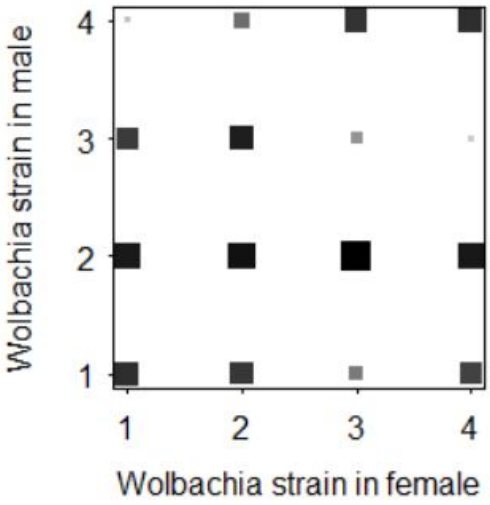
You must each turn in your own assignment. You can work together to discuss potential answers, but the answers you turn in to me must be in your own words. Plagiarism/copying is the only way to cheat, any resource is fair game, any amount of working together is also fair. Just don't directly copy.

Good luck!

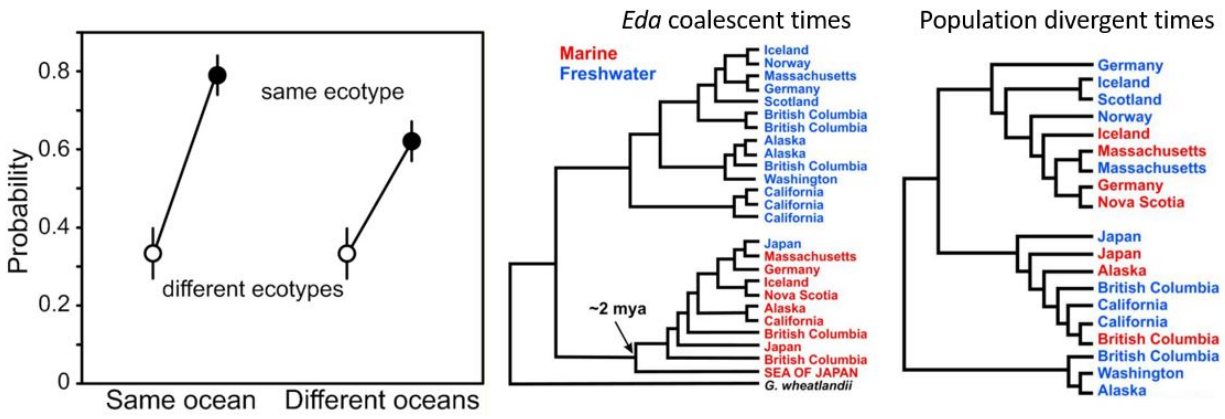
1. (1 point) *Sunflowers are fascinating plants. They can orient themselves to the sun over the course of the day, live in a wide-variety of habitats, and are incapable of self-fertilization. There are many species of sunflowers in North America, with the two most widespread being* Helianthus annus *and* Helianthus petiolaris*. If taken into a lab, less than 1% of crosses between these two sunflowers produce viable seeds, and the hybrids that result tend to have very low viability (around 5%). That is, 1% of the time you cross them you get viable seeds, then only 5% of those viable seeds are capable of growing into a plant that can also produce offspring. In the deserts of the southwest, among the dunes and in the salt flats, you may also find a few other strange sunflowers like* Helianthus anomalus*. The DNA of these desert sunflowers is about* 50% *identical to* H. annuus *and the other* 50% *of their DNA is identical to* H. petiolaris*. The genes in the desert sunflowers are found in long blocks (haplotypes) identical to similar chunks from the two widespread species, with many loci identical to one sunflower followed by a long stretch of loci identical to those from the other (think of long alternating bands). However, these desert sunflowers also often have large inversions and translocated genes. Cross-breeding the desert sunflowers to either of the two widespread species typically doesn't work well, with fewer than* 10% *of the offspring being viable, although the desert sunflowers breed just fine (*$\geq$ 90% *seed success, similar to the other species).* (a) What is the probable origin of the desert sunflowers? (b) What is the likely explanation for the "large blocks" of widespread sunflower DNA in the desert sunflowers? (c & d) Please briefly ($\leq$ 100 words) explain the importance of the inversions, translocations, and large blocks to understanding the origin of the desert sunflowers.



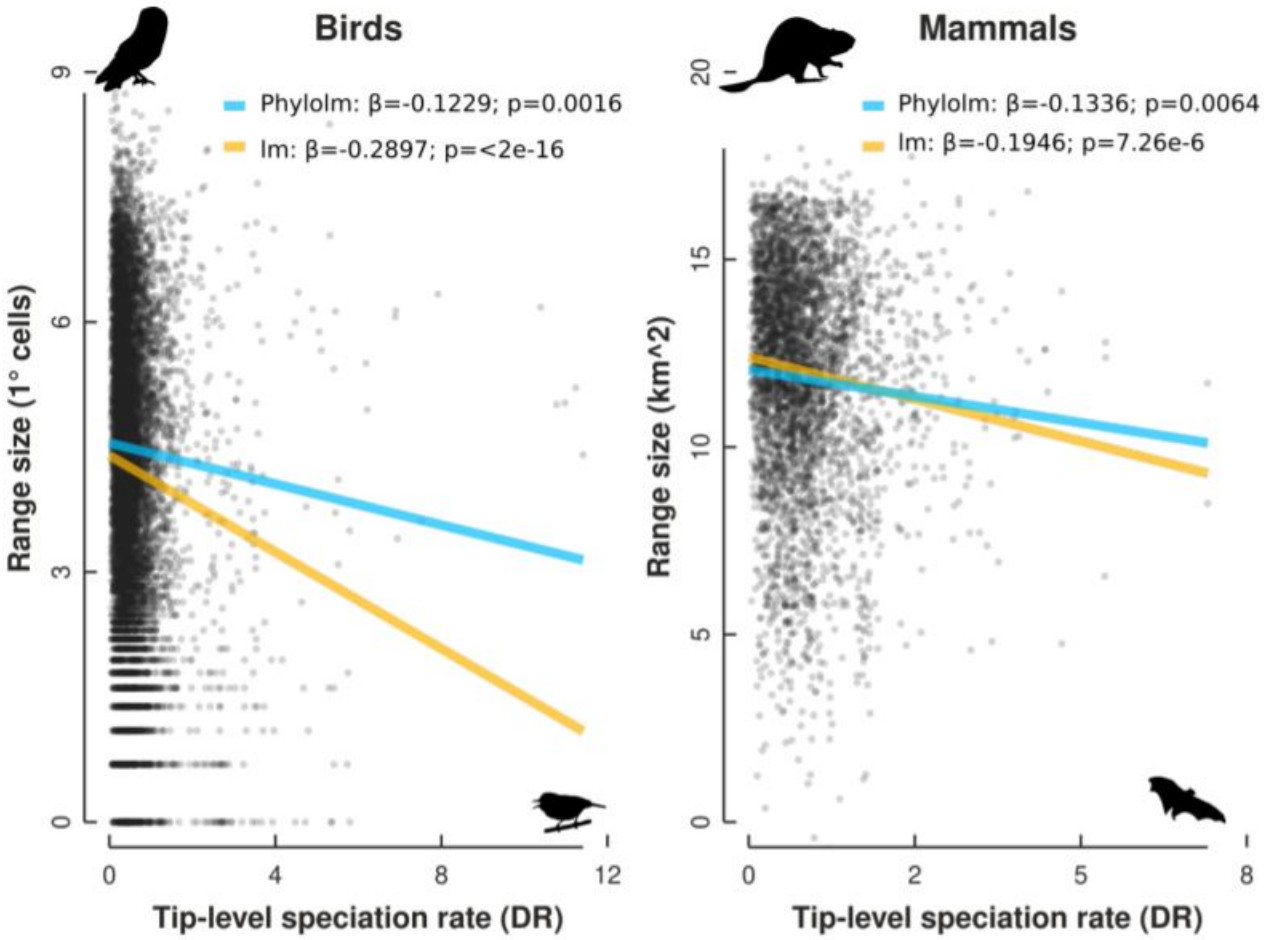
2. (1 point) *Above are data for 244 species of bird color-coded by clade from a 2013 paper by the Daniels Matute & Rabosky. The x-axis shows the estimated speed at which postzygotic barriers form in a specific species (based on rates of geneitc differences between populations, basically think F_{st}; lower numbers = more genetic differences). The y-axis shows the estimated speciation rates (based on the length of the branches that lead to each species; lower numbers = longer branches;* n.b. speciation rate is defined the same way anywhere else you see it in this exam*). In the same paper, they found essentially identical results across the many species of* Drosophila *fruit flies. Further, another study by Dr. Sonal Singhal and colleagues in 2018 found similar results when they compared F_{st} in Australian lizard populations to speciation rates.* Please clearly explain to me how you interpret the meaning of these results in 500 words or fewer.



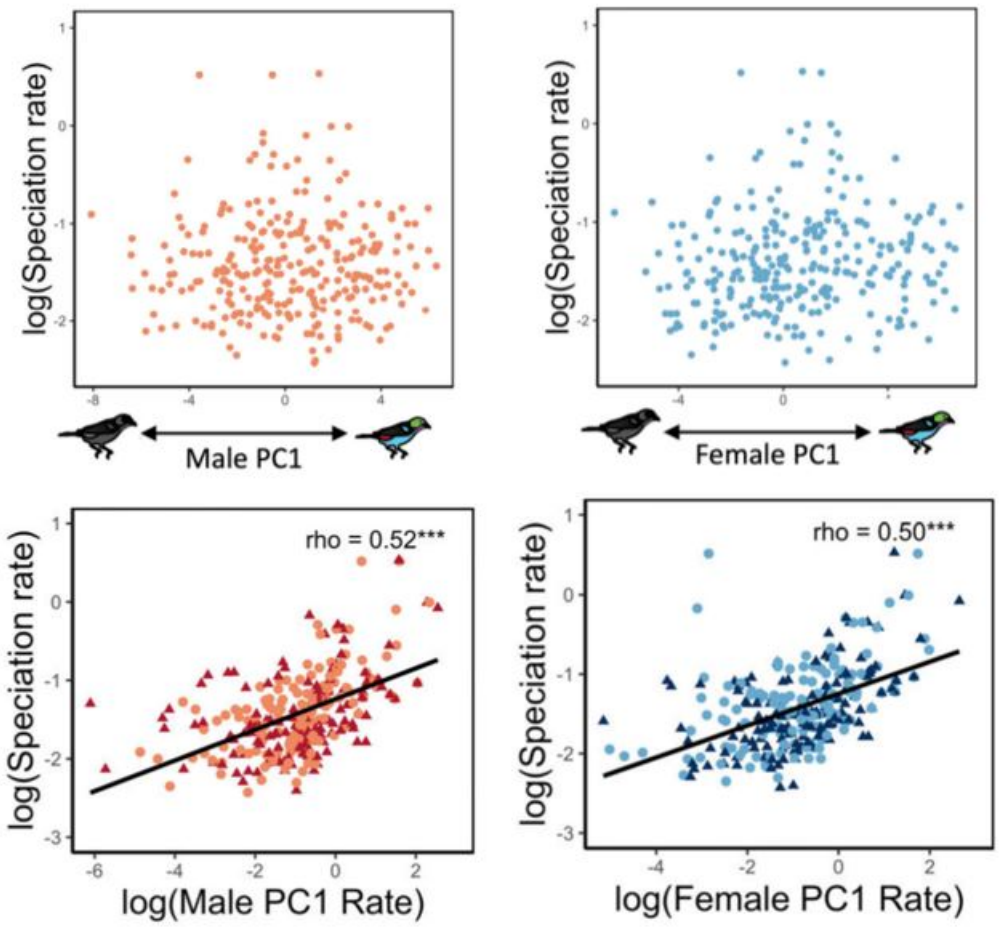
3. (2 points) Wolbachia *is a profoundly strange bacterium. It infects insects, but it does so by actively invading their cells and living in their cytoplasm. When* Wolbachia *invades the germline cells, it can be passed on by the female to her eggs. That is, baby insects inherit this bacterium from their mother. Further, it has evolved the ability to manipulate gene expression in the insects it infects, which can make the cytoplasm of the sperm from male insects incompatible with the cytoplasm of female eggs if one or the other is uninfected or infected with a different strain. A study published early this year by Sicard et al. examined how different strains of* Wolbachia *present in mosquitoes of the species* Culex pipiens *impact the hatching rates of their eggs. They brought males with one of four different* Wolbachia *strains, and females with one of those same four strains into a lab, cross-bred them, and analyzed the hatching rate of their eggs. The data shown above reveal the success rate by cross. The x-axis shows the strain infecting the female mosquito, the y-axis shows the strain infecting the male, and the size and darkness of the points plotted are proportional to the hatching success (defined as the proportion of crosses with* $\geq 78\%$ *hatching success). Wolbachia* strains vary in frequency by region. How do you expect *Wolbachia* strain to impact (a) the linkage disequilibrium in *Culex* mosquitoes? (b) mutation rate in *Culex*? (c) gene flow in *Culex*? (d) local adaptation in *Culex* mosquito genes?



4. (2 points) *Sticklebacks are fascinating little fish. They're small and they live mostly in the oceans of the northern hemisphere. However, they breed in streams, and since the last Ice Age ended about 10,000 years ago, several populations invaded the large lakes that formed from the melting of the glaciers. Some also have taken up permanent residence in streams, giving up the return to the ocean. Once adapted to a freshwater setting, the fish rarely (if ever) return to the Ocean, and so cannot directly immigrate to other freshwater basins. Stickleback ecotypes refer to whether they live in marine or freshwater settings. In these freshwater systems, their primary predators are insect larvae such as dragonfly nymphs, which means that the spikes and bony armor that serve them well in the ocean (where fish eat them) do not work as well in freshwater. So freshwater sticklebacks tend to be smaller, with fewer bony plates and fewer spines than their oceanic relatives. The* Ectodysplasin *(*Eda*) gene has a major influence on the degree of armor. Above are data from one of many, many studies on sticklebacks showing their mating preferences in lab studies (left), a pattern of coalescent times for the* Eda *locus in different populations (multiple samples from some populations), and the pattern of population-level divergence times.* (a - c) Provide three distinct but possible explanations for the discord between the population divergence and coalescent times brief (≤ 50 words for each explanation, so ≤ 150 total words). (d) Which of your answers from a-c is most likely? (e) Please describe a clear and specific dataset that could test your answer for part d.



5. (2 points) *The rate at which reproductive incompatibilities accumulate between populations without gene flow has been a contentious topic for decades. As mutations accumulate within a lineage, some of them will be incompatible with alleles at other loci, resulting in epistatic incompatibilities. Yet the way this must work is that different alleles at different loci that interact epistatically have to go to fixation in the two populations. A recent study modeled this in a demographic framework, and the data shown above compare geographic range size to speciation rate for birds (left) and mammals (right).* (a) What fitness effects do most mutations have? (b) Why might you expect the likelihood of a fitness effects for potentially-incompatible mutations to be different from the average mutation? (c) What force(s) have the strongest impact on the probability these incompatibilities go to fixation? (d) Please explain how the graphs reflects the relative importance of the force(s) described in part (c).



6. (2 points) *Shultz and Burns (2017) used spectrophotometry to measure the precise wavelengths of color coming from major feather patches from a 355 species of tanagers (Thraupidae). They used principal components analysis to reorient their data so they could analyze the axis of maximal color variation. More recently, Dr. Rosalyn Price-Waldman collaborated with them and used phylogenetic methods similar to what you did in your R assigment to calculate the speciation rates for the tanagers, and compared those rates to the plumage coloration (top row) and to the rate of change in plumage coloration (bottom row). Basically, for each species, they (1) measured the actual color of the bird, then used an evolutionary tree to estimate (2) the probability it would speciate soon, and (3) the probability it's colors would change soon. The top set of figures shows speciation rate against color, while the bottom plot shows speciation rate against the rate of color evolution.* Based on these data, please give a concise ($\leq$ 100 words) explanation of how color effects speciation rate in tanagers.