

Questions on Antonovics *et al.* (2002) & O'Keefe and Antonovics (2002)

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1. In Antonovics et al (2002), the author states that "over-lapping species distributions of a potential new host with the old host is the most obvious prerequisite for a host shift", as was seen with the two plants in the genus *Silene*. Does this occur in wild populations of animals as frequently or less frequently than it does in plants?
2. Have host shifts occurred in agricultural areas where soybeans have replaced most of the other major crop types on the landscape?
3. Did you consider using a more natural closed setting (e.g. a greenhouse and bees) to pollinate your flowers, instead of inoculating the flowers in a lab with teliospores in water?
4. How would you set up an experiment to detect disease shift between two populations of mammals with an intermediate host? For instance with bird flu, where it is hypothesized that genetic shift occurs when swine are infected by bird influenza, which then can cause the virus to pick up human host specific capabilities and be transferred?
5. Could an in vitro study be set up to study the evolution of virulence in sterilizing pathogens, if so how?

6. In O’Keefe and Antonovics (2002) it is stated that ‘The equilibrium densities of susceptible and infected hosts will change if β , α and ρ change by evolution of the pathogen.’ What if the background mortality rate μ was changed through intervention? The intervention could be related or unrelated to the pathogen and still have an effect on the host densities.

Answer: In most studies where α and β are coupled and have a positive relationship, increasing μ tends to increase their optimal values. Conceptually, this is because the value of transmission events late in the infection become less valuable because increasing μ decreases the probability the host will survive to a later time point. The reduction in importance as a function of time is exponential (i.e. survivorship to some time t after the start of an infection $\sigma(t) = e^{-(\mu+\alpha)t}$) so relative changes in $\sigma(t)$ when t is small are less than relative changes when t is large.

7. O’Keefe and Antonovics (2002) state ‘the pathogen with the β , α , and ρ that minimize S^* , if it exists, is uninvadable.’ Is that uninvadable by any other pathogen or just uninvadable by a different mutant of the same species?

Answer: In general, it means members of the same species. However, in such model you can not have multiple infections so the more general answer is any competitor that cannot coexist within a host with the other competitor.

8. Antonovics *et al.* tested transmission between old and new host species, is it possible that as further tests continue the anther smut will develop mutations in one species that make it more pathogenic to the other species?

9. Antonovics says that they assume no relationship between infection-induced host mortality and pathogen transmission in sterilizing pathogens. Isn’t the relationship an evolutionary one that resulted from low transmission rates and therefore a need to

exist longer in the host (i.e., to become more benign)? It seems to me that we cannot expect the system always to evolve toward some intermediate equilibrium of virulence and transmissibility as Antonovics suggests, and that there is likely a relationship (now weakened to a great extent via evolution) between host mortality and pathogen transmission in sterility-causing pathogens.

10. "In other words, this simple model predicts that if host fecundity declines with increasing pathogen transmission, then selection on the pathogen will cause host fecundity to decline toward 0." Doesn't this also cause transmission to decline toward 0, thereby decreasing the fitness of the pathogen?

Answer: Only in the cases where it drives the host to extinction which for this study appears to happen, but rarely. Reducing the fecundity of the host may reduce the number of hosts and, thereby, transmission events. This reduction appears to be especially important in the spatially explicit case, but the transmission rate itself β increases with increasing ρ . However, it's good to keep in mind that all of the competitors are affected by this reduction in host density as well, not just the most virulent one.

11. In contrast, in the sterilizing pathogen, there is no fitness cost associated with high transmission. For sterilizing pathogens, we assume that the transmission rate b is linked not to a but to the infected host fecundity r . However, r does not appear in equation (2) nor, therefore, in S^* (3). As S^* decreases and b increases, infected host fecundity r evolves downward because of its relationship with b ." I'm not sure I understand this—it seems like circular logic. If you didn't include the term in the model, of course it's not going to show up in the model. Can we be sure that p has no fitness cost?
12. Why would the pathogen effects on host fecundity be so drastically affected/selected

for in the spatial population structure compared to the non-spatial one? Is this because of spatial structure allows for increases in β and ρ without fitness cost to the pathogen?

Answer: Actually it's because when there is spatial structure in the distribution of offspring and transmission reducing the fecundity of your host reduces the number of susceptibles in the future but, unlike in the well mixed system, your offspring are the one's most likely to be affected by this host reduction. As the authors note it can be thought of as a form of kin selection. As a result, I expect if you were to only include spatial structure in the distribution of host offspring, this effect would disappear.

13. What prevents the presence of different transmission rates with a polymorphism from multiple genotypes in this model?

Answer:

14. Why would decreases in host fecundity benefit the pathogen? Would normal, or even increased, host fecundity not produce more potentially susceptible hosts for pathogen colonization?

Answer: This is an interesting question because it highlights a number of implicit assumptions in this and other evolutionary models. One idea is that host's have more control over their maximum level of reproduction than a pathogen and they've set it to some optimal value based on a trade-off between the allocation of resources to reproduction vs. survivorship. A second idea is that, like host survivorship and host reproduction, pathogen transmission requires host resources. So even if a parasite could (and I'm sure there's some that can) increase a host's reproduction rate and still transmit, it would be achieving this goal by reallocating resources from survivorship to these other purposes. As you might

imagine, whether or not this would be evolutionarily favored will depend on how host fecundity, host survivorship, and parasite transmission change with resource allocation.

15. In the article "The ecology and genetics of a host shift: Microbotryum as a model" - the new host (*S. vulgaris*) is more resistant to anther-smut disease than the old host (*S. alba*). What is the reason for the resistance?
16. Is there any reason why some pathogens are able to persist on new hosts while others are not?
17. According to the article "Playing by different rules: The evolution of virulence in sterilizing pathogens" - the well adapted pathogens are expected to inflict intermediate or severe damage on their host. Could you explain the reason?

Answer: If I interpret the idea correctly, you're referring to non-sterilizing pathogens.

The result rests on the idea that there is a trade-off between increasing a pathogen's transmission rate and its virulence rate. For most realistic forms of this trade-off it means that the virulence level that minimizes $S^* = (\mu + \alpha)/\beta(\alpha)$, is an intermediate or high value.

18. Have there been any taxonomic studies on Microbotryum in the U.S? How did results from this cross-inoculation study compare to other U.S. or regional studies?
19. Explain the basis of pulsed-field gel electrophoresis.
20. Aside from the facts that Microbotryum "has little direct social impact, and is readily amenable to experimental manipulation", why use it as a model system (i.e. how can we apply findings from manipulated experiments with a single species of smut and a single plant host to "real life" problems with many other plants and animals)?

21. Antonovics et. al. make the poorly founded statement that "many principles and processes we have outlined for *Microbotryum* are also applicable to host shifts onto economically important plants and animals as well as onto man." and then they give an example about what might have happened with AIDS research if we knew then what we know now... So, are there any real examples or real data to show how this study of an artificial system using one particular manipulated organism actually applies to processes seen in other organisms, or is this an example of one of those models that was developed by someone who simply wanted to "play with all kinds of interesting models"?
22. WHY DOES THE WORD VIRULENCE HAVE MORE THAN ONE DEFINITION?!?!?
Seems to me that we should follow whatever definition is in the dictionary for virulence, and then come up with other terms to describe that which is not [fill ONE definition of virulence here].
23. Is it possible, and in our best interests, to "make a priori statements about the likelihood of future host shifts."? p. S40 It was mentioned that previous studies were not able to do this. Is this something that modeling is moving toward or is able to do?
24. It is mentioned that the fungal spores are transmitted via insect pollinators and that the insects are less likely to visit malformed flowers affected by the *Microbotryum*. Won't the insects, too, be able to differentiate between pollen and spores? I would think that if the insects are avoiding flowers with obvious deformities that the transmission of spores would be incidental in regards to pollinators and due instead to wind.
25. Both *S. alba* and *S. vulgaris* are native to Europe but also occur in eastern North America p. S41. Do we know when introduction of these species occurred in the US? And if the range is spreading west? Do they out-compete any native wildflowers in the eastern US?

26. In Antonovics et al (2002) he author states that "Very few population-level studies have explained why some pathogens are able to persist on new hosts while others are not". What does she attribute this 'hole' in the information to and what are some other methods of attacking this scientific question?
27. pS49 bottom rt column: I am confused by the statement: .."the field data would have pointed to the need for an impracticably large number of target plants...to detect any transmission at all". If this statement is referring to calculations using the model, what data was left out that did not account for the transmission found in the experiment. Is it 'back transmission' or an immunity developed due to longer term exposure than predicted?
28. O'Keefe and Antonovics (2002): Can you better explain how and why the spatial structure of the host-pathogen population is vital to predictions about virulence evolution...(I think I might need a whole course on this topic!)
29. In the 'Ecology and Genetics of a Host Shift: Mycrobotryum as a Model System' paper says that sexually transmitted diseases are less likely to be transmitted across species. The reason of it could be that is not common between different species to have sexually interaction or because they are not able to replicate inside the new host?

Answer: I think the first answer is correct one. Different types of interactions between members of the same or different host species have different transmission rates. In animals, sexual interactions are almost exclusively restricted to members of the same species. Plants are a bit different and in the case of Mycrobotryum it can be transmitted inter-specifically if the pollinator visits different species of plants.

30. How does a pathogen that does not affect host longevity will be favored by mixed populations? Why is the sterilizing effect of pathogens neutral in this population?

31. There are several definitions of virulence even between pathologists. Which are the main disagreements that the verbal and mathematical areas have of this concept?
32. Is it really a host shift if the *Microbotryum* cannot maintain itself on the new host and continually needs to be replenished? Or is this just a case of the 'new host' being surrounded by so many infected plant that you find the disease organism on it.
33. Given the model in O'Keefe and Antonovics article, how would they predict the virulence of the bacterium *Wolbachia* to evolve? It only sterilizes some of the population and allows others to continue to reproduce even though they are infected.