

Questions on Gaff & Gross (2007) & Childs *et al.* (2000)

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1. Is eradication of the tick really even necessary? What is wrong with continuing to use ivermectin during the allowed months in order to suppress the tick population?
2. Sounds like the control with ivermectin was implemented in the Cumberland Co. case in order to get quick results to please the public...was this the case? Are the results a good indication that ivermectin is a good control method to use regularly, or was this case an exception to the rule?
3. I was under the impression that rabies was much less common these days...How often does rabies affect human beings—or even dogs in the U.S.?
4. I know this is off the subject, but Gaff's introduction mentions several diseases and state the pathogens that cause them include: viruses, bacteria, protozoan and rickettsia. One of the diseases he mentions is tick paralysis. Is this caused by a microorganism? I've only heard of it being caused by proteins in the tick's saliva. Is one of the microorganisms mentioned shown to cause tick paralysis?
5. What is a fourth order Runge-Kutta method?

Answer: Runge-Kutta is a general method for solving differential equations developed in the early 1900's. It works, in part, by estimating the slope of a function over

a step size Δt by evaluating the slope of a proposed solution at a number of different points and then averaging them in a weighted manner. The order of the method (here, fourth order) refers to relative error in the solution expected using the method. In general, for a fourth order method each step of size Δt has an accumulated error of order Δt^4 . Thus a 3rd order routine has more error in the solution than a fourth order.

6. Why is it assumed that tick mortality increases during the winter months? Lone star ticks go into diapause in the winter and become active in the spring. Can you assume a decrease in clutch size if the ticks are not reproducing during winter months?

Answer: I suspect that its assumed that most organisms which enter some kind of diapause may have a higher mortality rate due to the fact that they are immobile (thus perhaps more susceptible to predation) and no longer feeding (thus susceptible to starvation). It also seems likely that entering and exiting diapause is 'costly' (though less costly, when summed across the diapause period than not entering diapause)

7. It seems that fluctuations in rabies incidence among red foxes are influenced by the density of populations meanwhile with raccoons the incidence is not related with this factor. If this is correct, what factors cause the differences in rabies propagation amongst the two species?
8. The authors indicate the best model performance when raccoons are assumed to have little or no rabies immunity. Are raccoon populations actually able to develop some level of immunity? Is this related to the host or the virus? If it is the virus, could it be that it has a low immunological property?
9. In *Modeling Tick-Borne Disease: A Metapopulation Model*, the author says that tick-

borne diseases have had an increasing impact on human health during the past hundred years. Is this because of an evolution process or because of encroachment of human activity into wilderness?

Answer: I suspect it is likely do to the increased total number of humans that spend time in wilderness and the fact that, due to our overall increase in health and technology, more aware of these diseases. Given that we are not their primary hosts I think it is unlikely they have evolved to in direct response to our presence.

10. How well can these models be applied to Rocky Mountain spotted fever and Lyme disease?

Answer: This is a somewhat vague question and so I'll respond in a similar manner. I'd say it depends on how much the biology of the host, vector, and pathogen differs between these diseases and Ehrlichiosis. It also depends on how you specifically hope to apply these models. If the biology is similar and the questions are similar then I suspect it will apply, though the parameter values might be quite different.

11. How is the corn made available to the deer and how much is available? Could this produce ecological consequences similar to that of the eradication of a tick species?

12. In order to make useful predictions, how does one determine the initial conditions and the parameter values for the tick and host populations?

13. When designing a model, how do you decide which variables to account for and which to leave out? For instance, the Ehrlichiosis model did not include variables like 'host size in relation to [M] value', 'min tick attachment time required to transmit the pathogen' or the fact that multiple tick species can transmit Ehrlichiosis (*Amblyoma americanus*, *Ixodes persulcatus* and *Dermacentor virabilis*).

14. Assuming that there is more to this than the obvious (e.g. mosquitoes can fly, ticks cannot), why is spatial diffusion in relation to aspatial/partial differential equation models more appropriate for mosquitoes than ticks? (pg. 267)
15. Did the multiple attempts at trying to halt the spread of raccoon rabies (e.g. use of baiting and oral wildlife vaccines, etc.) during the study's time frame affect your data? What about the effects of post-vax immune status on antigen testing?
16. What effect on the ecosystem does summer feeding of deer have? Are they fed enough to become dependent on the corn? Does long-term exposure to the acaricide have any impact on the deer? Does long term exposure to the acaricide lead to selective pressure on the ticks that could lead to resistance?
17. In the rabies article they mention that the minimum duration of an epizootic was 5 months in order to reduce the effects of short term fluctuations, could rolling averages have been used to reduce fluctuations, yet detect shorter outbreaks?
18. What was so different about rabies in West Virginia that EA2 did more poorly there, while it did a good predicting job in other areas?
19. According to the article "Modeling Tick-Borne Disease: A Metapopulation Model", approximately 70% of tick's eggs survive and half of the population is female. Is there any reason why half of the population is female?

Answer: Most organisms have a 50-50 sex ratio (or close to it) at birth. There are many theoretical studies exploring why this commonly is the case and when we should expect it to differ dating back to R.A. Fisher 1930).

20. Why (is) tick to deer infection much lower than to deer to tick?

Answer: I'll take a stab at it, but I suspect it has to do with the fact that the flow of materials from the deer to the tick is much greater (both on an absolute and relative scale) than the converse.

21. Why immunity to rabies in natural populations of raccoons has never been established?

Answer: I'll just note that humans are also unable to develop immunity to rabies in the natural world. That's because the mortality rate/infection is, in the absence of prior vaccination, essentially 100%

22. I know that several state wildlife agencies (especially out in the mountain and plains states) have sampled for Chronic Wasting Disease at check-in stations over the past few years. Have researchers attempted to model its dynamics based on this type of surveillance data? Would the models be similar to those used in modeling epizootic rabies?

23. In the paper by Gaff and Gross, it seems simple and applicable to feed deer the acaricide-laced corn as a form of control in smaller, more isolated "patches" of habitat. However, do you think this form of control would be applicable across all types of habitat (e.g., riparian corridors stretching for many miles)? I guess I am thinking about situations in ag-producing states where areas that have large populations of ticks are completely surrounded by agricultural fields. These deer already have all the food resources they need to survive and the applicability of this form of control in these areas seems minimal. Are other forms of control used in these areas and have any models incorporated habitat variables such as this?

24. From Childs et al.: I don't understand why they define an epizootic as a time when numbers of rabid raccoons are greater than the county median. Doesn't this make the classification of rabies as enzootic vs. epizootic different for different areas? (i.e., if

rabies persists at high levels for long enough in a given county it would be classified as enzootic whereas if rabies increased from low to moderate levels in another county it would be considered an epizootic. It seems as if this makes the models difficult to interpret if one is interested in characterizing the threat that is posed by rabies in a given area.)

25. From Gaff and Gross: "the growth rate for hosts (beta) was set to an arbitrary value of 0.2." Why choose an arbitrary value? Why not leave this term out of the model if it is unknown?

Answer: Often modelers are faced with terms that are known to exist but their values are unknown. It would actually be impossible to leave this term out in the sense that if it was neglected, that would be equivalent to assuming a birth rate of 0. Given the fact that deer give birth and some of them survive to adulthood, we know such a value would be incorrect. Further, because there is a death rate, it would also mean that the deer population would decline towards zero.

26. From Gaff and Gross: "The model implies that control needs to be used in an area for longer than 5 years to reduce the disease to extremely low levels" Could a wildlife manager then stop treatment for a period of time? What are the risks of ivermectin to the public (e.g., if you ate venison with ivermectin residue, would it do more than simply de-worm a person)? What about the risks of ivermectin-resistance (especially if treatment is terminated 2 months prior to deer season each year)? What about other disease risks (Bovine TB, chronic wasting disease) associated with baiting deer? Would increasing the risk of these be a worthwhile trade-off to control tick-borne diseases?
27. "The model implies that control needs to be used in an area for longer than 5 years to reduce the disease to extremely low levels." p 281. Is this a feasible treatment options

for home owners, cost wise? Or is this a treatment strategy that we would most likely see on public grounds?

28. How does a person get involved in modeling? Was it an interest from an early age or did you have an experience that clicked for you?

Answer: I can't answer for Lou or Suzanne, but I got interested in it once I found out about it as a graduate student. Its appeal to me was the great insight that one could gain through the use of the clarifying formality of mathematics (n.b., before grad school the highest math I'd had was 'Calculus for Life-Science Majors').

29. Besides the tick-borne disease model, what other models have you worked on and what was the most exciting project for you?

30. Could surveillance data pertaining to other wildlife reservoirs (skunk, fox, bat) be modeled accurately enough to 'layer' or conjoin the data to the raccoon results such that a broader picture of wildlife species interaction (or transmission) might be detected?

31. How can the amount of ivermectin consumed per deer be controlled? (quantity/over time)

32. Ivermectin is a cornerstone anti-parasitic drug used for livestock and pets. Its use on wildlife/deer presents a major issue with regards to parasite resistance and possible effects of resistance on 'owned' animals in the community. (ie. animals with potential tick burdens come into direct contact with people on a regular basis).

Answer: I'm understanding of the situation is that the evolution of drug resistance is unlikely to occur in the wild under scenarios presented in the paper relative to the probability that it will occur in human managed livestock where the overall usage is more frequent, at higher doses.

33. Since the model takes into account host numbers (N) and growth rate of host (β) could hunting or culling of deer provide an effective control mechanism? For example: Should studies on the density of host (deer) without 'predation pressure' factor into how the deer are controlled as well? (ie. Should herds of deer really be roaming backyards and golf courses)

Answer: Increasing hunting pressures may be another option for control. Can you imagine how this might be easily incorporated into the model? Note that hunting, as with chemical control, will have its own set of costs and benefits associated with it. For example, most folks don't want hunters shooting into their yards or golf courses.

34. Using linear regression, it was found that the epizootic periods decreased by approximately 5 months between each successive epizootics. What could be the possible reason for such a trend?
35. In context of construction of viable models, could you elaborate the meaning of the terms: Metapopulation Models and Cellular automata.

Answer: A Metapopulation is a set of loosely interconnected subpopulations. The dynamics and events of the subpopulations are thought to be independent of one another in the absence of any migration. Allowing migration then begins tying these populations together. At the limit of very high migration, the metapopulation acts essentially the same as a single population. Cellular automata is a modeling that relies on the use of discrete 'cells' that take on different states. When applied to biological systems those cells often represent individual organisms or, depending on the scale of the model, biological cells.

36. The results dealing with the local dynamics of the epizootic rabies racoon indicated that

most counties that had detectable epizootics(55%) had only a single epizootic whereas counties involved earlier in the epizootic frequently, experienced multiple outbreaks. Why? Is that related to their proximity to the site of initial introduction of rabies racoon or are there other controlling factors.