Math 19a, readings review

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1 The case of the missing meat eaters

This article discusses the interesting fact that Australia lacks big mammalian predators. The author argues that the harsh climate imposed by the southern oscillation factor (El Niño), infertile soil due to the lack of geological processes this old continent and other environmental factors reduces the density of herbivores thereby making it very hard for large mammalian predators to survive because they require much more food and have big penalties for competition within species. However, Australia is very hospitable to a remarkable variety of big reptiles, who need much less food and can sustain longer without it as they don't need to maintain body temperature. This decreases their death rate (σ in the model below) and increases their benefit from the prey (efficiency, λ here).

In this case, the usual Lotka-Volterra predator-prey model applies:

$$\begin{aligned} \frac{dk}{dt} &= \alpha k - \beta k^2 - \gamma pk \\ \frac{dp}{dt} &= -\sigma p + \lambda pk, \end{aligned}$$

where k is the number of prey and p the number of predators. This model is extensively analyzed in Chapter 12. To summarize, in the case that $\alpha/\beta < \sigma/\gamma$ (either the death rate σ is large or the benefit from prey λ is small), the point $(k = \alpha/\beta, p = 0)$ is the only stable equilibrium in the first quadrant (biologically significant values), so in this case the predators will go extinct. In the other case $\sigma/\lambda < \alpha/\beta$, the stable equilibrium point is $(k = \beta/\lambda, p = \alpha/\gamma - \beta\sigma/(\lambda\gamma))$ and $p > 0$, which shows that predators with sufficiently low death rate (σ) and/or high prey benefit rate (λ), like reptiles, will survive.

2 Malaria: focus on Mosquito Genes

Malaria is one of the most common infectious diseases and an enormous public health problem. It is a vector-borne infectious disease caused by protozoan parasites. It is widespread in tropical and subtropical regions throughout the world. Each year, it causes disease in approximately 515 million people and kills between one and three million. The disease is caused by protozoan parasites of the genus Plasmodium, transmitted by female Anopheles mosquitoes. So far fighting the disease by exterminating these mosquitoes or by developing vaccines have been unsuccessful. This article proposes a new approach - creating transgenic mosquitoes resistant to malaria, whose resistance gene is attached to a highly movable part in the genome thereby increasing its chance to be passed among the population.

There are several different ways of creating resistant (inhospitable to malaria) mosquitoes, one of them is stopping the parasite from replicating on each of the three stages of reprodution or by simply making the mosquito hypersensitive to the parasite, dying instantly when infected and hence not allowing time for the parasite to develop. Both techiniques were unsucessful because genetically modified mosquitos were less fit than the wild ones, therefore, exponentially decreasing with time the number of resistance/hypersensitive genes in the population. A new approach suggested by researchers was to give the genetically modified mosquitos a little help enhancing the chances of passing the gene to the next generation by inserting the gene

inside mobile elements of the genome called transposable elements. These replicate themselves (most likely through an RNA intermediate) over the genome increasing their number of copies and enhancing the likelihood that the gene will be passed on. With that we could hope that the number of resistance/hypersensitivity genes would increase over time and start to spread over the population of wild mosquitos. There is a question on this article made by the books author: `Can the spread of the gene over time be modeled by an Advection Equation'. The answer is no. Advection equations model situations where there is one resultant force driving the spread of the subject (like the wind in the example given in class). In this example, there is no obvious direction for the spread of the resistance/hypersensitive gene throughout the continent. Therefore it could not be modeled by advection. If one considers the spread completely random (ignoring migration behaviour of the mosquitos) then it could be perhaps approximated by a diffusion model.

If everything was happening at one place, say in a closed room, then time would be the only variable and we can write a simple logistic equation for the proportion of resistant mosquitos in the entire population ($p(t)$) as

$$\frac{dp}{dt} = rp(1-p),$$

assuming that the resistant mosquitos will prevail (so 1 is the stable equilibrium here). If we consider the same situation over, say, Africa, then the resistant mosquitos will not only multiply at one place, but also diffuse over the whole continent. In this case their portion will be a function not only of time, but of location (x) also, denote it by $u(x,t)$ The rate of change in proportion will be equal to the proportion of resistant mosquitos which diffused across position x at time t + the proportion of resistant mosquitos that were created (birth - death) at this spot and moment, which is by the previous equation $ru(1-u)$. We suggest the following model (called Fisher's model):

$$\frac{\partial}{\partial t}u = \mu\frac{\partial^2}{\partial x^2}u + ru(1-u), \tag{1}$$

where μ and r are just constants. We can try to find a solution for it in the form of a traveling wave: $u(x,t) = f(x+ct)$ (c- constant, to be determined). Substituting this u in the equation we find an equation for f

$$c\frac{df(s)}{ds} = \mu\frac{d^2f(s)}{ds^2} + rf(1-f).$$