Adaptive Dynamics Course
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The Evolution of Virulence – II

A population of hosts grows, in absence of the disease, according to the simple ODE

$$\frac{dN}{dt} = \left[b(N) - d \right] N$$

where N is population density, $b(N)$ is the density-dependent birth rate, and d is the death rate. We consider a disease that causes lifelong infection (no recovery). Infected individuals die at a rate $d + \alpha$, where α, the extra mortality caused by the disease, is called the *virulence*. All individuals are born free of the disease. The disease is transmitted between infected and susceptible individuals at rate β, such that the dynamics of susceptible (S) and infected (I) individuals are given by

$$\frac{dS}{dt} = b(N)N - \beta SI - dS$$
$$\frac{dI}{dt} = \beta SI - (d + \alpha)I$$

with $N = S + I$.

The disease increases the rate of mortality because the pathogen is using resources to multiply in the body of the host and because it produces symptoms (such as coughing, bleeding, etc.) which enhance its transmission to other host individuals. Faster replication in the body and more symptoms ensure a higher transmission rate but cause higher mortality as well. We thus assume that β is an increasing function of virulence. Note however that β cannot exceed the contact rate between individuals, hence the function $\beta(\alpha)$ will saturate for large values of α. Different strains of the pathogen differ in their virulence, α, and in the associated transmission rate, $\beta(\alpha)$. We say that a strain is *viable* if it can spread in a disease-free population.

In this project, we study the consequences of *superinfection*. When superinfection occurs, a second strain of the pathogen infects an already infected host. Because more virulent strains replicate better in the body, it is expected that more virulent strains generally replace the less virulent strain within the host. We assume that the within-host dynamics is fast such that this replacement can be considered instantaneous. Upon superinfection, strain α_2 takes over a host infected by strain α_1 with probability $\rho(\alpha_2 - \alpha_1)$, where ρ is an increasing function of its argument.

There are very interesting results on different evolutionary scenarios depending on whether ρ is continuous or not and whether it is continuously differentiable or not. For example, a discontinuous function results if we take the within-host dynamics to be fully deterministic, such that even a slightly more virulent strain always wins. In this project however we make the mathematically most straightforward assumption that ρ is analytic. With this assumption, less virulent strains may win against more virulent ones with some small probability (ρ cannot be identically zero for negative arguments). This may be justified by stochastic effects within the host body.

With *n* strains α_i (*i*=1,...,*n*), the dynamics of the disease is given by

$$\frac{dS}{dt} = b(N)N - \sum_{i=1}^{n} \beta(\alpha_i) I_i S - dS$$

$$\frac{dI_i}{dt} = \beta(\alpha_i) I_i S + \sum_{j \neq i} \beta(\alpha_i)\rho(\alpha_i - \alpha_j) I_i I_j - \left[d + \alpha_i\right] I_i - \sum_{j \neq i} \beta(\alpha_j)\rho(\alpha_j - \alpha_i) I_i I_j$$

where I_i is the density of hosts infected with strain α_i and $N = S + \sum_{i=1}^{n} I_i$.

The aim of this project is to explore the adaptive dynamics of virulence. For numerical work, assume that the disease-free population grows logistically, i.e., $b(N) = a - cN$, and use $\rho(\Delta) = 1/\left[1 + v\exp(-k\Delta)\right]$ for the superinfection function with parameters such that $\rho(0)$ is small (equally and less virulent strains do not take the host over too often). For the transmission rate, start with $\beta(\alpha) = \gamma/\left[1 + \eta\exp(-\kappa\alpha)\right]$ (you can explore other functions later). A good starting set of parameter values is $a = 10$, $c = 0.03$, $d = 1$, $v = 10$, $k = 2$, $\gamma = 0.3$, $\eta = 20$ and $\kappa = 0.06$. When exploring the parameter space, make sure to study the effect of the shape (convexity) of $\beta(\alpha)$. Remember to study only viable strains (and hence populations at positive densities) only.