Adaptive Dynamics Course
É. Kisdi, Oslo 2008

The Evolution of Virulence – I

A population of disease-free hosts grows according to one of the two simple models

$$\frac{dN}{dt} = \left[b - d(N)\right]N \tag{1}$$

or

$$\frac{dN}{dt} = \left[b(N) - d\right]N \tag{2}$$

where N is population density, b is the per capita birth rate and d is the death rate. The two models differ in whether birth or death is density dependent: In model 1, the death rate is a monotonic increasing function of N and in model 2, the birth rate is a monotonic decreasing function of N.

Consider a disease that causes lifelong infection (no recovery). Infected individuals die at a rate of $d + \alpha$, 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

$$\begin{aligned}\frac{dS}{dt} &= bN - \beta SI - d(N)S \\ \frac{dI}{dt} &= \beta SI - \left[d(N) + \alpha\right]I\end{aligned} \tag{3}$$

in model 1 and

$$\begin{aligned}\frac{dS}{dt} &= b(N)N - \beta SI - dS \\ \frac{dI}{dt} &= \beta SI - (d + \alpha)I\end{aligned} \tag{4}$$

in model 2, 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, α. Note however that β cannot exceed the contact rate between individuals, hence the function $\beta(\alpha)$ must saturate for large values of α. Different strains of the pathogen differ in their virulence, α, and in the associated transmission rate, $\beta(\alpha)$. A strain is *viable* if it can spread in a disease-free population.

We further assume that once a host is infected with a certain strain, it cannot be infected by any other strain (no co-infection or superinfection). When a mutant strain with virulence α_{mut} appears in the population infected by strain α, but the density of hosts infected with the mutant strain is still low, the dynamics of mutant-infected hosts are given by the following two equations:

Model 1

$$\frac{dI_{mut}}{dt} = \beta(\alpha_{mut}) I_{mut} \hat{S} - \left[d(\hat{N}) + \alpha_{mut} \right] I_{mut} \tag{5}$$

Model 2

$$\frac{dI_{mut}}{dt} = \beta(\alpha_{mut}) I_{mut} \hat{S} - \left[d + \alpha_{mut} \right] I_{mut} \tag{6}$$

where $\hat{S}$ and $\hat{N}$ are the density of susceptibles and of all individuals, respectively, in the equilibrium population infected with the resident strain, as determined from equations 3 and 4 with virulence α and transmission rate $\beta(\alpha)$.

Start with model 1 and assume that the transmission rate is a simple hyperbolic function of virulence, $\beta(\alpha) = c\alpha/(a+\alpha)$ and the death rate is a linear function of density, $d(N) = A + BN$. Construct PIPs and show that two strains of the pathogen can coexist. Investigate the singular strategy and its stability properties. It is possible to prove also analytically that whenever $\beta(\alpha)$ is concave and the viable strains are in an interval (α_m, α_M) with $0 \le \alpha_m < \alpha_M$, there is a unique evolutionarily singular strain, which is both convergence stable and evolutionarily stable.

Next, find a different function $\beta(\alpha)$ such that the model exhibits evolutionary branching. Construct the isocline plot in an example with evolutionary branching to explore the coevolution of two strains.

In Model 2, show that different strains of the pathogen cannot coexist in equilibrium. Find the evolutionarily stable strain α^* and illustrate the results with a PIP.