EVOLUTIONARY INSIGHTS FROM SEMI-DISCRETE PLANT EPIDEMIC MODELS

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Many cropping systems are seasonal and involve abrupt changes in plant density due to planting and harvesting, as periodic impulses. *Semi-discrete* modelling, which is used in various fields of mathematical biology [1], is the way one usually deals with seasonal cropping in plant epidemiology [2]. We are interested in the evolutionary epidemiology of crop plant pathogens, as recently addressed by van den Berg et al [3].

We consider a classical SIR epidemic model in ODE during the n-th cropping seasons of length T:

$$\forall t \in [nT,\, (n+1)T)\,, \quad \begin{cases} \dfrac{\mathrm{d}S}{\mathrm{d}t} = -\beta S I\,, \\ \dfrac{\mathrm{d}I}{\mathrm{d}t} = \beta S I - \alpha I\,, \end{cases} \tag{1}$$

which is coupled to a difference equation system that depicts the inter-seasonal disease transmission:

$$\begin{cases} S((n+1)T^+) = p((n+1)T)S_0\,, \\ I((n+1)T^+) = (1 - p((n+1)T))S_0\,, \end{cases} \tag{2}$$

where the + superscript denotes the instant right after T and p is the probability of not being infected by the inoculum resulting from the current season epidemic at the very beginning of the next cropping season. This formulation and the explicit form of p arise from a mechanistic description of a preliminary primary infection phase (a rather generic feature in plant epidemiology), as originally suggested in [4].

Thus, the model combines the approaches of [4] and of [3] but differs from the latter in that the total host density is kept constant throughout the seasons, however the pathogen survives the intercrop season. This makes explicit the way density dependance acts at the beginning of each season, during the primary infection phase. As a result, evolution unlikely maximizes a quantity such $\mathcal{R}_0$ [5]. We will present first insights arising from evolutionary invasibility analysis, as illustrated by Pairwise Invasibility Plots (PIPs).

References

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