Emergence of Persistent Infection due to Heterogeneity

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Mathematically, epidemiological models have successfully captured the dynamics of infectious diseases. We explore the emergence of persistent infection in a population bounded in a closed region, where the disease progression of the individuals is given by the SIRS model.

Specifically, in this work we consider a cellular automata model of the SIRS cycle [1], with each cell representing an individual. The on-site dynamics is defined by the deterministic cycle, at the end of which individuals return to the susceptible state. An individual becomes infected on contact with another infected individual in the immediate neighbourhood.

We vary the heterogeneity of the initial composition of the population, and study the corresponding asymptotic behavior. In particular, we study the time and ensemble-averaged fraction of infecteds in the emergent system, denoted by $\langle I_t \rangle$.

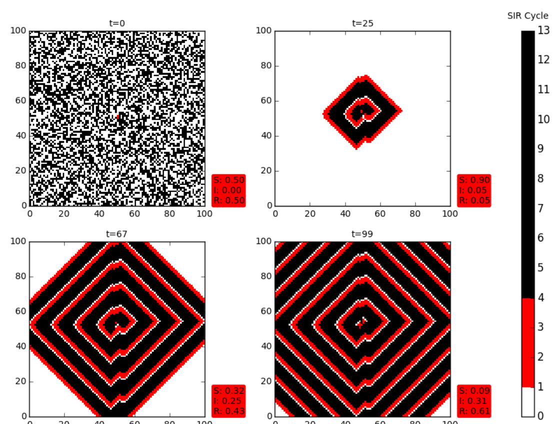

Figure 1: Snapshots of the infection spreading pattern in a heterogeneous population comprising initially of a random mixture of individuals ($S_0 = R_0 \sim 0.5$), with one infected individual at $t = 0$.

First we display the case of a single infected individual in a population in Fig. 3, from where it is clearly evident that there is a peak in persistence around $S_0 \sim 0.65 - 0.75$. So infection does not persist, i.e. $\langle I_t \rangle \to 0$, in the limits $S_0 \to 0$, which is the homogeneous limit of R_0, and $S_0 \to 1$, which is the homogeneous limit of S_0.

Further we vary the heterogeneity of the initial composition of the population, by increasing the initial fraction of infected individuals from 0 to 1. Again we find that there

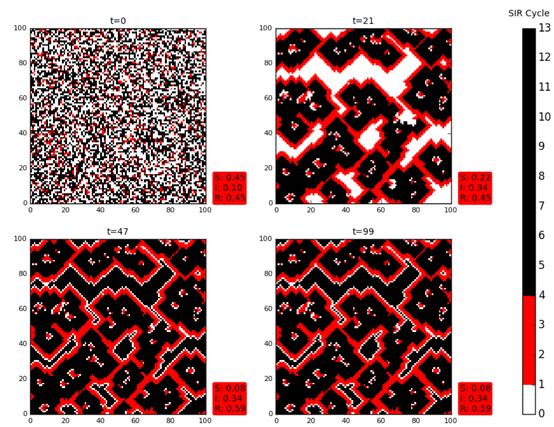

Figure 2: Snapshots of the infection spreading pattern in a heterogeneous population comprising initially of a random mixture of individuals, with $S_0 = R_0$ and $I_0 = 0.1$.

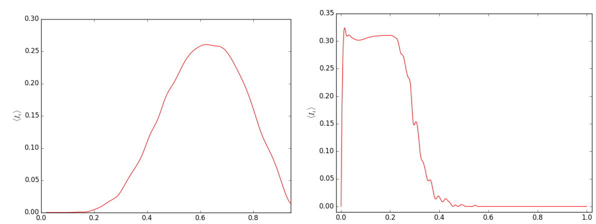

Figure 3: Variation of $\langle I_t \rangle$ (after transience) with respect to the initial fraction of susceptible S_0 (left) and infected individuals I_0 (right).

exists a *window of persistence*. This window starts with a sharp transition to a persistent state at I_0 close to zero, and extends to $I_0 \sim 0.4$, after which the infection ceases to persist. So, counter-intuitively, infection does not persist when the initial population has a large number of infected individuals.

In summary, we observe that infection persists for initial compositions that are more heterogeneous, and ceases to persist in the homogeneous limits. Moreover, when infection persists, the complex emergent spatial spreading patterns are recurrent.

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References

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