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Effects of Superspreaders in Spread of Epidemic

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1. Introduction

Spread of epidemic can be regarded as diffusion or random walk of disease on the fixed discrete elements. Severe Acute Respiratory Syndrome (SARS) which spread around the world during 2003 is no exception. It is now believed that the sharp increase and decrease in the number of SARS patients was caused by the existence of superspreaders. According to WHO, the patients are defined as superspreaders if they infect more than 10 people. However, the cause why such superspreaders appeared is not yet clear. Possible origins of appearance of them might be (i) the superspreaders have constitutional or hereditary strong infectiousness or (ii) the superspreaders have many social connections. In this presentation, we investigate the effects of such superspreaders on diffusion process, and show that the basic reproductive number plays a critical role.

2. Model and Results

In our study, we introduce two models as superspreaders in relation to two possible origins of appearance of them [1]. We investigate the percolation probability, the propagation speed, the epidemic curve and the distribution of secondary cases on the basis of a random walk model.

We consider N individuals randomly distributed on an $L \times L$ continuous space who take one of three possible state; susceptible (S), infected (I) and recovered (R). Superspreaders are mixed in normal individuals group, and they are characterized through the infection probability *w(r)*. We investigate two models for the superspreaders.

At first, we introduce *the strong infectiousness model* corresponding to the possible cause (i) whose infection probability *w(r)* is assumed as

$$w(r) = \begin{cases} w_0\left(1-\dfrac{r}{r_0}\right)^{\alpha} & 0 \le r \le r_0 \\ 0 & r_0 < r \end{cases}.$$

We set $\alpha = 2$ for the normal infection probability and $\alpha = 0$ for superspreaders (Fig. 1-i).

As an alternative model corresponding to the possible cause (ii), we introduce *the hub model* whose infection probability *w(r)* is assumed as

$$w(r) = \begin{cases} w_0\left(1-\dfrac{r}{r_h}\right)^{2} & 0 \le r \le r_h \\ 0 & r_h < r \end{cases}.$$

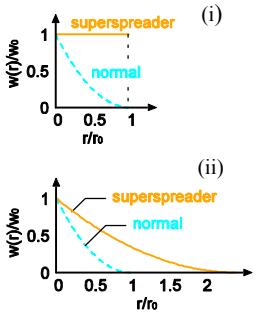

Fig. 1: Distance dependence of infection probability *w*(*r*); (i) the strong infectiousness model, (ii) the hub model.

We set $r_h = r_0$ for the normal infection probability and $r_h = \sqrt{6}\, r_0$ for superspreaders (Fig. 1-ii).

From Monte Carlo simulation, we obtained the following results.

(1) As the fraction of superspreaders is increased, the critical density of percolation transition shifts to the lower density. If all individuals are superspreaders for the strong infectiousness model, this epidemic threshold problem reduces to the question of overlap random disk percolation. We related the critical density of this case to the basic reproductive number R_0 (the mean of the number of newly infected individuals resulting from a single infected), and obtained the relation between the critical density and the basic reproductive number for the system consisting of nomals and superspreaders and for the hub model (Fig. 2).

(2) Both the size of infection and the velocity for the hub model are lager than those for the strong infectiousness model.

(3) The distributions of the number of links of the infection route network with superspreaders on both models show the feature of the distribution of secondary cases of SARS (large peak at zero and long tail) (Fig. 3).

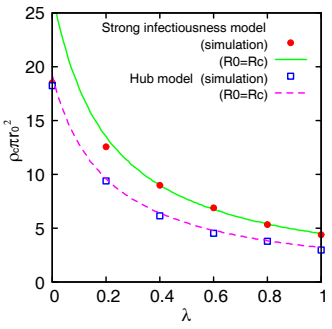

Fig. 2: Dependence of critical density on the fraction of superspreaders. The circles and the squares show the simulation results. The critical lines show R_0=R_c Above the critical curves, the disease percolates, and below the curves, the disease does not percolate.

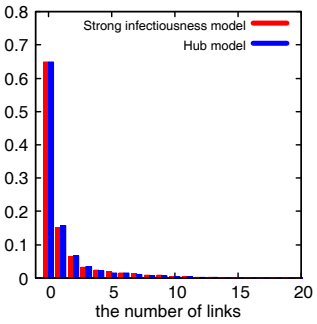

Fig. 3: Distribution of the number of links on the infection route network, both models for the superspreaders.

3. Conclusion

In this poster, we have studied the effects of superspreaders in spread of epidemic. We introduced two models of superspreaders assuming two kinds of distance dependence of infection probability. From Monte Carlo simulation, we obtained the percolation probability as functions of the density for the different fraction of superspreaders. The percolation transition appears at the critical density which decreases as the fraction of superspreaders is increased. We showed that the critical density coincides with the density at the critical basic reproductive number R_0=R_c. This result suggests that percolation transition can be understood by basic reproductive number R_0 for any mixing ratio in binary mixed percolation system.

Reference

[1] R. Fujie, T. Odagaki, Physica A 374 (2005) 843-852.