Continual Versus Occasional Spreading In Networks: Modeling Spreading Thresholds In Epidemic Processes

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1. INTRODUCTION

Epidemic processes are widely used as an abstraction for various real-world phenomena – human infections, computer viruses, rumors, information broadcasts, etc. [5, 1, 3]. Under the SIR model (susceptible-infected-removed/recovered) in finite networks, the *effective reproduction number*, $R()$, decreases as nodes become infected and removed. Hence, the spread process remains active for a while but eventually dies out (following $R < 1$, *"herd-immunity"*). Such threshold phenomena have been observed empirically. In these special days of COVID-19, estimations of the spread-induced Herd Immunity Threshold (HIT) are a key factor in directing strategic decisions concerning the fight against the pandemic.

Recent works showed that heterogeneity of spreading across nodes in the network decreases the traditionally predicted value of HIT dramatically [2, 4]. In this work we extend recent mathematical models and propose that the spreading intensity of a node and its heterogeneity are composed of two components. The first component reflects personal properties for each node and the second reflects occasional spreads across the network. Consequently, studying the spreading dynamics requires the analysis of a stochastic process consisting of two stochastic functions which drastically differ on their dynamics. One (continual) is biasly-modified throughout the process as infected nodes get immune and leave the game. The other (occasional) remains constant since it is identically distributed for the entire network.

By distinguishing between the different behavior of these components, we show that their relative weight affects dramatically the dynamics of the spread across the network. It reveals that different societies may engender significantly different HITs. Our approach can be used for addressing operational aspects and examine the effectiveness of preventive-measures used to mitigate the spread (by suppressing the stochastic functions, e.g., lockdowns and vaccines). We find that while some lockdowns decrease the HIT, others increase the HIT and may be counter-productive in the long-run.

The paper is organized as follows. In Section 2 we present our stochastic spreading model. We then (Section 3) move to analyze the disease dynamics and derive the Herd Immunity Threshold of a given network. In Section 4, we discuss the ongoing COVID-19 pandemic. Further work and concluding remarks are given in Section 5.

It should be noted that the model can apply to a variety of viruses (malware) spreading in networks. For the sake of clarity and actuality our presentation will focus on pandemics. We will refer to a network as *population/society* and nodes as *individuals*.

2. THE MODEL

Our analysis begins with a certain number of infected individuals (where all others are assumed to be initially susceptible (**S**)). As a result of an *infection*, susceptible individuals become infectious (**I**). After an infectious period, individuals are removed (recover or die) (**R**) and "leave the game".

We measure the spread of the disease as a function of the number of individuals who contracted it. Namely, the event whereby the nth individual gets infected is called the nth step of the disease. This indexing method will be useful in deriving the Herd Immunity Threshold.

Spreading Functions

We extend the model of [4] and classify spreading into two inherently different types: (1) *Personal-Trait-Spreading* and (2) *Event-Based-Spreading*. The first (Personal-Trait) stems from the personal traits of an individual, while the second (Event-Based) relates to social events in which every individual may participate, regardless of their personal traits[1].

The *susceptibility* of v at step i, which is the likelihood of v to be infected, is:

$$S^i(v) = p \cdot S_p(v) + (1-p) \cdot S_e^i(v), \quad (1)$$

and the *infectiousness* of v at step i, which is the likelihood of v to infect others is

$$I^i(v) = p \cdot I_p(v) + (1-p) \cdot I_e^i(v). \quad (2)$$

Where:

- S_p and I_p are the *personal-trait* susceptibility and infectiousness parameters of u, respectively. The values of $S_p(v)$ and $I_p(v)$ reflect personal traits of v, are drawn once for v (spread beginning) and accompany v throughout the entire progress of the disease, as in [4].

[1]As examples of these two types, consider a supermarket cashier compared to an academic researcher. In any given day, the cashier interacts with tens or hundreds of people, and therefore has high personal-trait spreading degree. In contrast, the researcher stays in the laboratory or interacts with a small research group. However, both may participate in a social-gathering event such as a concert, a wedding, or "just" a family birthday party. During such an event, both may engage in a similar amount of social interaction (which may be quite large), and therefore they both have the same event-based spreading degree.

- In addition, and beyond the model of [4], we assign *event-based* susceptibility and infectiousness parameters, S_e and I_e. These values reflect occasional event-based spreading and are subject to change throughout the progress of the disease since they probabilistically redrawn for each individual at every step of the pandemic. At step i we assign to v $S_e^i(v)$ and $I_e^i(v)$, the realizations for step i. These values are drawn from probability distributions that are common for the entire population, denoted Λ_S and Λ_I.

- p (and $q := (1-p)$) represents the *relative weight* of each spreading type in the society ($0 \leq p \leq 1$). Societies characterized by a low (high) level of social gatherings will have a high (low) value of p.[2]

The symmetry in structure between $S(v)$ and $I(v)$ stems from the assumption that infectiousness and susceptibility levels are both proportional to the interpersonal interactions and the biological characteristics of v.

We follow [4] and define $\varphi(\sigma)$, the *expected conditional infectiousness*. In our case, it is logical to parameterize $\varphi(\sigma)$ only by the personal-trait susceptibility and infectiousness:

$$\varphi(\sigma) := \mathbb{E}\left[I_p(v) | S_p(v) = \sigma\right]. \tag{3}$$

As was discussed, the heterogeneity of the spreading values of the population will play a major role in our analysis Hence, we will measure[3]:

$$\rho(\sigma, n) := \Pr\left[S_p(v) = \sigma \middle| v \in H_n\right] \tag{4}$$

where H_n is the healthy population at step n.

Basic and Effective Reproduction Rate

The *basic reproduction number*, R_0, is a measure of how transferable a disease is. It is defined as the expected number of secondary cases produced by a single (typical) infection in a completely susceptible population (of size N_0).

As the spread continues, varying proportions of the population are recovered/removed at any given time. Hence, we will measure the *effective reproduction number*, $R(n)$, which is defined as the expected number of infections directly generated by the nth infected individual (e.g., $R_0 = R(0)$).

Note that, intuitively, $R(n)$ is continuously decreasing as the susceptible population decreases when individuals become infected and then removed.

For any individual v, the probability that v will be infected at step n, assuming that v was susceptible at step $n-1$ is:

$$\frac{S^{n-1}(v)}{\sum_{u \in H_{n-1}} S^{n-1}(u)}. \tag{5}$$

Therefore,

$$R(n) = \mathbb{E}[I^n(v) \cdot \sum_{u \neq v} S^n(u)] =$$

$$= \sum_{v \in H_{n-1}} \frac{S^{n-1}(v)}{\sum_{u \in H_{n-1}} S^{n-1}(u)} I^n(v) \sum_{u \neq v \in H_{n-1}} S^n(u)$$

where the expectation is taken over all possible scenarios of infections. This can be approximated by:

$$R(n) \approx \sum_{v \in H_{n-1}} S^{n-1}(v) \cdot I^n(v). \tag{6}$$

Using Eq. (1), (2) and (6) we have:

$$R(n) \approx N(n) \int \rho(\sigma, n) \cdot (p \cdot \sigma + q \cdot \lambda_S)\,(p \cdot \varphi(\sigma) + q \cdot \lambda_I)\, d\sigma \tag{7}$$

where $N(n)$ is the size of the susceptible population at step n and λ_S, λ_I are the means of Λ_S, Λ_I, respectively.

3. HERD IMMUNITY THRESHOLD

We analyze the changes in the composition of the population throughout the spread of the disease, and the decrease of $R(n)$ as the fraction of the population that contracted the disease increases. This is established in Theorem 1 and can be used to derive the Herd Immunity Threshold

Theorem 1. For any $\delta > 0$ when

$$1 - \int \rho(\sigma) \cdot \exp\left(-\delta \cdot (p \cdot \sigma + q \cdot \lambda_S)\right) d\sigma \tag{8}$$

fraction of the population is infected, the effective reproduction number, $R(n)$, will be lower than the basic reproduction number, R_0, by a factor of

$$\frac{\int \rho(\sigma, 0) \cdot \exp\left(-\delta \cdot (p \cdot \sigma + q \cdot \lambda_S)\right) \cdot r(\sigma) d\sigma}{\int \rho(\sigma, 0) \cdot r(\sigma) d\sigma} \tag{9}$$

where

$$r(\sigma) = (p \cdot \sigma + q \cdot \lambda_S) \cdot (p \cdot \varphi(\sigma) + q \cdot \lambda_I).$$

The threshold for herd immunity (HIT) is when the value of the effective reproduction number is $R(n) \leq 1$ (and hence the number of infection cases decreases).

Outline of Proof 1. We begin by deriving the probability that an individual v is susceptible at step n. We take log over Eq. (5), and counting over the steps $1, \ldots, n$. Full calculations appear in [6] (archive). We then have:

$$\log(\Pr[v \text{ is susceptible at step } n]) \approx -\sum_{i=1}^{n-1} \frac{S^i(v)}{N(i) \cdot \mathbb{E}_{u \sim H_i}[S^i(u)]}. \tag{10}$$

Since our calculation is done by taking an expectation over all possible scenarios of infections, by Eq. (1) and Eq. (2):

$$\Pr[v \text{ is susceptible at step } n] \approx \exp\left(-\beta(n) \cdot (p \cdot S_p(v) + q \cdot \lambda_S)\right) \tag{11}$$

where

$$\beta(n) = \sum_{i=0}^{n-1} \frac{1}{N(i) \cdot \mathbb{E}_{u \sim H_i}[S^i(u)]}. \tag{12}$$

According to Eq. (11), for any $s \in \mathrm{Supp}(S_p)$,

$$\rho(s, n) \approx \frac{\rho(s, 0) \cdot \exp\left(-\beta(n) \cdot (p \cdot s + q \cdot \lambda_S)\right)}{\int \rho(\sigma, 0) \cdot \exp\left(-\beta(n) \cdot (p \cdot \sigma + q \cdot \lambda_S)\right) d\sigma}, \tag{13}$$

and

$$N(n) \approx N_0 \cdot \int \rho(\sigma, 0) \cdot \exp\left(-\beta(n) \cdot (p \cdot \sigma + q \cdot \lambda_S)\right) d\sigma. \tag{14}$$

[2]Note that the special case where $p = 1$ gives exactly the model of [4] (only one spreading type).

[3]For continuous σ , Eq. (4) should be considered as a density function.

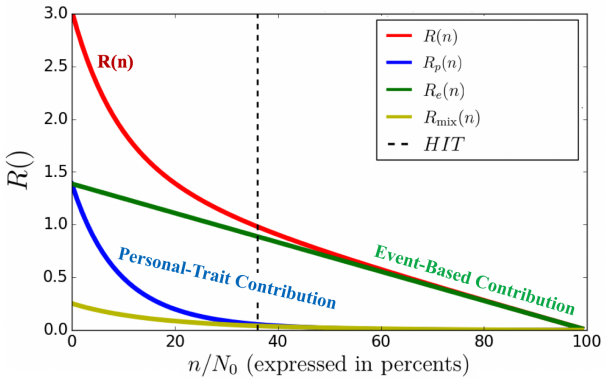

Figure 1: The over-time reduction in $R(n)$, and its contributing factors as a function of n assuming $p = 0.5$, $k = 0.1$, and $R_0 = 3$.

Following Eq. (7), Eq. (13) and Eq. (14),

$$\frac{R(n)}{R_0} = \frac{\int \rho(\sigma,0) \cdot \exp\left(-\beta(n) \cdot p \cdot \sigma + q \cdot \lambda_S\right) \cdot r(\sigma) d\sigma}{\int \rho(\sigma,0) \cdot r(\sigma) d\sigma}.$$

This concludes the proof as the value of Eq. (8) is $1 - \frac{N(n)}{N_0}$.

4. COVID-19: DISCUSSION

To demonstrate our results we use a Gamma distribution with shape and scale parameters k and θ, respectively. The Gamma distribution was previously attributed to the infectiousness of COVID-2 and COVID-19 [4]. We substitute the estimates for COVID-19: $R_0 \approx 3$ and $k \approx 0.1$.

Figure 1 depicts the decay of the factors contributing to $R(n)$, classified by their spreading types. In red: $R(n)$; In blue: Personal-trait pure contribution to $R(n)$; In green: Event-based pure contribution to $R(n)$; In yellow: Mutual contribution to $R(n)$. As can be seen, the contribution of the personal-trait spreading drops sharply at early stages of the disease. On the other hand, the contribution of the event-based spreading is affected very little at early stages.

This is because super-spreaders (individuals with high values of S_p and I_p) are likely to get infected early in the pandemic, and their resultant immunity then decreases the level of personal-trait spreading. In contrast, any reduction of the event-based spreading level is merely proportional to the decrease in susceptible members of the population, which is linear in n, as it is identically distributed for all individuals.

In addition, we use our model to inspect the sensitivity of the HIT to the social characteristics of a society. Per Eq. (9), we find that the HIT value is very sensitive to p, the relative weight of the spreading types, and thus different societies may engender significantly different HITs. In addition, and in accordance with previously reported results [2, 4], the HIT is influenced by the coefficient of variation (Cv) of the personal-trait spreading distribution, i.e. the level of personal-trait spreading heterogeneity, and by the correlation between the infectiousness and susceptibility.

This is demonstrated in Figure 2 which plots the HIT as a function of both the relative weight p and the shape of the personal spreading distribution (heterogeneity level). Note that for a fully personal-trait society the HIT prediction is as low as 5% (coinciding with [4]) and for a fully event-based society it as high as 67%, coinciding with the allegedly "axiomatic" cutpoint of the traditional homogeneous models prediction.

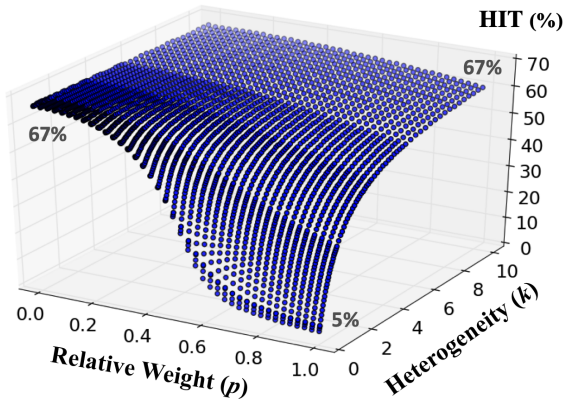

Figure 2: The HIT as a function of p (relative weight) and k (distribution shape) where $R_0 = 3$.

5. CONCLUSIONS AND FURTHER WORK

We studied the effects of the spreading function composition on the spread of a disease in a given network, and on the Herd Immunity Threshold. Having developed the model and gained an understanding of the epidemic behavior in a no-intervention environment, we can address operational aspects and examine intervention measures, which suppress the stochastic functions, including lockdowns and vaccination strategies. In further research we study the effects of lockdowns on the HIT and the disease dynamics. Our model reveals that the effect of lockdowns is very sensitive to their focus: while event-based-targeted lockdowns act positively by reducing HIT, personal-trait-targeted lockdowns increase HIT and may be counter-productive. Furthermore, population heterogeneity significantly affects vaccination strategies efficacy.

6. REFERENCES

[1] S. Banerjee, A. Chatterjee, and S. Shakkottai. Epidemic thresholds with external agents. In *IEEE INFOCOM 2014*, pages 2202–2210. IEEE, 2014.

[2] T. Britton, F. Ball, and P. Trapman. A mathematical model reveals the influence of population heterogeneity on herd immunity to sars-cov-2. *Science*, 369, 2020.

[3] Y. Lu, M. S. Squillante, and C. Wah Wu. Epidemic-like stochastic processes with time-varying behavior: Structural properties and asymptotic limits. *SIGMETRICS Perform. Eval. Rev.*, 45(3), 2018.

[4] Y. Oz, I. Rubinstein, and M. Safra. Heterogeneity and superspreading effect on herd immunity. *Journal of Statistical Mechanics: Theory and Experiment*, 2021(3):033405, 2021.

[5] R. Pastor-Satorras, C. Castellano, P. Van Mieghem, and A. Vespignani. Epidemic processes in complex networks. *Reviews of modern physics*, 87(3):925, 2015.

[6] J. Tavori and H. Levy. Super-spreaders out, super-spreading in: The effects of infectiousness heterogeneity and lockdowns on herd immunity. *arXiv preprint arXiv:2101.09188*, 2021.