MODELING THE HETEROGENEOUS DISEASE-BEHAVIOR-INFORMATION DYNAMICS DURING EPIDEMICS

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ABSTRACT

The transmission of infectious diseases depends on the social networks among people and the protections that people have taken before being exposed to the disease. Mass media is playing a key role in making the public aware of the disease and its transmissibility and severity. Motivated by the importance of heterogeneous risk perception in the population to response to infectious disease outbreaks (particularly the ongoing COVID-19 pandemic), we propose a heterogeneous three-layer network model, namely the Susceptible-Exposed-Infectious-Recovered-Uncertain-Aware-Protected (SEIR-UAP) model, where people’s vulnerability to the disease is influenced by the processes of awareness information diffusion, preventive behavior change and infectious disease transmission. Analytical and simulation results are presented to validate the model. We found that (a) the awareness of the disease plays the central role in preventing disease outbreak; (b) we need a reasonable ratio of “over-reacting” nodes to effectively control the disease outbreak; (c) diseases with a longer incubation period and a higher recovery rate are easier to control because the processes of information diffusion and behavior change can help people prepare for the upcoming exposure to the disease; (d) it is more difficult to control the disease with asymptomatic cases. The results provide evidence that mass media should not play down the transmissibility and severity of diseases, so that the public can become aware of the disease as soon as possible.

1 introduction

Public’s behavioral responses to an infectious disease could greatly affect the transmission patterns of the disease, and it has been proved that information about transmissibility and severity of the disease conveyed through the mass media plays the central role in raising the awareness and influencing individuals’ decision making on whether or not to take self-protectors [1][2][3][4][5][6].

Many studies [7][8][9][10][11][12] have used mathematical models to investigate how the awareness of diseases affects the outbreak of diseases. Most approaches [13][14][15] explored this problem by modifying the parameters in standard epidemic models. Funk et al. first incorporated the impact of awareness into classic epidemic models and found that the spread of awareness could significantly reduce the epidemic size [16]. Wu et al. modeled the effect of three forms of awareness: global awareness, local awareness, and contact awareness [17]. The model studied by Chen focuses on the quality of information in an agent-based model [18].
Multiplex (also named as multilayer) networks \cite{19} have been adopted to model the dynamic interactions between the spread of information and infection. In a multiplex network, we assume that there exist the spreading processes of both information and infection, represented by multiple layers of the network. For example, the \textit{UAU-SIS} (susceptible-infected-susceptible unaware-aware-unaware) model, proposed by Granell et al., is able to capture the critical point of disease outbreak determined by the topological structure of the virtual contact network formed by the information propagation\cite{20}. It can be extended to many model variants, such as the multiple-information model, which incorporates more than one type of information \cite{21}, the local awareness controlled contagion spreading model \cite{22}, in which the awareness transition is further influenced by the extent of the awareness of all neighbors, and an \textit{SIR-UA} (susceptible-infected-recovered unaware-aware) model, which considers all possible schemes of dynamics\cite{23}. In addition to information and disease transmissions, some studies also considered the transmission of preventive behavior in an simulation study \cite{24}.

Recently, a number of studies examined the effect of mass media in infectious disease epidemics \cite{25,26,27,28,29,30,31,32}. Liu et al. proposed a mechanism to illustrate the media effect by incorporating the reported number of infectious and hospitalized individuals into classic epidemic models\cite{27}. Wang and Xiao used a threshold model where the media can exhibit its effect only when the number of reports reaches a certain value\cite{28}. Dubey et al. discussed the optimal amount of information which can not only suppress the spread of disease but also avoid "media-fatigue"\cite{29,30}. Song and Xiao further considered the delay of media effects on human responses\cite{31,32}.

However, few studies have devoted to consider the heterogeneity of public responses to media reporting during the course of epidemic. People’s willingness to take self-protection behaviors and to share disease-related information can be influenced not only by the transmissibility and severity of the disease as reported by the mass media, but also by the personal risk perception, which is the subjective judgement about the transmissibility and severity of the disease\cite{33,34,35}. Similar to smart nodes in information diffusion Ruan paper ref \cite{36}, those individuals who are more fearful of being infected are found to be more actively engaging in self-protections and information sharing. We label these people as "over-reacting" as compared to those "under-reacting" people, who have a lower risk perception. While others who are less willing to do such things are "under-reacting". This is of particular relevance to the ongoing novel coronavirus pandemic (COVID-19), in which a clear disparity in risk perception caused diverse reaction to control measures \cite{27,38,39,40}. The lessons we learned from COVID-19 outbreak in many countries is that, making the public aware of the disease is not sufficient, it is very important to understand how to increase the ratio of "over-reacting" people in the population via media reporting.

Motivated by the importance of heterogeneous risk perception in the population to response to infectious disease outbreaks (particularly the ongoing COVID-19 pandemic), we propose a heterogeneous three-layer network model, namely the \textbf{Susceptible-Exposed-Infectious-Recovered Unaware-Aware-Protected (SEIR-UAP) model}, which consists of the process of awareness information diffusion, preventive behavior change and infectious disease transmission. The aim of our study is to describe how different types of nodes ("over-reacting" versus "under-reacting") shape the prevalence of preventive behaviors and affect the natural course of diseases. The contribution of this study is threefold: First, this is the first study that considers both the risk perception and the awareness of diseases. Second, we consider the heterogeneity in the response to the awareness information of the disease. This heterogeneity in the response comes from the heterogeneity in the distribution of the risk perception among nodes. Third, we study analytically and numerically the effect of such heterogeneity on the epidemic process.

The rest of the paper is organized as follows. First, we describe the model details in section 2. Second, we adopt the mean field method to formulate the problem mathematically in section 3. Third, we perform extensive experiments with Poisson degree distribution-based random networks, and explore the effects on different parameters on the epidemic size in section 4. Last we conclude the paper with discussions of future work in section 5.

\section{model}

We propose a novel three-layer network model, namely the \textbf{SEIR-UAP} model, to incorporate the processes of information diffusion, behavior change and disease transmission. In the SEIR-UAP model, nodes may become aware of the risk for getting infected, and then change their behaviors by adopting self-protections, which will further affect the disease transmission process. We first introduce the details as shown in Fig. 1.

The bottom layer, \textit{Information Diffusion Layer}, represents the information diffusion process. The source of the information is the mass media. Nodes are divided into three classes on this layer: Ignorants, Spreaders and Stiflers, denoted by \textit{IG}, \textit{SP} and \textit{ST} respectively. Ignorants are those who are \textit{unaware} of the information about the disease. Ignorants can become Spreaders (with a probability \(\alpha\)) or Stiflers (with a probability \(1 - \alpha\)) if at least one neighbor is a Spreader. Spreaders and Stiflers are those who have been exposed to the information, in other words, they are \textit{aware}
of the disease. Their states do not change. Once become a Spreader, the node will keep spreading the information to all its neighbors in each time period. Stiflers, on the other hand, do not further spread the information.

We represent the scheme mentioned above in panel(b) of Fig. 2. Each piece of information has an alarming level $y$, which represents the transmissibility and severity of the disease as reported by the mass media. Each node in the network has a personal risk perception, which is a constant parameter $x_i$ for node $i$. Comparing the values of $y$ and $x_i$, we classify nodes into two sets: $\{i \mid y \geq x_i\}$, “over-reacting” and $\{i \mid y < x_i\}$, “under-reacting”. “over-reacting” nodes are set to have a higher probability to spread the information, as follows.

$$\alpha = \begin{cases} \alpha_o & y \geq x_i \\ \alpha_u & y < x_i \end{cases}$$

where $0 \leq \alpha_u < \alpha_o \leq 1$.

The middle layer, Behavior Change Layer, represents the behavior change process. Nodes on this layers have two states: with self-protection ($Y$) and without self-protection ($N$). Ignorants on the Information Diffusion Layer are always with the $N$ state on the Behavior Change Layer, because they are not aware of the risk. Spreaders and Stiflers are with a tendency $p$ to change the behavior by adopting self-protections, such as wearing a mask and washing hands with sanitizer in the COVID-19 and other influenza-like context. “Over-reacting” nodes have a higher behavior change tendency $p$ than “under-reacting” nodes, as follows

$$p = \begin{cases} p_o & y \geq x_i \\ p_u & y < x_i \end{cases}$$

where $0 \leq p_u < p_o \leq 1$.

The probability of a node to change behavior is not only dependent on $p$, but also on the behaviors of the neighbors of the node. Let $k_i$ denote the degree of node $i$, $\vartheta$ denote the proportion of neighbors who have already changed behavior, and $\varsigma$ denote the neighbors’ status as follows

$$\varsigma = \begin{cases} 1 & k_i \vartheta > k_i(1-\vartheta) + 1 \\ 0 & k_i \vartheta \leq k_i(1-\vartheta) + 1 \end{cases}$$

The overall behavior change probability is as follows

$$P = 1 - (1 - p)(1 - \varsigma)$$

Thus, the model determines if a node changes the behavior in two steps. First, we use the tendency $p$ to determine if the node changes the behavior independent from the social influence. If the node doesn’t change the behavior, the model further check the node’s neighbors’ states. If more than half of the neighbors plus the node have changed their behavior, the node will be influenced to change the behavior as well. We represent the scheme mentioned above in panel(c) of Fig. 2.
Figure 2: Illustration of the transitions between states on (a) the Disease Transmission Layer, (b) the Information Diffusion Layer and (c) the Behavior Change Layer. On the Information Diffusion Layer, the Ignorant (IG) nodes are unaware of the disease and the Spreader (SP) and Stifler (SF) nodes have become aware of the disease.

The top layer, Disease Transmission Layer, is modeled by the generalized Susceptible-Exposed-Infectious-Recovered (SEIR) model. The susceptible, exposed, infectious and recovered individuals are denoted by $S$, $E$, $I$ and $R$ respectively. A susceptible individual can be transmitted by either an infectious individual with an infection rate $\beta I$ or an exposed individual with an infection rate $\beta E$. Here we allow the transmission from exposed individuals to model the widely reported asymptomatic infected cases in the COVID-19 pandemic. If we set $\beta E = 0$, the model becomes a classic SEIR model.

The infection rate $\beta$ is a constant value that is only related to the disease. However, the actual infection rate among people could be various given different self-protection behaviors taken by people. Consider an edge connecting two nodes, we use $n \in \{0, 1, 2\}$ to denote the number of nodes with self-protections. Then, the actual transition rate is $\eta n \beta$, where $\eta$ is efficiency of self-protection behaviors. Note that $\eta \in [0, 1]$. $\eta$ is 0 if the self-protection is fully preventive, and 1 if the self-protection is entirely useless. An exposed individual has a transition rate $\sigma$ to become infectious. So the incubation period is $1/\sigma$. An infectious individual has a transition rate $\gamma$ to become recovered and immune to the same disease. All possible state transitions are shown in panel(a) of Fig. 2.

3 Theoretical analysis

We adopt the mean field approximation approach to analyze the dynamics of the SEIR-UAP model. For simplicity, we consider a three-layer network with the same topological structure in all three layers in this section. Consider a random network of $N$ nodes with degree distribution function $P(k)$, let $\rho_{IG}(t)$, $\rho_{SP}(t)$, $\rho_{SF}(t)$ denote the ratio of nodes in Ignorant, Spreader, and Stifler states, respectively. Thus, $\rho_{IG}(t) + \rho_{SP}(t) + \rho_{SF}(t) = 1$. The transition rate for an IG node being informed by neighbors is

$$h_t = 1 - \sum_{k=0}^{\infty} P(k)q_{k,0},$$

where $q_{k,0} = (1 - \rho_{SP}(t))^k$, $k$ is the degree of the node.

Assuming a standard normal distribution of the risk perception $x_i$ in the network, then we can calculate the ratio of “over-reacting” nodes by evaluating the corresponding accumulated distribution function at the alarming level $y$. 


denoted by $a$. Let $\rho_Y(t)$ denote the ratio of nodes who have already taken self-protection behaviors (i.e. changed the behavior) at time $t$, then the value of $\rho_Y(t + 1)$ is

$$\rho_Y(t + 1) = (\rho_{SP}(t + 1) + \rho_{SF}(t + 1))(ap_o + (1 - a)p_u) + (a(1 - p_o) + (1 - a)(1 - p_u)) \sum_{k=0}^{\infty} P(k)W_k$$

(6)

where $W_k = \sum_{b=\lceil \frac{k-2}{2} \rceil}^{k} \binom{k}{b}(\rho_Y(t))^b(1 - \rho_Y(t))^{k-b}$ denotes the probability for a node with degree $k$ choosing to protect itself because of the influence from neighbors. Denote $z$ as the number of neighbors who have already changed the behavior, only when $z > \frac{k-1}{2}$, can a node change its behavior because of the influence from neighbors, thus the minimum integer value of $z$ satisfying this condition is $\lceil \frac{k-2}{2} \rceil$. Therefore,

$$\rho_N(t + 1) = 1 - \rho_Y(t + 1),$$

(7)

which is the ratio of nodes without health protections at time $t + 1$.

The ratio of nodes in one of four health status at time $t$ is denoted by $\rho_S(t)$, $\rho_E(t)$, $\rho_I(t)$, $\rho_R(t)$ and $\rho_S(t) + \rho_E(t) + \rho_I(t) + \rho_R(t) = 1$. For a single edge between a susceptible node and an infected one, the actual infection rate (considering the behavioral changes) $l(\beta)$ is

$$l(\beta) = \rho_Y(t)^2 \eta^2 \beta + (1 - \rho_Y(t))^2 \beta + [1 - (\rho_Y(t))^2] - (1 - \rho_Y(t))^2 \eta \beta = \beta [1 + (\eta - 1)\rho_Y(t)]^2,$$

(8)

For a node of state $I$, the infection propagates to its $S$ neighbors with probability $l_I = l(\beta_I)$. For an exposed node who is asymptomatically infected, the corresponding probability $l_E = l(\beta_E)$. Then, for a node of degree $k$ with $b_E$ neighbors in health states $E$ and $b_I$ neighbors in state $I$, the probability of being infected is $t_{b_E,b_I} = 1 - (1 - l_E)^{b_E}(1 - l_I)^{b_I}$. Thus the transition probability for a susceptible individual being infected at time $t$ is $c_t$,

$$c_t = \sum_{k=0}^{\infty} P(k) \sum_{b_E=0}^{k} \sum_{b_I=0}^{k-b_E} r_{k,b_E,b_I} t_{b_E,b_I},$$

(9)

where

$$r_{k,b_E,b_I} = \binom{k}{b_E} \binom{k-b_E}{b_I} [(\rho_E(t))^{b_E}(\rho_I(t))^{b_I} \left(1 - \rho_E(t) - \rho_I(t)\right)^{k-b_E-b_I}].$$

(10)

Then, we can obtain the following differential equations to illustrate the dynamics on the Disease Transmission Layer and the Information Diffusion Layer in Fig. 1

$$\begin{align*}
\frac{d\rho_S(t)}{dt} &= -c_t \rho_S(t) \\
\frac{d\rho_E(t)}{dt} &= -\sigma \rho_E(t) + c_t \rho_S(t) \\
\frac{d\rho_I(t)}{dt} &= -\gamma \rho_I(t) + \sigma \rho_E(t) \\
\frac{d\rho_R(t)}{dt} &= \gamma \rho_I(t) \\
\frac{d\rho_{IG}(t)}{dt} &= -h_t \rho_{IG}(t) \\
\frac{d\rho_{SP}(t)}{dt} &= h_t \rho_{IG}(t)(a\alpha_o + (1 - a)\alpha_u) \\
\frac{d\rho_{SF}(t)}{dt} &= h_t \rho_{IG}(t)(a(1 - \alpha_o) + (1 - a)(1 - \alpha_u))
\end{align*}$$

(11)

4 Results

To validate the numerical results obtained by equation[11] we performed extensive Monte Carlo simulations. For the sake of simplicity, we assume that $\alpha_o = p_o = 0.99$ and $\alpha_u = p_u = 0.01$, implying an extremely high probability to
inform others about the disease and to change behaviors for "over-reacting" nodes, and a much lower probability for "under-reacting" nodes.

We present the comparison between the simulation and numerical results in Fig. 3 for a three-layer network with 2,000 nodes with a Poisson degree distribution and the mean degree of 15. As illustrated in Fig. 3 we observe a high $R^2$ of 0.9781 for the top panel (ratio of susceptible nodes) and 0.9783 for the bottom one (ratio of Spreaders), which validates that the differential equations in (11) are able to effectively capture the dynamics of the model. Moreover, We find that the ratio of "over-reacting" nodes $a$ has an almost linear effect on the final ratio of Spreaders, while an S-shape effect on the final ratio of susceptible nodes $\rho_s(\infty)$.

In the following, we examine the degree to which the "over-reacting" nodes influence disease dynamics under different situations. It’s extremely hard to obtain the analytical solutions for equation (11) thus, full phase diagrams are used to illustrate the results instead. We adopt the same three-layer network in the above Monte Carlo simulations.

First, we focus on diseases with $\beta_E = 0$ (classic SEIR model without asymptomatic cases). The final ratio of nodes $\rho_s(\infty)$ is shown in Fig. 4 with respect to the infection rate $\beta_I$ and the ratio of "over-reacting" nodes $a$ for different values of $\sigma$ and $\gamma$. Similar to Fig. 3, the ratio of "over-reacting" nodes has an S-shape effect on the final ratio of susceptible nodes regardless of the value of parameters on the Disease Transmission Layer, but the corresponding range of $a$ for each stage is different, as shown in Fig. 4. We find that $\rho_s(\infty)$ generally has three phases: when $a$ is small, $\rho_s(\infty)$ is close to 0; when $a$ is large, $\rho_s(\infty)$ is close to 1. There exists a threshold that triggers the rapid increase of $\rho_s(\infty)$. The exact value of the threshold cannot be analytically obtained. This indicates that by increasing the ratio of "over-reacting" nodes through highlighting the transmissibility and severity of the disease in media reporting, we can largely prevent the disease outbreak. More importantly, as long as the ratio of "over-reacting" nodes reaches the threshold, we do not need to increase the media reporting too much as the benefits of doing so is not slim. This finding is interesting because it shows that, disease outbreak is preventable if the mass media is playing an effective role of the whistle-blower.

More specifically, when the value of $\beta_I$ is extremely small, all possible values of $a$ yield to the fully prevented scenario (red). As $\beta_I$ increases, there is a rapid increase in the range of $a$ yielding to the full outbreak scenario (blue). If $\beta_I$ keeps increasing, the range of $a$ yielding to full outbreak scenario gradually increases.

Additionally, we observe an increase in the final ratio of susceptible nodes with a longer incubation period (smaller $\sigma$) and a higher recovery rate $\gamma$. It might be due to the fact that longer incubation period can provide more time for the awareness to be transmitted among people and a larger $\gamma$ means a lower level of severity of the disease, which means a smaller ratio of "over-reacting" nodes are needed to prevent full outbreak.

Second, we consider the asymptomatic cases. The asymptomatic infection rate $\beta_E = \mu \beta_I$, where $\mu \in (0, 1)$. We demonstrate the final ratio of susceptible nodes $\rho_s(\infty)$ in Fig. 5 where $\mu \in \{0.2, 0.4, 0.8\}$. The values of other parameters are the same as that in panel (b) of Fig. 4, where asymptomatic infection is not considered. We find that when there is asymptomatic infection, the range of $a$ yielding to the full outbreak scenario is larger, and the...
improvement becomes more significant with a larger value of $\mu$. These results indicate that asymptomatic cases make it harder for the media reporting to exhibit its effect, in some cases it is even impossible to control the disease by only increasing the value of $a$ (panel (c) of Fig. 5). Furthermore, we explore the effects of “over-reacting” nodes for various parameter settings on the Disease Transmission Layer. As shown in Fig. 6, we obtain a similar pattern to that in Fig. 4: a longer incubation period and a higher recovery rate lead to a larger ratio of susceptible (not-infected) nodes $\rho_S(\infty)$ in the end.

Third, we further clarify the effect of “over-reacting” nodes on disease control for different values of $\alpha$ and $p$ in Fig. 7. For the sake of simplification, we assume $\alpha_u = \frac{1}{2} \alpha_o$ and $p_u = \frac{1}{2} p_o$. It is obvious that fewer nodes will be infected as $\alpha$ and $p$ increase. Interestingly, we observe that when the value of $a$ increases, the disease is easier to be controlled (the red region in the figure). There is little space transitioning between controlled (red) and full outbreak (blue) scenarios, indicating that the epidemic can either be well contained, or will likely to infect the majority of people in the population.

Finally, we examine final ratio of susceptible nodes $\rho_S(\infty)$ in two scenarios: with and without the social influence on the behavioral change. Here, the scenario without social influence is modeled by setting $\sigma = 0$ regardless of the behaviors of the node’s neighbors. We consider two diseases with different epidemiological parameter settings in Fig. 8. It is obvious that, with the social influence on the Behavior Change Layer, we can achieve the fully controlled result ($\rho_S(\infty) \to 1$) with a lower value of $a$ for both diseases, indicating that we can effectively utilize the social influence among people to enhance the disease prevention. Additionally, such benefits of social influence is based on a reasonable ratio of “over-reacting” nodes (larger than 0.3 in Fig. 8), otherwise, the fully control result cannot be obtained.
Figure 6: Full phase diagrams $a - \beta_I$ of the final ratio of susceptible nodes $\rho_S(\infty)$ for different values of $\sigma$ and $\gamma$ with asymptomatic cases, where $\eta = 0.1, \alpha_o = p_o = 0.99, \alpha_u = p_u = 0.01, \mu = 0.5$.

Figure 7: Full phase diagrams $\alpha_o - p_o$ of the final ratio of susceptible nodes $\rho_S(\infty)$ for different values of $a$, where $\beta_I = 0.5, \beta_E = 0, \sigma = \gamma = 0.2, \eta = 0.1$.

Figure 8: Comparison of the final ratio of susceptible individuals $\rho_S(\infty)$ with respect to $a$ for two scenarios of two diseases, where $\eta = 0.1, \alpha_o = p_o = 0.99, \alpha_u = p_u = 0.01, \beta_I = 0.5, \beta_E = 0$.

5 Conclusion

In this study, we developed a three-layer network **SEIR-UAP** model to characterize the heterogeneous processes of information diffusion, behavior change and disease transmissions in social networks. We adopted the mean field approximation approach to obtain the analytical results, and did extensive simulations to examine the patterns of diseases transmissions in the presence of information diffusion and behavior change among people. We found that (a) the awareness of the disease plays the central role in preventing disease outbreak; (b) we need a reasonable ratio of “over-reacting” nodes to effectively control the disease outbreak; (c) diseases with a longer incubation period and
a higher recovery rate are easier to control because the processes of information diffusion and behavior change can help people prepare for the upcoming exposure to the disease; (d) it is more difficult to control the disease with asymptomatic cases.

In practice, with the absence of vaccine and control measure, the epidemic can still be well contained if the people are aware of the transmissibility and severity of disease and take proper self-protections (such as wearing a mask and using sanitizers frequently). Mass media is playing a key role in making the public aware of the disease and its transmissibility and severity. If the transmissibility and severity is played down by the mass media, more people will remain “under-reacting” and thus more people being infected eventually. However, if the mass media report unreasonably high transmissibility and severity of the disease, the “crying wolf” effect could result in people losing confidence in the public health system. Further research is needed to identify the optimal degree of transmissibility and severity being reported by the mass media.

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