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Effects of asymptomatic infection on the dynamical interplay between behavior and disease transmission in multiplex networks

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HIGHLIGHTS

- We study the impact of asymptomatic infection on the epidemic spread dynamics in multiplex networks.
- We assume infected can be isolation and non isolation, then compare the research results of these two cases.
- We take the individual heterogeneity into consideration and study whether it affect research results.

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ABSTRACT

Multiplex network theory is widely introduced to deepen the understanding of the dynamical interplay between self-protective behavior and epidemic spreading. Most of the existing studies assumed that all infected individuals can transmit disease-related information or can be perceived by their neighbors. However, owing to lack of distinct symptoms for patients in the initial stage of infection, the disease information cannot be transmitted in the population, which may lead to the wrong perception of infection risk and inappropriate behavior response. In this work, we divide infected individuals into Exposed-state (without obvious clinical symptoms) individuals and Infected-state (with evident clinical symptoms) individuals, both of whom can spread disease, but only Infected-state individuals can diffuse disease information. Then, in this work we establish UAU-SEIS (Unaware-Aware-Unaware-Susceptible-Exposed-Infected-Susceptible) model in multiplex networks and analyze the effect of asymptomatic infection and the isolation of Infected-state individuals on the density of infection and the epidemic threshold. Furthermore, we extend the UAU-SEIS model by taking the individual heterogeneity into consideration. Combined Markov chain approach and Monte-Carlo Simulations, we find that asymptomatic infection has an effect on the density of infected individuals and the epidemic threshold, and the extent of the effect is influenced by whether Infected-state individuals are isolated or treated. In addition, results show that the individual heterogeneity can lower the density of infected individuals, but cannot enhance the epidemic threshold.

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1. Introduction

The outbreaks of disease evoke the diffusion of disease-related information, which can trigger individuals' behavioral responses toward diseases, and these responses in turn bring profound impacts on the transmission of disease. For example, during the outbreak of severe acute respiratory syndrome (SARS) in 2003, people adopted some simple self-protective actions after they got the SARS-related information, including reducing travel, avoiding crowded places, washing hands frequently, staying at home, wearing a mask and so forth. The spontaneous protection of the public, combined with some compulsory measures implemented by governments or departments of health and hygiene, effectively controlled the spread of the disease [1–7]. about the researches on the effect of individual behavior on the transmission of disease attracted much attention [8–17]. For example, Funk et al. studied the effect of locally spreading awareness on the transmission of disease, and they found that in a well-mixed population, the spread of awareness can reduce the size of the outbreak, but cannot enhance the transmission of threshold [18]. Kiss et al. studied the impact of health information dissemination on epidemic outbreaks, and they found that if information spread fast enough, infection cannot survive [19].

Information disseminates on the information network and epidemic usually spreads through contact network. Besides, there are many differences between the topology structure of the information network and the contact network. However, most previous works assumed that the transmission of information and epidemic occurred on the same network but with different dynamics [20,21]. Recently, some scholars began to study the interplay dynamics of information and epidemic on multiplex networks [22–25]. For instance, Granell et al. established an SIS-UAU model, and used a Microscopic Markov Chain Approach to investigate the intertwined competition effects between awareness diffusion and epidemic transmission on multiplex networks, and they found that the threshold of epidemic depended on the awareness dynamics and the topology of information network [26]. They also extended their model to the general scenario where acquiring awareness did not imply total immunization, and analyzed the effect of mass media on epidemic transmission dynamics [27]. Mengfeng Sun et al. proposed a concrete interplay between individuals and spread of disease in quenched multiplex networks, and they found the phenomenon of oscillation in epidemic network with adaptive behaviors [28].

In addition, most previous studies assumed that once individuals got infected, they showed obvious symptom, and they can spread disease information to others [26,27]. However, for some infectious diseases (like Human Immunodeficiency Virus (HIV) [29]), in the early stage of infection, infected individuals have unobvious clinical symptoms, and they may even not be aware of infection. In other words, they cannot spread disease-related information to others, but they can infect others. There is the other scenario, they know that they are infected through medical examination or other means, but they worry about being discriminated against or isolated, so asymptomatic infected individuals may not choose to tell others the fact that they are infected. In both cases, the asymptomatic infection can affect individuals' assessment of risk and then may make inappropriate behavioral responses.

Recently, Zhang et al. studied the impacts of asymptomatic infection on the interplay between disease transmission and individuals' protection behaviors by dividing infectious individuals into U-state (without obvious clinical symptoms) and I-state (with obvious clinical symptoms) [30]. They found that asymptomatic infection had an important impact on the disease dynamics. However, they did not consider the dissemination of disease-related information, and assumed that susceptible individuals got disease information and took behavioral responses only when they contact with I-state individuals. In reality, individuals can get disease-related information from other individuals and take protective actions before an infected person appears in their neighborhoods [18,31]. Moreover, most previous researches assumed that the aware susceptible individuals can get infected with a smaller infection rate than an unaware susceptible individual, and the infection rate is same for all the aware individuals [19,26,27,32]. However, heterogenetic individuals [33] with different infected neighbors and perceived infection risk [34–37] took various behavioral responses and their infection rates were also different. Some studies incorporated the heterogeneity of individuals' perceived risk into individual behavior response model and got some interesting results. Wu et al. assumed that individuals' perceived infection risk depended on local information, global information and contact information. They found that local information and contact information could enhance the epidemic threshold [38]. Zhang et al. assumed that individuals took behavioral response based on the number of infected neighbors, and they found that hub nodes with different roles in speeding the spread of disease and preventing the transmission of disease [39]. Therefore, it is necessary to consider the impact of the individuals' heterogeneity on disease transmission in order to better predict and control the disease spread.

Inspired by previous works, we divided infected individuals into two classes: E-state (without obvious clinical symptoms) and I-state (with evident clinical symptoms) [30], and assumed that susceptible individuals can be infected by both E-state and I-state individuals in the physical contact layer, but only I-state individuals can disseminate disease-related information to their neighbors in the information layer. We applied Microscopic Markov Chain Approach (MMCA) and Monte-Carlo Simulations to analyze the impact of asymptomatic infection on the dynamical interplay between behavior and disease transmission on the two-layer network [25–27]. Furthermore, we extended the model by considering the individuals' heterogeneity and studied the influence of individuals' heterogeneity to infection risk on the disease transmission dynamics.

Fig. 1. Description of state transition and illustration of the structure of the multiplex network.

The rest of this paper is organized as follows. Section 2 briefly describes the proposed model including basic model and individual heterogeneity model. We present the theoretical analysis from Microscopic Markov Chain Approach (MMCA) in Section 3. Then Section 3.2 presents the simulation results based on Monte-Carlo approach. Finally, we give the conclusions and discussions in Section 4.

2. Model

2.1. Basic model

In this work, we extend the model of reference [30] from a single layer network to a multiplex network. As described in Fig. 1, we build a multiplex network with two layers that one is physical contact layer on where the disease spreads and the other is information layer on where the dissemination of information evolves [30]. On the information layer, we use a UAU (Unaware–Aware–Unaware) process to describe the disease-related information dissemination. U-state (Unaware state) individuals do not acquired any information about the disease, and have not perceived any infection risk. A-state (Aware state) individuals are aware of the infection risk and can transmit their information to their neighbors on the information layer. Each A-state individual transmits the disease information to their U-state neighbors on information layer with a probability λ . A-state individuals lose (forget) the disease information and return to state of U-state with a probability δ . On physical contact layer, we adopt the SEIS (Susceptible–Exposed–Infected–Susceptible) epidemiological model. S-state (Susceptible state) nodes get infected and firstly go to E-state (Exposed state) with a probability β , and then E-state individuals enter the I-state with a probability σ , and I-state nodes can go back to the S-state with a recovery rate μ . The interaction between information dissemination and disease transmission is modeled as follows. A U-state node on information layer will automatically become aware and transmit the disease information to their neighbors on the information layer once its counterpart on contact layer enter I-state. Similarly, S-state nodes on contact layer will take behavior response for reducing susceptibility once their counterpart on information layer become A-state.

In addition, when infected individuals present obvious clinical symptoms, they usually will be hospitalized for treatment or be kept from contact [26]. In the circumstances, S-state node can only be infected by E-state, so we consider two types of I-state individuals: I-state individuals who can spread viruses [30] and cannot spread viruses [40] in this study.

The disease transmission rate between susceptible individuals and infected individuals is determined by two factors: the susceptibility of susceptible individuals and the infectivity of infected individuals. We use A to indicate the susceptibility and apply T to indicate the infectivity. We assume that the transmission rate between susceptible individual i and infected individual j is p_{ij} , then we obtain [38]:

$$p_{ij} = \begin{cases} A_i T_j & i \text{ is susceptible and } j \text{ is infectious or exposed} \\ A_j T_i & j \text{ is susceptible and } i \text{ is infectious or exposed} \\ 0 & \text{otherwise} \end{cases} \quad (1)$$

The susceptibility of S-state individuals is $A = 1$ and the infectivity of E-state (I-state) individuals is $T = \beta$ when their counterparts in information layer are U-state; An Aware susceptible individual reduces its own susceptibility by a factor θ (here $0 \leq \theta \leq 1$), in the other word the susceptibility of Aware susceptible individuals is $\theta\beta$.

Then the transmission rate p_{ij} between susceptible individual i and infected individual (E-state or I-state) j in the two different types of I-state is shown in Table 1.

Table 1

The transmission rate p_{ij} in both cases of I-state individuals can spread disease and cannot spread disease.

The type of I	$i \& j$					
	US&UE	US&AE	AS&UE	AS&AE	US&AI	AS&AI
Spread disease	β	β	$\theta\beta$	$\theta\beta$	β	$\theta\beta$
Cannot spread disease	β	β	$\theta\beta$	$\theta\beta$	0	0

Table 2

The transmission rate p_{ij} in the case of I-state individuals cannot spread viruses.

$i \& j$	US&UE	US&AE	AS&UE	AS&AE
p_{ij}	β	$e^{-\alpha k_j^{inf}} \beta$	$e^{-\alpha k_i^{inf}} \beta$	$e^{-\alpha k_i^{inf}} e^{-\alpha k_j^{inf}} \beta$

Fig. 2. The relationship between the susceptibility coefficient of node i and the number of I-state neighbors of node i .

2.2. Individual heterogeneity model

We do not consider the heterogeneity of individuals' perceived risk in basic model. In reality, individuals who have different numbers of infected neighbors perceive different level of infection risk and take different behavior responses. In addition, existing works usually assumed that only susceptible individuals adopted protective measures, and E-state individuals did not take behavioral response [30,32,41,42]. However, owing to E-state individuals have no obvious clinical symptoms, most of them are not aware that they have been infected, so they will take behavioral responses as susceptible individuals when they perceive the infection risk. In this study, we assume that S-state individuals and E-state individuals change their behaviors based on the number of I-state neighbors. We adopt exponential function to describe the susceptibility of S-state individuals and infectivity of E-state individuals. If a S-state(E-state) node i has k_i^{inf} I-state neighbors, then the susceptibility (infectivity) of node i reduces to $e^{-\alpha k_i^{inf}} (\beta e^{-\alpha k_i^{inf}}) (\alpha \geq 0)$, where α stands for individuals' sensitivity for perceived infection risk and individuals are more sensitive to the risk of disease when α is larger. From Fig. 2, we can observe that in the case of $\alpha = 0.2$, individuals will take strong protective measures only when the number of I-state neighbors reaches 20; however, in the case of $\alpha = 1$, individuals will take strong protective behavior as long as one infected appears in their neighbors. In this part, we focus on the impact of the individual heterogeneity on the transmission of disease. Therefore, for simplifying the individual heterogeneity model, we only consider one scenario that I-state individuals cannot spread viruses, and the transmission rate p_{ij} between susceptible individual i and infected individual (E-state or I-state) j is shown in Table 2.

3. Main results

3.1. Microscopic Markov Chain Approach (MMCA)

In this part, we use MMCA to analyze the effect of asymptomatic infection on the interplay between information diffusion and epidemic transmission in case of basic model and Individual heterogeneity model respectively.

3.1.1. Basic model

Let us denote a_{ij} and b_{ij} as the adjacency matrices of the information layer and the physical contact layer, respectively. Each node i is in one of the five states at time t : US (Unaware–Susceptible), AS (Aware–Susceptible), UE (Unaware–Exposed), AE (Aware–Exposed) and AI (Aware–Infected). The probability of node i in one of the five states at time t is $p_i^{US}(t)$, $p_i^{AS}(t)$, $p_i^{UE}(t)$, $p_i^{AE}(t)$ and $p_i^{AI}(t)$. We assume that the probability for an unaware node without being informed by any neighbors is $r_i(t)$, the probability for an unaware susceptible node without being infected by any neighbors is $q_i^U(t)$, and the probability for an aware susceptible node without being infected by any neighbors is $q_i^A(t)$.

The $r_i(t)$ is same for two cases (I-state individuals can spread viruses and cannot spread viruses):

$$r_i(t) = \prod_j (1 - a_{ji} p_j^A(t) \lambda) \quad (2)$$

There are some differences between $q_i^U(t)$ and $q_i^A(t)$, therefore we give $q_i^U(t)$, $q_i^A(t)$ respectively:

I-state individuals can spread viruses:

$$q_i^U(t) = \prod_j (1 - b_{ji} (p_j^{AI}(t) + p_j^{UE}(t) + p_j^{AE}(t)) \beta) \quad (3)$$

$$q_i^A(t) = \prod_j (1 - b_{ji} (p_j^{AI}(t) + p_j^{UE}(t) + p_j^{AE}(t)) \theta \beta) \quad (4)$$

I-state individuals cannot spread viruses:

$$q_i^U(t) = \prod_j (1 - b_{ji} (p_j^{UE}(t) + p_j^{AE}(t)) \beta) \quad (5)$$

$$q_i^A(t) = \prod_j (1 - b_{ji} (p_j^{UE}(t) + p_j^{AE}(t)) \theta \beta) \quad (6)$$

For each possible state of a node at time t , there is a certain probability that may change to other states at time $t + 1$. Taking node i as an example, the Markov state transition trees are presented in Fig. 3 Based the Markov state transition trees, we can easily derive the MMCA equations for each state [23,24]:

$$p_i^{US}(t + 1) = p_i^{US}(t) r_i(t) q_i^U(t) + p_i^{AS}(t) \delta q_i^U(t) + p_i^{AI}(t) \delta \mu \quad (7)$$

$$p_i^{AS}(t + 1) = p_i^{US}(t) (1 - r_i(t)) q_i^A(t) + p_i^{AS}(t) (1 - \delta) q_i^A(t) (1 - \delta) \mu \quad (8)$$

$$p_i^{UE}(t + 1) = p_i^{US}(t) r_i(t) (1 - q_i^U(t)) + p_i^{AS}(t) \delta (1 - q_i^U(t)) + p_i^{UE}(t) r_i(t) (1 - \sigma) + p_i^{AE}(t) \delta (1 - \sigma) \quad (9)$$

$$p_i^{AE}(t + 1) = p_i^{US}(t) (1 - r_i(t)) (1 - q_i^U(t)) + p_i^{AS}(t) (1 - \delta) (1 - q_i^U(t)) + p_i^{UE}(t) (1 - r_i(t)) (1 - \sigma) + p_i^{AE}(t) (1 - \delta) (1 - \sigma) \quad (10)$$

$$p_i^{AI}(t + 1) = p_i^{UE}(t) \sigma + p_i^{AE}(t) \sigma + p_i^{AI}(t) (1 - \mu) \quad (11)$$

The epidemic threshold determines whether the disease can spread through the individuals or die out, so it is necessary to analyze the relationship between other parameters and the epidemic threshold. We obtain the epidemic threshold according to the above MMCA equations. When the system goes to the stationary state, we have $p_i(t + 1) = p_i(t)$ for all the nodes and states. Since we assume that S-state individuals firstly go to E-state after being infected by E-state or I-state individuals, therefore the epidemic threshold is given by the order parameter p^E with the representation of the final fraction for E-state nodes in the system. Near the onset of the epidemic spreading, the probability of nodes to be infected is approaching 0, i.e. $p_i^E = \epsilon_i \ll 1$.

We also analyze the relationship between other parameters and the epidemic threshold in two cases: I-state individuals can spread viruses and cannot spread viruses.

Fig. 3. The Markov state transition trees.

(1) I-state individuals can spread viruses

Accordingly, Eqs. (3) and (4) are approximated as:

$$\begin{aligned} q_i^U &\approx 1 - \sum_j b_{ji}(p_j^{AI} + p_j^{UE} + p_j^{AE})\beta \\ &= 1 - \sum_j b_{ji}(p_j^{AI} + p_j^E)\beta \\ &= 1 - \sum_j b_{ji}\left(\frac{\sigma}{\mu}p_j^E + p_j^E\right)\beta \end{aligned} \quad (12)$$

$$\begin{aligned} q_i^A &\approx 1 - \sum_j b_{ji}(p_j^{AI} + p_j^{UE} + p_j^{AE})\theta\beta \\ &= 1 - \sum_j b_{ji}(p_j^{AI} + p_j^E)\theta\beta \\ &= 1 - \sum_j b_{ji}\left(\frac{\sigma}{\mu}p_j^E + p_j^E\right)\theta\beta \\ &= 1 - \theta\left(\frac{\sigma}{\mu} + 1\right)\eta_i \end{aligned} \quad (13)$$

$$\text{where } \beta \sum_j b_{ji}p_j^E = \eta_i \quad (14)$$

In the steady state, we can get:

$$p_i^E = (1 - \sigma)p_i^E + p_i^{US}[(1 - r_i)(1 - q_i^A) + r_i(1 - q_i^U)] + p_i^{AS}[(1 - \delta)(1 - q_i^A) + \delta(1 - q_i^U)] \quad (15)$$

$$\text{where } p_i^U = p_i^{US} + p_i^{UE} \quad (16)$$

Near the onset of the spread of epidemic $p_i^{UE} \rightarrow 0$, We have $p_i^{US} \approx p_i^U$ in a similar way. therefore, Eqs. (15) can be rewritten as:

$$p_i^E = (1 - \sigma)p_i^E + p_i^U[(1 - r_i)(1 - q_i^A) + r_i(1 - q_i^U)] + p_i^A[(1 - \delta)(1 - q_i^A) + \delta(1 - q_i^U)] \quad (17)$$

$$\eta_i^E = (1 - \sigma)\eta_i + p_i^U \left[(1 - r_i)\left(\frac{\sigma}{\mu}\right)\theta\eta_i + r_i\left(\frac{\sigma}{\mu} + 1\right)\eta_i \right] + p_i^A \left[(1 - \delta)\left(\frac{\sigma}{\mu} + 1\right)\theta\eta_i + \delta\left(\frac{\sigma}{\mu} + 1\right)\eta_i \right] \quad (18)$$

which can be written as:

$$\sigma\eta_i = [p_i^U(1 - r_i) + p_i^A(1 - \delta)]\left(\frac{\sigma}{\mu} + 1\right)\theta\eta_i + (p_i^U r_i + p_i^A \delta)\left(\frac{\sigma}{\mu} + 1\right)\eta_i \quad (19)$$

Removing $O(\epsilon_i)$ terms in the stationary state of Eqs. (7) and (8) we can get:

$$p_i^A = p_i^U(1 - r_i) + p_i^A(1 - \delta) \quad (20)$$

$$p_i^U = p_i^U r_i + p_i^A \delta \quad (21)$$

Substituting Eqs. (20) and (21) in Eqs. (19) lead to:

$$\sigma\eta_i = p_A\left(\frac{\sigma}{\mu} + 1\right)\theta\eta_i + p_i^U\left(\frac{\sigma}{\mu} + 1\right)\eta_i \quad (22)$$

which can be written as:

$$\sum_j t[\beta(p_i^A\theta + p_i^U)\left(\frac{\sigma}{\mu} + 1\right)b_{ji} - v_{ij}\sigma]\epsilon_j = 0 \quad (23)$$

where v_{ij} are the elements of the identity matrix. Defining matrix $\mathbf{H}$ with elements

$$h_{ij} = (p_i^A\theta + p_i^U)b_{ji} \quad (24)$$

The non-trivial solutions of Eq. (23) are eigenvectors of $\mathbf{H}$, whose eigenvalues are equal to $\frac{\sigma\mu}{\beta(\sigma+\mu)}$, therefore the epidemic threshold is given by the largest eigenvalue of $\mathbf{H}$:

$$\beta^c = \frac{\sigma\mu}{\Lambda_{\max}(\mathbf{H})(\sigma + \mu)} \quad (25)$$

The matrix $\mathbf{H}$ depends on p_i^A and p_i^U , and there is $p_i^A + p_i^U = 1$, so we can get:

$$p_i^A = (1 - p - i^A)(1 - r_i) + p_i^A(1 - \sigma) \quad (26)$$

$$\text{where } r_i(t) = \prod_j (1 - \alpha_{ji} p_j^A(t) \lambda) \quad (27)$$

(2) I-state individuals cannot spread viruses Accordingly, Eqs. (5) and (6) are approximated as:

$$q_i^U \approx 1 - \sum_j b_{ji}(p_j^{UE} + p_j^{AE})\beta = 1 - \sum_j b_{ji}p_j^E\beta = 1 - \eta_i \quad (28)$$

$$q_i^A \approx 1 - \sum_j b_{ji}(p_j^{UE} + p_j^{AE})\theta\beta = 1 - \sum_j b_{ji}p_j^E\theta\beta = 1 - \theta\eta_i \quad (29)$$

$$\text{where } \beta \sum_j b_{ji}p_j^E = \eta_i \quad (30)$$

In the steady state, we can get:

$$p_i^E = (1 - \sigma)p_i^E + p_i^{US}[(1 - r_i)(1 - q_i^A) + r_i(1 - q_i^U)] + p_i^{AS}[(1 - \delta)(1 - q_i^A) + \delta(1 - q_i^U)] \quad (31)$$

$$\text{where } p_i^U = p_i^{US} + p_i^{UE} \quad (32)$$

Near the onset of the spread of epidemic $p_i^{UE} \rightarrow 0$, then we have $p_i^{US} \approx p_i^U$, and we have $p_i^{AS} \approx p_i^A$ in a similar way, therefore, Eqs. (31) can be rewritten as:

$$p_i^E = (1 - \sigma)p_i^E + p_i^U[(1 - r_i)(1 - q_i^A) + r_i(1 - q_i^U)] + p_i^A[(1 - \delta)(1 - q_i^A) + \delta(1 - q_i^U)] \quad (33)$$

$$\eta_i = (1 - \sigma)\eta_i + p_i^U[(1 - r_i)\theta\eta_i + r_i\eta_i] + p_i^A[(1 - \delta)\theta\eta_i + \delta\eta_i] \quad (34)$$

which can be written as:

$$\sigma\eta_i = [p_i^U(1 - r_i) + p_i^A(1 - \delta)]\theta\eta_i + (p_i^U r_i + p_i^A \delta)\eta_i \quad (35)$$

Removing $O(\epsilon)$ terms in the stationary state of Eqs. (7) and (8) we can get:

$$p - i^A = p_i^U(1 - r_i) + p_i^A(1 - \delta) \quad (36)$$

$$p - i^U = p_i^U r_i + p_i^A \delta \quad (37)$$

Substituting Eqs. (36) and (37) in Eqs. (35) lead to:

$$\sigma\eta_i = p_i^A\theta\eta_i + p_i^U\eta_i \quad (38)$$

which can be written as:

$$\sum_j [\beta(p_i^A\theta + p_i^U)b_{ji} - \gamma_{ij}\sigma]\epsilon_j = 0 \quad (39)$$

where γ_{ij} are the elements of the identity matrix. Defining matrix $\mathbf{H}$ with elements

$$h_{ij} = (p_i^A\theta + p_i^U)b_{ji} \quad (40)$$

The non-trivial solutions of Eq. (39) are eigenvectors of $\mathbf{H}$, whose eigenvalues are equal to $\frac{\sigma}{\beta}$, therefore the epidemic threshold is given by the largest eigenvalue of $\mathbf{H}$:

$$\beta^c = \frac{\sigma}{\Lambda_{\max}(\mathbf{H})} \quad (41)$$

The matrix $\mathbf{H}$ depends on p_i^A and p_i^U , and there is $p_i^A + p_i^U = 1$, so we can get:

$$p_i^A = (1 - p_i^A)(1 - r_i) + p_i^A(1 - \delta) \quad (42)$$

where $r_i(t) = \prod_j (1 - \alpha_{ji} p_j^A(t) \lambda)$

Table 3

The descriptions and baseline values used in simulations.

Parameter	Meaning	Value
N	Network size	1000
k	Average degree of nodes	6
β	The infectivity of disease	0~1
σ	The transition rate form E-state to I-state	0.5
μ	The transition rate form I-state to S-state	0.4
λ	The transmission rate of information/awareness	0.3
δ	The rate of losing information/awareness	0.6
θ	The reduction factor of susceptibility	0.5
α	Degree of individual sensitivity to infection risk	0.4

3.1.2. Individual heterogeneity model

$r_i(t)$ is same as the above model without considering the heterogeneity of individuals' perceived risk:

$$r_i(t) = \prod_j (1 - \alpha_{ji} p_j^A(t) \lambda) \quad (43)$$

Since $q_i^U(t)$ and $q_i^A(t)$ have some differences with the basic model, we present $q_i^U(t)$ and $q_i^A(t)$ as below:

$$q_i^U(t) = \prod_j [1 - b_{ji}(p_j^{UE}(t) + p_j^{AE}(t)e^{-\alpha k_j^{inf}})\beta] \quad (44)$$

$$q_i^A(t) = \prod_j [1 - b_{ji}(p_j^{UE}(t)e^{-\alpha k_i^{inf}} + p_j^{AE}(t)e^{-\alpha k_i^{inf}} e^{-\alpha k_j^{inf}})\beta] \quad (45)$$

The Markov state transition trees and MMCA equations are the same as the basic model without considering the heterogeneity of individuals' perceived risk. Therefore, MMCA equations and The Markov state transition trees are omitted here. Due to the complexity of the individual heterogeneity model, it is hard to get the analytic solutions of the epidemic threshold by using MMCA.

3.2. Numerical simulations and results

3.2.1. Basic model

Here we investigate the effects of asymptomatic infection and the heterogeneity of individuals' perceived risk on the interactive dynamics of behavior and disease in multiplex networks. First, we build a configuration network with degree distribution $p(k) \sim k^{-2.5}$ and the number of nodes $N = 1000$ as the physical contact layer. Moreover, we generate the information layer by adding 400 extra random links in the physical contact layer [23,24]. a_{ij} and b_{ij} denote the adjacency matrices of the physical contact layer and the information layer, respectively. All simulation results are obtained by averaging 100 independent realizations.

We crosscheck the effectiveness of our analysis results from MMCA with Monte-Carlo simulation in Fig. 4, and the parameter descriptions and baseline values in simulations are presented in Table 3. Without special instructions, the value of parameters in each figure is the same as the baseline of the parameters. From Fig. 4, we can observe that the results of MMCA and Monte-Carlo are similar. Therefore, we mainly use MMCA approach to obtain the following results.

We then study the effects of main parameters of the model: σ (the transition rate form E-state to I-state), θ (the reduction factor of susceptibility), ω (the reduction factor of infectivity) and α (Degree of individual sensitivity to infection risk). Fig. 5 describes the density of sum of I-state and E-state nodes for different values of σ . The panels depict the scenarios where I-state individuals can spread viruses (left) and I-state individuals cannot spread viruses (right). The parameter σ accounts for the transition rate of infected individuals from asymptomatic to obvious symptomatic. From Fig. 5, we can see that, for high values of σ , the fraction of I-state and E-state nodes are diminished, and the onset of the epidemic is enhanced. The result is not difficult to understand. Since increasing σ means that asymptomatic infected individuals more quickly enter I-state and then unaware nodes have more chances to obtain disease-related information; As a result, the epidemic size is reduced and the epidemic threshold is enhanced. We can also see, if comparing with the left and right panels, we can also see that σ has a more pronounced influence in the right panel.

Fig. 6 depicts the density of the sum of I-state and E-state nodes for different values of θ . In accordance with Fig. 5, the left panel for the case of I-state individuals can spread viruses and the right one for the case of I-state individuals cannot spread viruses. The parameter θ regulates the susceptibility of A-state nodes which ranges from 0 (representing total immunization) to 1. From Fig. 6, we can observe that for low values of θ the fraction of I-state and E-state nodes is lowered, and the onset of the epidemic is slightly enhanced. With the decreasing of θ , the susceptibility of A-state nodes is reduced. That is the reason why decreasing the value of θ can effectively suppress the outbreak of epidemic.

In order to systematically investigate the effects of σ on the epidemic dynamics, we further explore the full phase diagram $\lambda - \beta$ in Fig. 7. From Fig. 7, we can observe that the epidemic dynamic is not influenced by λ when β is smaller

Fig. 4. The comparison of the stationary fraction of A-state, I-state and E-state nodes using Monte Carlo (dotted line) simulations and the MMCA approach (solid line) as a function of the infectivity β .

Fig. 5. The stationary fraction of I-state and E-state nodes as a function of the infectivity β for different value of σ . Left panel (for case of I-state individuals) can spread viruses, and right panel (for case of I-state individuals) cannot spread viruses. The value of other parameters is the same as Table 3.

than the epidemic threshold, since the disease cannot spread out itself. When β exceed the epidemic threshold, the sum stationary fraction of I-state and E-state nodes decreases with λ for different values of σ . Overall, we can see that the epidemic size and epidemic threshold are influenced by the value of σ . With the increasing of σ , the epidemic size is reduced, and the epidemic threshold is enhanced. In addition, by comparing with the left panels and the right panels, σ has a more pronounced influence on epidemic dynamics in right panels than in left panels.

For checking the effectiveness of our analytical results from MMCA approach, then we investigate the impact of asymptomatic infection and disease-related information dissemination on epidemic threshold. Combined Figs. 8 and 9, we can see that the epidemic threshold β_c increases with λ and reaches a certain value when λ exceeds a certain value for different σ . And the epidemic threshold β_c increase with σ for different λ . By comparing with the left panel and right panel in Fig. 9, we can see that in case of I-state individuals can spread viruses the epidemic threshold β_c increases linearly with σ , in the case of I-state individuals spreading viruses, but the epidemic threshold β_c shows curve growth with σ in the case of I-state individuals not spreading viruses. The above results are in good agreement with the analytical results from MMCA approach. The mechanism of such difference between the left panel and right panel of Fig. 9 needs further investigation.

Fig. 6. The stationary fraction of I-state and E-state nodes as a function of the infectivity β for different value of θ . Left panel for case of I-state individuals can spread viruses; Right panel for case of I-state individuals cannot spread viruses. The value of other parameters is the same with Table 3.

3.2.2. Individual heterogeneity model

Fig. 10 depicts the stationary fraction of I-state and E-state nodes as a function of the infectivity β for different value of σ after taking the heterogeneity of individuals' perceived risk into consideration. From Fig. 10, we can observe that the effect of σ on the epidemic transmission dynamics still exists and is similar with the effect without considering the heterogeneity of individuals' perceived risk. The stationary fraction of I-state and E-state nodes decreases significantly with the increasing of σ . But the effect of σ on epidemic threshold is not obviously compared with the change of epidemic size. Whether the disease being prevented is determined by the number of individuals taking behavior response and the degree of behavior response. When setting a small α which describes individuals' sensitivity for perceived infection risk, we can find that the degree of behavior response is not strong. Therefore, the change of epidemic threshold is not large.

Fig. 11 describes the stationary fraction of I-state and E-state nodes as a function of the infectivity β for different value of α . From Fig. 11, we can see that the stationary fraction of I-state and E-state nodes decreases as the increasing of α . But, the effect of α on the epidemic threshold is not evident. When β is smaller than the epidemic threshold, the disease cannot spread out through the individuals and k_i^{inf} is very small. As the susceptibility (infectivity) $e^{-\alpha k_i^{inf}} (\beta e^{-\alpha k_i^{inf}})$ depends on α and the number of I-state neighbors k_i^{inf} simultaneously, so no matter what the value of α is, the epidemic threshold almost has no change.

In order to systematically investigate the effects of σ on the epidemic dynamics in the case of individual heterogeneity model, we further explore the full phase diagram $\lambda - \beta$ for different values of σ in Fig. 12. We also investigate the influence of σ and the transmission rate of information/awareness λ on the epidemic threshold β_c in the Individual heterogeneity model. The result is similar with the second case of the basic model, as the assumption that I-state individuals cannot spread viruses in the individual heterogeneity model is consistent with the assumption in the second case of the basic model. In other words, asymptomatic infection still has an impact on the epidemic dynamics after taking the heterogeneity of individuals' perceived risk into consideration (see Figs. 13 and 14).

4. Conclusions and discussions

Considering that the asymptomatic infection of exposed individuals may lead to information inconsistency between disease and information layers and further influence people's inappropriate behavior, this paper proposed a SEIS-UAU model on a two-layers network which can characterize this kind of phenomenon very well. We analyzed the effect of asymptomatic infection on the coupled process of awareness/information and disease under different circumstances and obtained some interesting results. The results based on Microscopic Markov Chain approach and Monte-Carlo approach revealed that the size of infection can be reduced once the transition rate from E-state to I-state σ is increased or the reduction factor of susceptibility θ is decreased. In addition, the impact of asymptomatic infection on the epidemic dynamic is more pronounced in the case of I-state individuals not spreading viruses than in the case of I-state individuals spreading viruses. We further extended the model by considering the heterogeneity of individuals' perceived risk. The results showed that σ still had an impact on the epidemic dynamics, and the density of infection could be reduced with the increase of individual's sensitivity degree to infection risk α , but α did not have an obvious impact on the epidemic threshold.

Fig. 7. The stationary fraction of I-state and E-state nodes. Full phase diagram $\lambda - \beta$ for different values of σ . Left panels for case of I-state individuals can spread viruses; Right panels for case of I-state individuals cannot spread viruses.

Fig. 8. The epidemic threshold β_c as a function of the transmission rate of information/awareness λ for different values of σ . Left panel for case of I-state individuals can spread viruses; Right panel for case of I-state individuals cannot spread viruses.

Fig. 9. The epidemic threshold β_c as a function of σ for different values of the transmission rate of information/awareness λ . Left panel for case of I-state individuals can spread viruses; Right panel for case of I-state individuals cannot spread viruses.

Fig. 10. The stationary fraction of I-state and E-state nodes as a function of the infectivity β for different value of σ .

Fig. 11. The stationary fraction of I-state and E-state nodes as a function of the infectivity β for different value of α .

Fig. 12. The stationary fraction of I-state and E-state nodes. Full phase diagram $\lambda - \beta$ for different values of σ .

Fig. 13. The epidemic threshold β_c as a function of the transmission rate of information/awareness λ for different values of σ .

Fig. 14. The epidemic threshold β_c as a function of σ for different values of the transmission rate of information/awareness λ .

The interplay between the disease and the information transmission has attracted much interest, but still face many challenges. In this work, we investigated the effect of asymptomatic infection on the epidemic dynamics in multiplex networks. However, in order to simplify the model, we only considered the transmission of information/awareness among individuals and ignored the impact of other sources of information (mass media, self-awareness). In addition, we did not consider the impact of the cost of the response behavior of individuals. In the future, we will improve the model to better predict and control disease transmission.

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