The impact of the global and local awareness diffusion on epidemic transmission considering the heterogeneity of individual influences

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Abstract In this paper, we propose a coupled awareness—epidemic spreading model considering the heterogeneity of individual influences, which aims to explore the interaction between awareness diffusion and epidemic transmission. The considered heterogeneities of individual influences are threefold: the heterogeneity of individual influences in the information layer, the heterogeneity of individual influences in the epidemic layer and the heterogeneity of individual behavioral responses to epidemics. In addition, the individuals’ receptive preference for information and the impacts of individuals’ perceived local awareness ratio and individuals’ perceived epidemic severity on self-protective behavior are included. The epidemic threshold is theoretically established by the microscopic Markov chain approach and the mean-field approach. Results indicate that the critical local and global awareness ratios have two-stage effects on the epidemic threshold. Besides, either the heterogeneity of individual influences in the information layer or the strength of individuals’ responses to epidemics can influence the epidemic threshold with a nonlinear way. However, the heterogeneity of individual influences in the epidemic layer has few effect on the epidemic threshold, but can affects the magnitude of the final infected density.

Keywords Heterogeneity of individual influences · Epidemic transmission · Awareness diffusion · Microscopic Markov chain approach

1 Introduction

Throughout history, the outbreak of epidemics has been a major disaster threatening human survival. For example, from 2019 to now, the sudden outbreak of COVID-19 [1–4] has deeply affected the lifestyle of people all over the world and has an indelible negative impact on the world economy. During the outbreak of COVID-19, epidemic information is transmitted by word of mouth or social media [5–9]. In order to avoid being infected by a disease, people will take various self-protective measures, such as reducing outdoor activities, wearing masks, and paying attention to disinfection and vaccination [10–13]. Therefore, more and more researchers focus on the interaction between awareness diffusion and epidemic transmission [14–17]. Funk et al. firstly proposed a mathematical model of the interaction between epidemic and awareness [18]. Then, Wang et al. established an asymmetrically coupled epidemic-awareness spreading model and found that the information threshold remains unchanged, and the epidemic threshold could be increased if there is structural correlation between the contact layer and the communication layer [19].

With the development of technology and society, people can conveniently obtain epidemic informa-
tion through different channels. The information we get is mainly from our acquaintance and the news media, which are classed as the local information [20,21,30] and the global information [14,24], respectively. At the same time, people usually take different behaviors based on the information they received, which affects the spread of the disease. Consequently, some researchers considered that individuals could be directly or indirectly affected by different information in the process of epidemic transmission, thereby affecting the disease spreading. For instance, Guo et al. investigated the effects of local awareness on epidemic transmission in the coupled UAU-SIS model. The result indicated that the local awareness ratio have profound effects on epidemic threshold [22]. Kan et al. assumed that the generation of self-awareness of susceptible individuals is affected by their infected neighbors. In addition, they proved that self-awareness could reduce the infected density but could not enhance the epidemic threshold [23]. Zuo et al. found that the disease spreading could be affected by the effective transmission rate of awareness and the neighbors’ behavior status [24]. Moreover, in real life, there are many factors play an important role on information dissemination. These factors affect the spread of diseases by influencing the information transmission. Hence, a number of researchers had studied the influences of various factors in the awareness transmission on epidemic spreading from a macroscopic perspective. For example, Wang et al. studied the co-evolution spreading of positive information, negative information and epidemic in the influence of mass media. They found that enhancing information dissemination and the propaganda of media could suppress the epidemic spreading [25]. Zhang et al. researched the impacts of awareness diffusion, individual vigilance and rational behavior on epidemic transmission. Their result indicated that the rational behavior plays an important role in controlling epidemic transmission [26].

In the above studies, most researchers focused on the effect of the individual behavior changes induced by information transmission on epidemic spreading. However, almost all of these studies assumed that all individuals were treated equally [21]. In other words, the infection or awareness rate was the same for each individual. Obviously, it is not consistent with the real situation. In fact, different people have different sensitivities or reactions to diseases and information. Therefore, an increasing number of investigators have considered the heterogeneity of individuals while modeling the dynamic processes between epidemic transmission and information diffusion [27–29]. For instance, Pan et al. explored the impacts of the influence heterogeneity in the information layer, the influence heterogeneity in the epidemic layer and the heterogeneity of individual responses to epidemics on epidemic spreading [30]. Guo et al. described the heterogeneity of individuals in a heterogeneous spreading model by the degree approach and the k-core measure, respectively. The result demonstrated that the k-core measure leads to a much larger final epidemic size than the degree approach [31]. Chen et al. investigated the effects of heterogeneous self-awareness distribution and heterogeneous degree distribution on the epidemic dynamic. The result showed that the heterogeneity of self-awareness distribution could suppress the outbreak of an disease and the heterogeneity of degree distribution could enhance the epidemic spreading [32]. Nevertheless, there is still a significant gap in the study of heterogeneity. In the face of complex reality, the current researches still need to be expanded and innovated.

In this paper, to explore the impact of the global and local awareness diffusion on epidemic transmission, we propose a coupled awareness-epidemic spreading model considering the heterogeneity of individual influences. The considered heterogeneities of individual influences are as follows: the heterogeneity of individual influences in the information layer, the heterogeneity of individual influences in the epidemic layer, and the heterogeneity of individual behavioral responses to epidemics. In addition, the contributions of this paper are threefold as follows: (1) using the threshold model, we present and analyze the epidemic spreading problem with the global awareness diffusion; (2) individuals’ perceived local awareness ratio and individuals’ self-protective behavior are incorporated into the proposed epidemic spreading model; (3) we confirm that individuals’ information receptive preference has a powerful influence on epidemic spreading.

The rest of this paper is organized as follows: in Sect. 2, we introduce a coupled awareness-epidemic spreading model considering the heterogeneity of individual influences. Then, in Sect. 3, we use the microscopic Markov chain approach (MMCA) to analyze the model and derive the analytical expression of the epidemic threshold. In Sect. 4, according to the simulation results, we explore the impact of the heterogeneity of individual influences on the the interplay of epidemics.
and awareness. Finally, we conclude the paper in Sect. 5.

2 Model description

In this paper, we propose a coupled awareness—epidemic spreading model on a two-layered multiplex network. In addition, we describe the processes of local and global awareness diffusion by the threshold model. Meanwhile, we employ an SIS model to describe the process of epidemic transmission. As shown in Fig. 1, the information layer describes the process of awareness diffusion, where links represent the virtual contacts among individuals. In this layer, there are two possible states for each node: Aware (A) or Unaware (U), which means that individuals are aware or unaware of epidemics, respectively. The epidemic layer describes the process of epidemic transmission, where links represent the physical contacts among individuals. In this layer, the state of each node is the one of two states: Susceptible (S) or Infected (I), which means that individuals are susceptible or infected, respectively. For the sake of simplicity, both the information layer network and the epidemic layer network are undirected and unweighted. Furthermore, there is a one to one correspondence between the nodes of these two layer networks, and different layers have different topologies.

In the information layer, an unaware individual becomes aware when its perceived local awareness ratio $A^\text{perc}_i$ exceeds the critical local awareness ratio $\theta_1$. Otherwise, the individual will continue to be unaware. Moreover, all individuals become aware when the average awareness rate of all individuals exceeds the critical global awareness $\theta_2$. In addition, an aware individual becomes unaware with probability $\delta$. In the epidemic layer, a susceptible individual gets infected with probability $\beta$ after a contact with an infected individual, and an infected individual recovers to be susceptible with probability $\mu$.

For simplicity, we assume that infected individuals become aware in the information layer. Obviously, susceptible individuals will take self-protective measures to avoid being infected if they become aware in the information layer. Therefore, the probability of individual $i$ being infected is affected by the status of individual $i$ in the information layer. The unaware susceptible individual gets infected with probability $\beta^U = \beta$. The aware susceptible individual gets infected with probability $\beta^A_i = \gamma_i \beta^U$ ($\gamma_i \in [0, 1]$). The parameter $\gamma_i$ regulates the probability of an aware individual $i$ being infected. $\gamma_i = 0$ denotes that the aware individual $i$ is completely immune to the infection. $\gamma_i = 1$ denotes that the aware individual $i$ is ineffective to reduce the infectivity of the epidemic by taking self-protective measures. In previous studies, researchers assumed that
aware individuals would take equivalent self-protective measures and have the same risk of being infected. However, the heterogeneous effects of individuals on epidemics are ignored. Then, we extend our proposed model by introducing three types of heterogeneity.

Firstly, we introduce the heterogeneity of individual influences in the information layer. In the information layer, different individuals may have different influences on others. The rate of individuals’ perceived local awareness depends on the state and influence of their neighbors. We assume that $A_i^\text{perc}$ and $w_{ij}$ represents the perceived local awareness ratio of individual $i$ [30] and the information reception rate after mutual sharing of information between individuals $i$ and $j$, respectively. Then, $A_i^\text{perc}$ can be described as follows:

$$A_i^\text{perc} = \sum_{j \in \Gamma_i} w_{ij} \cdot A_j$$  \hspace{1cm} (1)

In Eq. (1), $\Gamma_i$ represents the set of neighbors of individual $i$ in the information layer and $A_j$ denotes whether individual $j$ becomes aware. If individual $j$ becomes aware, $A_j = 1$. Otherwise, $A_j = 0$.

Considering that neighbors with different node degrees have different influences on individual $i$, we define $T_{ij}$ as

$$T_{ij} = \frac{k^\alpha_j}{\sum_{s \in \Gamma_i} k^\alpha_s}$$  \hspace{1cm} (2)

where $T_{ij}$ denotes the influences of individual $j$ on individual $i$. $k_j$ represents the node degree of individual $j$ in the information layer. $\alpha$ controls the heterogeneity of individual influences in the information layer, which $\alpha$ takes an arbitrary constant.

In fact, in the process of information diffusion, the information transmission and reception processes of each individual are independent of each other, and individuals have different preferences for information received from different individuals. These factors jointly determine the level of individual understanding of information. Thus, we define $w_{ij}$ as

$$w_{ij} = \pi T_{ij} + (1 - \pi)T_{ji}$$  \hspace{1cm} (3)

where $\pi$ represents the receptive preference of individual $i$ for the information shared by individual $j$, $1 - \pi$ denotes the receptive preference of individual $j$ for the information shared by individual $i$; $\pi \in [0, 1]$. When $\pi = 0$, $w_{ij}$ is determined only by $T_{ji}$. When $\pi = 1$, $w_{ij}$ is determined only by $T_{ij}$.

Secondly, we introduce the heterogeneity of individual influences in the epidemic layer. Due to their geographic location and exposure environment, individuals have different perceptions of epidemic severity [30] in the epidemic layer. Let $B_j^\text{perc}$ denotes the perceived epidemic severity of individual $j$, $w_{ij}$ represents the influence of individual $j$ on individual $i$ in the epidemic layer. $B_i^\text{perc}$ can be described as follows:

$$B_i^\text{perc} = \sum_{j \in \Gamma'_i} w'_{ij} \cdot B_j$$  \hspace{1cm} (4)

where $\Gamma'_i$ represents the set of neighbors of individual $i$ in the epidemic layer and $B_j$ denotes whether individual $j$ becomes infected. If individual $j$ becomes infected, $B_j = 1$. Otherwise, $B_j = 0$.

Considering that individuals with different node degrees have different influences on individual $i$, we define $w'_{ij}$ as

$$w'_{ij} = \sum_{s \in \Gamma'_i} k''_{js}$$  \hspace{1cm} (5)

In Eq. (5), $k''_{js}$ represents the node degree of individual $j$ in the epidemic layer and $\eta$ controls the heterogeneity of individual influences in the epidemic layer, which $\eta$ takes an arbitrary constant.

Finally, we introduce the heterogeneity of individual behavioral responses to epidemics. Apparently, if a unaware susceptible individual $i$ becomes aware in the information layer, they will take self-protective measures and reduce their infectivity $\beta$. In fact, when individuals have a high perceived local awareness ratio and a high perceived epidemic severity, individuals will become more cautious and take more effective self-protective measures. We assume that self-protective measures taken by individuals are mainly determined by their perceived local awareness ratio and their perceived epidemic severity. Thus, $\beta_i^A$ can be described as

$$\beta_i^A = \gamma_i \beta = \beta(1 - \lambda)k^\eta(\tau A_i^\text{perc} + (1 - \tau)B_i^\text{perc})$$  \hspace{1cm} (6)

where $\tau$ denotes the weight of individuals’ perceived local awareness ratio, and $1 - \tau$ represents the weight of individuals’ perceived epidemic severity, $\tau \in [0, 1]$. When $\tau = 0$, $\beta_i^A$ is determined only by individuals’ perceived epidemic severity. When $\tau = 1$, $\beta_i^A$ is determined only by individuals’ perceived local awareness ratio. The parameter $\lambda$ represents the strength of individuals’ responses to epidemics. In particular, $\gamma_i$ equals 0 for $\lambda = 1$, representing total immunization, and $\gamma_i$ equals 1 for $\lambda = 0$, representing self-protective measures taken are not effective for epidemics.
3 Analytical results based on MMCA

According to our proposed model, the \( N \) nodes in the multiplex model can be in the following states: unawareness-susceptible (US), awareness-susceptible (AS), awareness-infected (AI). For ease of analysis, we assume that both awareness diffusion and epidemic transmission are Markovian processes \([7, 9, 11, 14, 15, 17, 18, 22, 24–26, 30]\). We assume that the probabilities that individual \( i \) being in the above three states at time \( t \) as \( p_{US}^{i}(t) \), \( p_{AS}^{i}(t) \), \( p_{AI}^{i}(t) \). Evidently, each time step is satisfied

\[
p_{US}^{i}(t) + p_{AS}^{i}(t) + p_{AI}^{i}(t) = 1.
\]

Let \( A = (a_{ij})_{N \times N} \) and \( B = (b_{ij})_{N \times N} \) be the adjacency matrices of the information layer network and the epidemic layer network, respectively. Furthermore, in the information layer, the probability that individual \( i \) not being informed of epidemic information by any neighbors is defined as \( r_{i}(t) \), and the probability that individual \( i \) not being informed of epidemic information by mass media is defined as \( m_{i}(t) \). In the epidemic layer, the probabilities that an aware or unaware susceptible individual has not been infected is defined as \( q_{A}^{i}(t) \) or \( q_{U}^{i}(t) \). Thus, \( r_{i}(t), m_{i}(t), q_{A}^{i}(t) \) and \( q_{U}^{i}(t) \) can be described as follows:

\[
\begin{align*}
    r_{i}(t) &= H[\theta_{1} - A_{perc}^{i}] \\
    m_{i}(t) &= H[\theta_{2} - \sum_{j=1}^{N} p_{j}^{AS}(t)] \\
    q_{A}^{i}(t) &= \prod_{j} \left[ 1 - b_{ji} p_{j}^{AI}(t) \gamma_{i} \beta_{i} \right] \\
    q_{U}^{i}(t) &= \prod_{j} \left[ 1 - b_{ji} p_{j}^{AI}(t) \right]
\end{align*}
\]

where \( \theta_{1} \) and \( \theta_{2} \) denotes the critical local awareness ratio and the critical global awareness ratio, respectively. \( H(x) \) is a Heaviside step function, \( H(x) \) equals 1 for \( x > 0 \), which represents the unaware individual will continue to be unaware; \( H(x) \) equals 0 for \( x \leq 0 \), which represents the unaware individuals will be aware by their neighbors or mass media.

Then, we construct a transition probability tree, as shown in Fig. 2. In addition, the perceived local awareness of individual \( i \) and the perceived epidemic severity of individual \( i \) are defined as \( A_{perc}^{i} = \sum_{j} a_{ij} w_{ij} p_{j}^{A}(t) \) and \( B_{perc}^{i} = \sum_{j} b_{ij} w'_{ij} p_{j}^{A}(t) \), respectively. where \( p_{j}^{A}(t) = p_{j}^{AS}(t) + p_{j}^{AI}(t) \).

According to the microscopic Markov chain approach (MMCA), we obtain the following equation of dynamic evolution:

**Fig. 2** The transition probability tree of the three states in the model. \( r_{i}(t) \) and \( m_{i}(t) \) denotes the probability that individuals \( i \) not being informed of the epidemic information by any neighbors and mass media, respectively. \( \delta \) represents the probability that awareness individuals forget the epidemic information. \( q_{A}^{i}(t) \) and \( q_{U}^{i}(t) \) denotes the probabilities that an aware susceptible individual and an unaware susceptible individual have not been infected, respectively. \( \mu \) represents the probability that an infected individual recovers to be susceptible.
\begin{align*}
    p_{i}^{US}(t+1) &= p_{i}^{US}(t)r_{i}(t)m_{i}(t)q_{U}^{i}(t) \\
    &\quad + p_{i}^{AS}(t)\delta m_{i}(t)q_{U}^{i}(t) \\
    &\quad + p_{i}^{AI}(t)\delta m_{i}(t)\mu \\
    p_{i}^{AS}(t+1) &= p_{i}^{US}(t)[r_{i}(t)[1-m_{i}(t)] + [1-r_{i}(t)]]q_{A}^{i}(t) \\
    &\quad + p_{i}^{AS}(t)[\delta[1-m_{i}(t)] + (1-\delta)]q_{A}^{i}(t) \\
    &\quad + p_{i}^{AI}(t)[\delta[1-m_{i}(t)] + (1-\delta)]q_{A}^{i}(t) \\
    &\quad + p_{i}^{AI}(t)(1-\mu) \\
    p_{i}^{AI}(t+1) &= p_{i}^{US}(t)r_{i}(t)m_{i}(t)[1-q_{U}^{i}(t)] \\
    &\quad + p_{i}^{US}(t)[r_{i}(t)[1-m_{i}(t)] + [1-r_{i}(t)]][1-q_{A}^{i}(t)] \\
    &\quad + p_{i}^{AS}(t)[\delta[1-m_{i}(t)] + (1-\delta)][1-q_{A}^{i}(t)] \\
    &\quad + p_{i}^{AI}(t)(1-\mu)
\end{align*}

(11)

When the time step is large enough, the proportions of states for each node reach a steady state. Thus, when \( t \to +\infty \), we obtain:

\begin{align*}
    p_{i}^{US}(t+1)_{t\to+\infty} &= p_{i}^{US}(t)_{t\to+\infty} = p_{i}^{US} \\
    p_{i}^{AS}(t+1)_{t\to+\infty} &= p_{i}^{AS}(t)_{t\to+\infty} = p_{i}^{AS} \\
    p_{i}^{AI}(t+1)_{t\to+\infty} &= p_{i}^{AI}(t)_{t\to+\infty} = p_{i}^{AI}
\end{align*}

(12)

Near the onset of epidemics, the probability of nodes to be infected is close to zero. Thus, we assume that \( p_{i}^{AI} = 0, \delta_{i} > 0 \), and Eqs. (9) and (10) are approximated as:

\begin{align*}
    q_{i}^{A} &= \prod_{j}[1 - b_{ij}p_{i}^{AI}(t)\gamma_{i}^{j}\beta_{i}] \approx 1 - \gamma_{i}^{j}\beta_{i} \sum_{j} b_{ij}\delta_{i} \\
    q_{i}^{U} &= \prod_{j}[1 - b_{ij}p_{i}^{AI}(t)\beta_{i}] \approx 1 - \beta_{i} \sum_{j} b_{ij}\delta_{i} \\
\end{align*}

(13)

\( \gamma_{i}^{j} \) denotes the element of the identity matrix. Let \( \Phi_{ij} = [1 - (1-\gamma_{i})p_{i}^{A}]b_{ji} \) be the element of matrix \( \Phi \). Then, Eq. (21) can be rewritten as

\[ \Phi E = \frac{\mu}{\beta} E \]

(22)

where \( E \) represents the identity matrix, \( E = (\zeta_{1}, \zeta_{2}, ..., \zeta_{N})^{T} \). \( \zeta_{i} \) denotes the unit vector \( (i = 1, 2, ..., N) \). Defining the nontrivial solutions of Eq. (22) are eigenvectors of \( \Phi \), whose largest real eigenvalues are equal to \( \frac{\mu}{\beta} \). Therefore, the epidemic threshold \( \beta_{c} \) is to satisfy \( \frac{\mu}{\beta} = \Lambda_{\text{max}}(\Phi) \), where \( \Lambda_{\text{max}}(\Phi) \) denotes that the maximum eigenvalue of the matrix \( \Phi \). Then, the epidemic threshold can be described as

\[ \beta_{c} = \frac{\mu}{\Lambda_{\text{max}}(\Phi)} \]

(23)

Based on Eqs. (22) and (23), the epidemic threshold \( \beta_{c} \) depends on the infection attenuation factor \( \gamma \) and the density of aware individuals in the information layer \( p_{i}^{A} \), where \( p_{i}^{A} \) is further determined by the network structure, the recovery rate \( \mu \) and the forgetting rate \( \delta \).
4 Numerical simulation

According to Eq. (11), given the initial conditions, we can obtain the probabilities of each individual being in a different state at any time by iterations. The stationary fraction of aware individuals and infected individuals can be described as \( \rho^A = \frac{\sum I_i^A}{N} + \frac{\sum I_i^I}{N} \) and \( \rho^I = \frac{\sum I_i^I}{N} \), respectively.

Considering the heterogeneity of individual influences on others depends on the degree of node, we construct a two-layer BA scale-free network [33] with no correlation between layers. There are totally \( N = 1000 \) nodes in each layer network, and the average degree of BA networks is set as \( \langle k \rangle = 5.98 \). The BA scale-free network of each layer starts from 3 connected nodes, and the number of edges linking newly added nodes to the existing nodes is set to be 3. The initial infection fraction \( I_0 = 0.1 \).

Firstly, we use Monte Carlo (MC) simulations to test the accuracy of MMCA. As shown in Fig. 3, the results of MMCA and MC simulations for \( \rho^I \) and \( \rho^A \) are in agreement, which demonstrates the accuracy of MMCA in solving the coupling dynamics proposed in this paper. At the same time, we find that the size of N is independent of the analysis results and simulation results after extensive simulations. Then, we use MMCA to analyze the effects of different parameters on \( \rho^I \).

In Fig. 4, we mainly consider all individuals are informed of the epidemic-related information. As shown in Fig. 4a, \( \rho^I \) decreases as \( \lambda \) increases for the same sets of \( \alpha \) and \( \eta \). Therefore, improving the strength of the individuals’ responses to epidemics can effectively reduce the final density of infected individuals and suppress the outbreak of an epidemic. In addition, we find that decreasing \( \alpha \) and \( \eta \) can reduce the final density of infected individuals, and suppress the epidemic transmission. Noticed that reducing the final density of infected individuals \( \rho^I \) by the parameter \( \eta \) has a greater effect than decreasing \( \rho^I \) by the parameter \( \alpha \). In reality, epidemics can only spread in the physical contact layer, but not in the information transmission layer. However, the information dissemination can warn people to take appropriate self-protective measures by spreading the epidemic-related information, thereby suppressing the spread of diseases. Furthermore, in Fig. 4b and c, we observe that as \( \pi \) or \( \tau \) increases, \( \rho^I \) decreases. This indicates that in the process of epidemic transmission, the more individuals can accept information shared by others, the more beneficial to suppress the outbreak of a disease. Meanwhile, it also emphasizes that the suppressive impact of awareness transmission on epidemic spreading.

Our proposed model show that the critical local awareness ratio \( \theta_1 \) and the critical global awareness ratio \( \theta_2 \) controls the awareness threshold. When only the local or global awareness is regarded, we investigate the impacts of the critical local awareness ratio \( \theta_1 \) and the critical global awareness ratio \( \theta_2 \) on the epidemic transmission and the awareness diffusion, respectively. Thus, we plot the four related phase diagrams, as shown in Fig. 5.

In Fig. 5, we find the thresholds for \( \theta_1 \) and \( \theta_2 \), respectively. If \( \theta_1 \) or \( \theta_2 \) is less than its threshold, both \( \rho^A \) and \( \rho^I \) have an abrupt transition, i.e., \( \rho^A \) abruptly increase and \( \rho^I \) suddenly decrease. In addition, we also find that the threshold for \( \theta_2 \) increases as the infection rate \( \beta \) enhances. The results show that improving the awareness diffusion among individuals and the strength of publicity for epidemic by mass media are effective ways to suppress the epidemic transmission.

Furthermore, we examine the impacts of the critical local and global awareness on the epidemic threshold, respectively. In Fig. 6, we find that both \( \theta_1 \) and \( \theta_2 \) have two-stage effects on the epidemic threshold \( \beta_c \). Appar-
Fig. 4  
(a) $\rho$ as a function of $\beta$ for different values of $\alpha$, $\eta$ and $\lambda$, where $\pi = 0.5$, $\tau = 0.2$. 
(b) $\rho$ as a function of $\beta$ for different values of $\pi$, where $\alpha = 2$, $\eta = 2$, $\lambda = 0.5$, $\tau = 0.2$. 
(c) $\rho$ as a function of $\beta$ for different values of $\tau$, where $\alpha = 2$, $\eta = 2$, $\lambda = 0.5$, $\pi = 0.5$. The remaining parameters are $\theta_1 = 0.1$, $\theta_2 = 0.1$, $\mu = 0.8$, $\delta = 0.6$.

ently, the effect of $\theta_2$ on $\beta_c$ is larger than that of $\theta_1$ on $\beta_c$. Besides, as the parameter $\alpha$ reduces, the value of $\theta_1$ which make $\beta_c$ undergo an abrupt transition decreases. In addition, the value of a low phase of the epidemic threshold (i.e., the smaller $\beta_c$ in Fig. 6) is independent of $\alpha$ and $\eta$, which is similar with previous studies [30]. Compared Fig. 6b with Fig. 8c, the value of a high phase of the epidemic threshold is related to the value of $\beta_c$ for $\alpha$ when only $\theta_2$ is considered. That is, the larger value of $\beta_c$ for $\alpha$, the greater value of the high phase of the epidemic threshold. Noticed that the value of $\beta_c$ for $\alpha$ is close to its maximum when $\alpha = -0.6939$.

In Fig. 6, we only consider the impact of the critical local awareness $\theta_1$ (or the critical global awareness $\theta_2$) on the epidemic threshold $\beta_c$. In order to explore the impacts of different $\theta_1$ and $\theta_2$ on the epidemic threshold $\beta_c$, we utilize MC simulation and MMCA to derive the full phase diagrams $(\theta_1 - \theta_2)$ of $\beta_c$, respectively.

As shown in Fig. 7, we observe a discrepancy between Fig. 7a and b. In the analytical results of MMCA, the threshold of abrupt transition of $\beta_c$ for $\theta_1$ is smaller than that shown in the results of MC simulations. Note that Eqs. (7)–(8) are obtained based on the assumption of an absence of dynamical cor-
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Fig. 5 a–b The full phase diagrams \((\theta_1 - \beta)\) of \(\rho^A\) and \(\rho^I\), respectively, where \(\theta_2 = 1\). c–d The full phase diagrams \((\theta_2 - \beta)\) of \(\rho^A\) and \(\rho^I\), respectively, where \(\theta_1 = 1\). The four phase diagrams are obtained by averaging 50 MC simulations for each point in the grid 100 × 100. The remaining parameters are \(\mu = 0.8\), \(\delta = 0.6\), \(\alpha = 2\), \(\eta = 2\), \(\lambda = 0.5\), \(\tau = 0.2\), \(\pi = 0.5\).

Then, we explore the impacts of the heterogeneity of individual influences in the information layer and the heterogeneity of individual influences in the epidemic layer on epidemic transmission. In Fig. 8, the results of MMCA and MC simulations are consistent. Meanwhile, the result shows that \(\alpha\) takes effect on \(\beta_c\) when \(\theta_1\) or \(\theta_2\) needs to be less than the threshold which makes \(\beta_c\) undergo an abrupt transition at a certain \(\theta_1\) or \(\theta_2\). In addition, the parameter \(\alpha\) has a nonlinear influence on \(\beta_c\). Nevertheless, we find that the parameter \(\eta\) has few influence on \(\beta_c\). For simplicity, we consider that...
Fig. 6  a $\beta_c$ as a function of $\theta_1$ for different values of $\alpha$ and $\eta$, where $\theta_2 = 1$. b $\beta_c$ as a function of $\theta_2$ for different values of $\alpha$ and $\eta$, where $\theta_1 = 1$. The two diagrams are obtained by averaging 100 MC simulations. The remaining parameters are $\mu = 0.8$, $\delta = 0.6$, $\lambda = 0.5$, $\tau = 0.2$, $\pi = 0.5$

Fig. 7  a The full phase diagrams $(\theta_1 - \theta_2)$ of $\beta_c$ derived from MC simulations. This phase diagram is obtained by averaging 50 MC simulations for each point in the grid $50 \times 50$. b The full phase diagrams $(\theta_1 - \theta_2)$ of $\beta_c$ derived from MMCA. The remaining parameters are $\mu = 0.8$, $\delta = 0.6$, $\alpha = 2$, $\eta = 2$, $\lambda = 0.5$, $\tau = 0.2$, $\pi = 0.5$

most individuals are informed in Fig. 8c. Thus, we set $\theta_1 = 0.1$, $\theta_2 = 0.1$. Note that $\beta_c$ increases as $\lambda$ raises. To summarize, improving the strength of an individuals’ responses to epidemics and moderately adjusting the heterogeneity of individual influences in the information layer can enhance the epidemic threshold.

To further explore the effects of $\alpha$ and $\eta$ on epidemic spreading, we investigate the impacts of $\alpha$ and $\eta$ on the final density of infected individuals $\rho^I$. Hence, we plot the two phase diagrams as the shown in Fig. 9.

Compared Fig. 8 with Fig. 9, the variation of $\rho^I$ is related to the value of $\beta_c$ for $\alpha$, i.e., $\rho^I$ increases as $\beta$ increases when $\beta$ is larger than $\beta_c$ for a certain
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Fig. 8  

(a) $\beta_c$ as a function of $\alpha$ for different values of $\theta_1$ and $\theta_2$, where $\lambda = 0.5$, $\eta = 2$. 

(b) $\beta_c$ as a function of $\eta$ for different values of $\theta_1$ and $\theta_2$, where $\lambda = 0.5$, $\alpha = 2$. 

(c) $\beta_c$ as a function of $\alpha$ for different values of $\lambda$ and $\eta$, where $\theta_1 = 0.1$, $\theta_2 = 0.1$.

The three diagrams are obtained by averaging 50 MC simulations. The remaining parameters are $\mu = 0.8$, $\delta = 0.6$, $\tau = 0.2$, $\pi = 0.5$.

$\alpha$. At the same time, $\rho^I$ increases as the parameter $\eta$ enhances, i.e., $\eta$ can affect the final density of infected individuals $\rho^I$. In summary, these results demonstrate that the parameter $\alpha$ has influences on the size of $\beta_c$ and $\rho^I$. However, the parameter $\eta$ has few effects on $\beta_c$, but can affect the magnitude of $\rho^I$.

In addition, we find that the parameter $\lambda$ has a nonlinear influence on the epidemic threshold $\beta_c$ in Fig. 10. And then, $\beta_c$ enhances as the parameter $\lambda$ increases. Furthermore, we assume that $\lambda_c$ denotes the value of $\lambda$ when $\beta_c$ as the functional curves of $\lambda$ intersect for different values of $\alpha$. When $\lambda < \lambda_c$, the greater $\alpha$, the larger $\beta_c$; When $\lambda > \lambda_c$, the smaller $\alpha$, the larger $\beta_c$. The results demonstrate that the epidemic threshold can be increased by adjusting the value of $\alpha$ when the power to improve the strength of an individuals’ responses to epidemics is limited.
Fig. 9  a The full phase diagrams \((\alpha - \beta)\) of \(\rho^I\), where \(\eta = 2\).
b The full phase diagrams \((\eta - \beta)\) of \(\rho^I\), where \(\alpha = 2\). The two phase diagrams are obtained by averaging 50 MC simulations for each point in the grid 100 \times 100. The remaining parameters are \(\mu = 0.8, \delta = 0.6, \theta_1 = 0.1, \theta_2 = 0.1, \tau = 0.2, \pi = 0.5, \lambda = 0.5\)

Fig. 10  a \(\beta_c\) as a function of \(\lambda\) for different values of \(\alpha\) and \(\eta\), where \(\lambda_c\) represents the value of \(\lambda\) when \(\beta_c\) as the functional curves of \(\lambda\) intersect for different values of \(\alpha\). b \(\beta_c\) as a function of \(\lambda\) for different values of \(\alpha\), where \(\eta = -2\). The two diagrams are obtained by averaging 100 MC simulations. The remaining parameters are \(\mu = 0.8, \delta = 0.6, \theta_1 = 0.1, \theta_2 = 0.1, \tau = 0.2, \pi = 0.5\)

5 Conclusion

In this paper, to explore the interplay of awareness and epidemic, we propose a coupled awareness-epidemic spreading model considering the heterogeneity of individual influences. Moreover, the processes of local and global awareness diffusion are described by the threshold model in our proposed model. Being different from researches in the past, we take into account that the individuals’ receptive preference for information and the impacts of the perceived local awareness ratio of individuals and the perceived epidemic severity of individuals on self-protective behavior. The results show that the more individuals can accept information shared
by others, the more beneficial to suppress the epidemic transmission. In addition, only when $\theta_2$ or $\theta_1$ increases, $\beta_c$ will undergo a second-order phase transition, which is similar with the results of previous studies [30]. In our proposed model, the heterogeneity of individual influences in the epidemic layer has few effect on the epidemic threshold, but can affects the magnitude of the final density of infected individuals. Furthermore, we find that either the heterogeneity of individual influences in the information layer or the strength of individuals’ responses to epidemics has a nonlinear effect on the epidemic threshold. At the same time, when the power to improve the strength of an individuals’ responses to epidemics is limited, the epidemic threshold can be increased by adjusting the value of $\alpha$. In summary, our proposed model contributes to our understanding of the interaction between epidemic transmission and awareness diffusion, and reveals the important impact of the heterogeneity of individual influences on disease spreading. Nevertheless, there are still many deficiencies. In the future, the discussion of the effect of heterogeneities on epidemic spreading will continue to broaden. For example, the influence of the heterogeneity of individuals in temporal networks or the heterogeneity of different perspectives of individuals on epidemic transmission, and the impact of the heterogeneity of individuals in different network structures on epidemic spreading.

**Author contributions** All authors contributed to the study conception and design. Material preparation, data collection and analysis were performed by Haidong Xu, Ye Zhao and Dun Han. The first draft of the manuscript was written by Haidong Xu and all authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

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**Availability of data and material** All data and materials can be obtained under the proposed model and algorithms.

**Declarations**

**Competing interests** The authors have no relevant financial or non-financial interests to disclose.

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