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The outbreak of coronavirus disease 2019 (COVID-19) threatens the health and safety of all humanity. This disease has a prominent feature: the presymptomatic and asymptomatic viral carriers can spread the disease. It is crucial to estimate the impact of this undetected transmission on epidemic outbreaks. Currently, disease-related information has been widely disseminated by the mass media. To investigate the impact of both individuals and mass media information dissemination on the epidemic spreading, we establish a new UAU-SEIR (Unaware–Aware–Unaware–Susceptible–Exposed–Infected–Recovered) model with mass media on two-layer multiplex networks. In the model, E-state individuals denote asymptomatic infections, and a single node connecting to all individuals denotes the mass media. In this work, we use the Microscopic Markovian Chain Approach (MMCA) to derive the epidemic threshold. Comparing the MMCA theoretical results with Monte Carlo (MC) simulations, we find that the MMCA has a good consistency with MC simulations. In addition, we also analyze the impact of model parameters on epidemic spreading and epidemic threshold. The results show that reducing the proportion of asymptomatic infections, accelerating the dissemination of information between individuals and the dissemination of information via the mass media can effectively inhibit the epidemic spreading and raise the epidemic threshold.

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1. Introduction

With the continuous development of the information society, the information explosion has closely linked information dissemination with epidemic spreading. In the field of transmission dynamics on complex networks, the information dissemination and epidemic spreading on complex networks as two independent dynamic processes have been extensively studied [1–3]. We have noticed that the epidemic spreading on the contact network and information dissemination on the information network are closely related and affect each other. For example, the outbreak of COVID-19 has caused the spread of COVID-19-related information among the population, which can trigger the public’s behavioral responses to reduce the risk of infection (e.g., wearing masks, reducing party activities, washing hands, disinfecting frequently and so on), and in turn impact on the epidemic spreading process [4–8]. Therefore, the researches on the interplay between the information dissemination and epidemic spreading have become an important topic in the field of transmission dynamics.

In recent years, with the continuous improvement and development of multilayer networks theory, people have begun to use multilayer networks as a basic tool to quantitatively describe the interaction among network layers as well...
The diagram of two-layer multiplex networks used in the UAU-SEIR model with mass media. The upper layer is called the virtual communication layer to describe the process of information dissemination, and nodes have two possible states: unaware (U) and aware (A). The lower layer is called the physical contact layer to describe the process of epidemic spreading, and nodes have four possible states: susceptible (S), exposed (E), infected (I) and recovered (R). The top node represents the mass media, which is connected to all nodes and regularly releases disease-related information.

Fig. 1. The diagram of two-layer multiplex networks used in the UAU-SEIR model with mass media. The upper layer is called the virtual communication layer to describe the process of information dissemination, and nodes have two possible states: unaware (U) and aware (A). The lower layer is called the physical contact layer to describe the process of epidemic spreading, and nodes have four possible states: susceptible (S), exposed (E), infected (I) and recovered (R). The top node represents the mass media, which is connected to all nodes and regularly releases disease-related information.

as between these constituents [6,9–11]. Among them, the multiplex networks is an interesting example of multilayer network setup [12,13], in which the nodes represent the same entities in all layers, but the link relationship among nodes may be completely different [14]. Naturally, many scholars have begun to use the multiplex networks as the underlying framework to study the dynamical interplay of information dissemination and epidemic spreading [7,8,14–26]. Granell et al. have proposed a UAU-SIS model to analyze a coupled dynamical process of awareness and infection on the multiplex networks, and discovered a meta-critical point where the onset of the epidemics can be controlled by the diffusion of awareness [15]. They have further extended the model to study the influence of self-awareness and mass media on dynamics, and found that the meta-critical point disappears due to the mass media [16]. Wang et al. have established a UAU-SIR model to investigate the multiple impacts between awareness diffusion and epidemic spreading, and found that the epidemic threshold has relation with the awareness diffusion and the topology of epidemic networks [14]. Shi et al. have proposed the UAU-SEIS model to analyze the dynamical interplay between behavior and epidemic spreading in multiplex networks, and considered the impact of individual heterogeneity on the disease transmission dynamics [8].

However, for many infectious diseases, such as SARS, influenza A (H1N1) and COVID-19, a prominent feature among them is the presence of a large number of asymptomatic infections. The infected individuals will not show obvious clinical symptoms during a period of time, but they can infect others. Based on the above characteristics, the SEIR model describes these diseases more appropriately than SIS and SIR [27–36]. On the other hand, with the rapid development of the information society, information dissemination has been playing a non-negligible impact on the epidemic spreading. We have also noticed that information can not only be disseminated between individuals but also the disease-related information will be widely published in the mass media (TV, radio, newspapers, etc.). Therefore, based on previous works, we introduce mass media into the multiplex networks framework to study the dynamic interaction between information dissemination and SEIR-based epidemic spreading. In this work, we will focus on the impact of asymptomatic infections and the mass media on epidemic spreading.

The paper is organized as follows. In Section 2, the UAU-SEIR model with mass media on multiplex networks is described in detail. In Section 3, we apply the Microscopic Markov Chain Approach (MMCA) to perform theoretical analysis on the model and calculate the epidemic threshold. In Section 4, we verify the accuracy of the theory through extensive numerical simulations, and analyze the impact of various parameters on the epidemic spreading and the epidemic threshold. In Section 5, the conclusions and discussions are presented.

2. The UAU-SEIR Model with mass media

Based on the works of Granell et al., we use the setup of two-layer multiplex networks as the modeling framework for studying the dynamic interaction between information dissemination and SEIR-based epidemic spreading. In this two-layer multiplex networks, every node on one layer is individually mapped to the corresponding node on another layer (i.e., two-layer networks have the same nodes), and the same pair of nodes have links in one layer but may not exist in another layer (i.e., two-layer networks have different connectivity). For the sake of simplicity, we assume that the two-layer networks are both unweighted and undirected. As shown in Fig. 1, the upper layer represents the virtual
Fig. 2. Information dissemination transition diagram (left panel) and epidemic spreading transition diagram (right panel) under the UAU-SEIR model with mass media. For the information dissemination, the U-state individuals become A-state individuals by being told the disease-related information, and there are three sources of information: spread among individuals with probability $\lambda$, spread by mass media with probability $m$, and I-state individuals automatically become A-state individuals. The A-state individuals return to U-state with probability $\delta$. For epidemic spreading, the unaware (aware) S-state individuals become E-state with probability $\beta^U = \beta$ ($\beta^A = \gamma \beta$) after being infected, and the E-state individuals enter I-state with probability $\sigma$, and the I-state individuals return to R-state with probability $\mu$. Communication network used for information dissemination, called the virtual communication layer. The lower layer represents the physical contact network used for epidemic spreading, called the physical contact layer.

In the virtual communication layer, we describe the process of information dissemination via the UAU (Unaware–Aware–Unaware) model. U-state (Unaware state) individuals do not know disease-related information and they will not react to prevent the disease, while A-state (Aware state) individuals know disease-related information and will take preventive measures to reduce the risk of infection. U-state individuals will be informed of disease-related information by their neighbors in A-state, and become the A-state with a probability $\lambda$. Due to the periodicity of the epidemic spreading, A-state individuals will turn back to U-state with a probability $\delta$. The above-mentioned model is restricted to the dissemination of information between individuals, but the impact of mass media on information dissemination is crucial. Considering a more realistic situation, we introduce mass media into our model. As described in Fig. 1, we add a single node linked with all other nodes in the virtual communication layer, that will regularly report disease-related information and each individual will be informed with probability $m$.

In the physical contact layer, we describe the process of epidemic spreading via the SEIR (Susceptible–Exposed–Infected–Recovered) model. Here, we divide the infected individuals into E-state (Exposed state) and I-state (Infected state). Among them, E-state means infected individuals without obvious clinical symptoms, and I-state means infected individuals with obvious clinical symptoms. We assume that both E-state and I-state individuals can infect S-state (Susceptible state) individuals. S-state individuals are infected by neighbors in E-state and I-state and first enter E-state with a probability $\beta$, and then E-state individuals enter I-state with a probability $\sigma$, and finally I-state individuals enter R-state (Recovered state) with a probability $\mu$. Here, we assume that the R-state individuals have produced antibodies and will no longer be infected.

Information dissemination and epidemic spreading are two competitive spread processes: the spread of epidemic will promote the dissemination of information, and the dissemination of disease-related information will inhibit the spread of epidemic. Next, we model the interaction between both processes. On the one hand, we assumed that the symptomatic individuals would immediately become aware [15]. Therefore, I-state nodes in the physical contact layer will automatically become A-state in the virtual communication layer and spread disease-related information. On the other hand, A-state nodes in the virtual communication layer will take measures for preventing infection. Therefore, nodes in different states in the virtual communication layer have different possibilities of being infected in the physical contact layer. To this end, we introduce the infection attenuation factor $\gamma$ ($0 \leq \gamma \leq 1$) to adjust the possibility of nodes being infected. Here, the infection rates of the U-state node and the A-state node are denoted as $\beta^U = \beta$ and $\beta^A = \gamma \beta^U = \gamma \beta$, respectively. In Fig. 2, we show the transition diagram for the information dissemination and the epidemic spreading under the UAU-SEIR model with mass media.

In this model, each individual have seven different states: unaware and susceptible (US), unaware and exposed (UE), unaware and recovered (UR), aware and susceptible (AS), aware and exposed (AE), aware and infected (AI), aware and recovered (AR). We do not consider the unaware and infected (UI) state because we assume that I-state individuals will automatically become A-state.

3. MMCA theoretical analysis

In the study of epidemic transmission dynamics, the epidemic threshold of infectious diseases is of great significance to the assessment of epidemics, early warning, and choice of intervention strategies. In the past ten years, many scholars have proposed numerous methods for calculating epidemic thresholds [26,37,38]. Among them, the Microscopic Markov Chain Approach (MMCA) proposed by Gomez et al. has been widely used to solve the model of the interplay between information dissemination and epidemic spreading in multiplex networks [39,40]. This method is of high accuracy for solving the transmission dynamics and can display the infection information of each individual. Therefore, we use MMCA to conduct a theoretical analysis of the proposed model and calculate the epidemic threshold.
3.1. MMCA equations

We define \( A = (a_{ij}) \) and \( B = (b_{ij}) \) as the adjacency matrix of the virtual communication layer and the physical contact layer, where the elements \( a_{ij} = 1 \) and \( b_{ij} = 1 \) if there is a link between node \( i \) and node \( j \), else \( a_{ij} = 0 \) and \( b_{ij} = 0 \). At time \( t \), each individual \( i \) can be in one of the seven states mentioned in Section 2 with a certain probability, denoted by \( p_{i}^{US}(t) \), \( p_{i}^{UE}(t) \), \( p_{i}^{AE}(t) \), \( p_{i}^{AS}(t) \), \( p_{i}^{AE}(t) \), \( p_{i}^{REP}(t) \), and \( p_{i}^{AR}(t) \), respectively. In the virtual communication layer, we use \( r_{i}(t) \) to represent the probability that the U-state individual \( i \) is not informed by any neighbors. In the physical contact layer, we use \( q_{i}^{U}(t) \) and \( q_{i}^{I}(t) \) to denote the probability that the unaware and aware S-state individual \( i \) is not infected by any neighbors, respectively. Based on the above definitions, \( r_{i}(t) \), \( q_{i}^{U}(t) \) and \( q_{i}^{I}(t) \) can be expressed as follows:

\[
\begin{align*}
    r_{i}(t) &= \prod_{j} \left[ 1 - a_{ij} p_{j}^{U}(t) \lambda_{i} \right], \\
    q_{i}^{U}(t) &= \prod_{j} \left[ 1 - b_{ij} \left( p_{j}^{UE}(t) + p_{j}^{AE}(t) \right) \right] \beta_{i}^{U}, \\
    q_{i}^{I}(t) &= \prod_{j} \left[ 1 - b_{ij} \left( p_{j}^{UE}(t) + p_{j}^{AE}(t) + p_{j}^{AI}(t) \right) \right] \beta_{i}^{A},
\end{align*}
\]

where \( p_{i}^{Ai}(t) = p_{i}^{AS}(t) + p_{i}^{AE}(t) + p_{i}^{AR}(t) \).

Considering that each time step is divided into three continuous processes: information dissemination between individuals (UAU process), information dissemination by mass media and epidemic spreading (SEIR process), we build the Markov state transition trees to represent the state transition between seven possible states shown in Fig. 3. According to Eqs. \((1)-(3)\) and the Markov model for each state is developed as follows:

\[
\begin{align*}
    p_{i}^{US}(t + 1) &= p_{i}^{US}(t) r_{i}(t)(1 - m) q_{i}^{U}(t) + p_{i}^{SU}(t) \delta [1 - m] q_{i}^{I}(t) \\
    &= p_{i}^{US}(t) r_{i}(t) + p_{i}^{SU}(t) \delta [1 - m] q_{i}^{I}(t), \\
    p_{i}^{AE}(t + 1) &= p_{i}^{AE}(t) \left[ r_{i}(t) m q_{i}^{A}(t) + (1 - r_{i}(t)) q_{i}^{I}(t) \right] + p_{i}^{AS}(t) \left[ \delta m q_{i}^{A}(t) + (1 - \delta) q_{i}^{I}(t) \right] \\
    &= p_{i}^{AS}(t) r_{i}(t) m + 1 - r_{i}(t) + p_{i}^{AS}(t) \delta m + 1 - \delta \right] q_{i}^{I}(t), \\
    p_{i}^{UE}(t + 1) &= p_{i}^{UE}(t) \left[ r_{i}(t) m [1 - q_{i}^{U}(t)] + (1 - r_{i}(t)) [1 - q_{i}^{I}(t)] \right] + p_{i}^{SU}(t) \left[ \delta m [1 - q_{i}^{U}(t)] + (1 - \delta) [1 - q_{i}^{I}(t)] \right] \\
    &= p_{i}^{SU}(t) \left[ r_{i}(t) m + 1 - r_{i}(t) + p_{i}^{SU}(t) \delta m + 1 - \delta \right] [1 - q_{i}^{I}(t)] \\
    + p_{i}^{SE}(t) \left[ r_{i}(t) m + 1 - r_{i}(t) + p_{i}^{SE}(t) \delta m + 1 - \delta \right] (1 - \sigma), \\
    p_{i}^{AI}(t + 1) &= p_{i}^{AI}(t) \sigma + p_{i}^{AE}(t) \sigma + p_{i}^{AR}(t) \left( 1 - \mu \right), \\
    p_{i}^{UR}(t + 1) &= p_{i}^{UR}(t) \delta (1 - m) \mu + p_{i}^{UR}(t) r_{i}(t) (1 - m) + p_{i}^{UR}(t) \delta (1 - m) \\
    &= p_{i}^{US}(t) \delta \mu + p_{i}^{UR}(t) r_{i}(t) + p_{i}^{UR}(t) \delta (1 - m), \\
    p_{i}^{AR}(t + 1) &= p_{i}^{AR}(t) \left[ \delta m \mu + (1 - \delta) \mu \right] + p_{i}^{UR}(t) [r_{i}(t) m + 1 - r_{i}(t)] + p_{i}^{AR}(t) \left[ \delta m + 1 - \delta \right],
\end{align*}
\]

where \( t \) and \( t + 1 \) represent the state evolution of individual \( i \) from the current time step to the next time step. In addition, the following normalization condition:

\[
p_{i}^{US}(t) + p_{i}^{AS}(t) + p_{i}^{UE}(t) + p_{i}^{AE}(t) + p_{i}^{AR}(t) + p_{i}^{UR}(t) + p_{i}^{AI}(t) = 1,
\]

holds for each time step.

3.2. Epidemic threshold

In this part, we use stationarity and the MMCA equation derived from Markov state transition trees to calculate the epidemic threshold \( \beta^{c} \).

When the system arrives at the stationary state, \( p_{i}(t + 1) = p_{i}(t) = p_{i} \) holds for all the nodes and possible states. Near the epidemic threshold, the probability of individuals to be infected approaches 0. It is assumed that the S-state individuals first enter the E-state after being infected, we set \( p_{i}^{E} = \epsilon_{i} \ll 1 \). In the stationary state, Eq. \((8)\) can be simplified as \( p_{i}^{U} = \frac{\sigma}{\mu} p_{i}^{U} \approx \frac{\sigma}{\mu} p_{i} \), where \( p_{i}^{E} = p_{i}^{UE} + p_{i}^{AE} \). Accordingly, Eqs. \((2)\) and \((3)\) are approximated as:

\[
\begin{align*}
    q_{i}^{I} &\approx 1 - (1 + \frac{\sigma}{\mu}) \beta_{i}^{U} \sum_{j} b_{ij} e_{ij} = 1 - (1 + \frac{\sigma}{\mu}) \eta_{i},
\end{align*}
\]
Fig. 3. The Markov state transition trees of seven possible states including US, AS, UE, AE, AI, UR and AR. Each time step is divided into three continuous processes: information dissemination between individuals (UAI process), information dissemination by mass media and epidemic spreading (SEIR process).

\[ q^U_i \approx 1 - \gamma (1 + \sigma/\mu) \beta^U \sum_j b_{ji} \epsilon_j = 1 - \gamma (1 + \sigma/\mu) \eta_i, \]  \hspace{1cm} (13)

where

\[ \eta_i = \beta^U \sum_j b_{ji} \epsilon_j. \]  \hspace{1cm} (14)
In the steady state, adding Eqs. (6) and (7), we have
\[ p_i^U = (1 - \sigma)p_i^U + p_i^{US} \left\{ r_i(1 - m)(1 - q_i^U) + [1 - (1 - m)r_i](1 - q_i^U) \right\} + p_i^{AS} \left\{ \delta(1 - m)(1 - q_i^U) + [1 - (1 - m)\delta](1 - q_i^U) \right\}. \]  
(15)

As a further step, substituting Eqs. (12) and (13) into Eq. (15), we have
\[ \epsilon_i = (1 - \sigma)\epsilon_i + p_i^{US} \left\{ r_i[1 - m](1 + \frac{\sigma}{\mu})\eta_i + [1 - (1 - m)r_i](1 + \frac{\sigma}{\mu})\gamma\eta_i \right\} + p_i^{AS} \left\{ \delta(1 - m)(1 + \frac{\sigma}{\mu})\eta_i + [1 - (1 - m)\delta](1 + \frac{\sigma}{\mu})\gamma\eta_i \right\}. \]  
(16)

Around the epidemic threshold, \( p_i^{US} \to 0, p_i^{AE} \to 0, p_i^{AI} \to 0, p_i^{UR} \to 0 \) and \( p_i^{AR} \to 0 \). We can get \( p_i^U = p_i^{US} + p_i^{AE} + p_i^{AI} + p_i^{AR} \approx p_i^{AS} \) and \( p_i^A = p_i^{AS} + p_i^{AE} + p_i^{AI} + p_i^{AR} \approx p_i^{AS} \). Therefore, Eq. (16) can be written as
\[ \epsilon_i = (1 - \sigma)\epsilon_i + \left\{ p_i^U r_i[1 - m] + p_i^A[1 - (1 - m)\delta] \right\} (1 + \frac{\sigma}{\mu})\eta_i + \left\{ p_i^A [1 - (1 - m)r_i] + p_i^A [1 - (1 - m)\delta] \right\} (1 + \frac{\sigma}{\mu})\gamma\eta_i. \]  
(17)

In the steady state, removing \( O(\epsilon_i) \) terms of Eqs. (4) and (5), we can get
\[ p_i^U = p_i^U r_i[1 - m] + p_i^A[1 - (1 - m)\delta]. \]  
(18)
\[ p_i^A = p_i^U[1 - (1 - m)r_i] + p_i^A[1 - (1 - m)\delta]. \]  
(19)

Substituting \( p_i^U + p_i^A = 1 \), Eqs. (18) and (19) into Eq. (17), we obtain
\[ \epsilon_i = (1 - \sigma)\epsilon_i + \left\{ p_i^U[1 - (1 - m)r_i] + p_i^A[1 - (1 - m)\delta] \right\} (1 + \frac{\sigma}{\mu})\eta_i + \left\{ p_i^A[1 - (1 - m)r_i] + p_i^A[1 - (1 - m)\delta] \right\} (1 + \frac{\sigma}{\mu})\gamma\eta_i. \]  
(20)

Considering Eq. (14), Eq. (20) can be further transformed into
\[ \sum_j \left\{ (p_j^U + \gamma p_j^A) b_{ji} - \frac{\mu\sigma}{\beta^U(\sigma + \mu)} \alpha_{ij} \right\} \epsilon_j = 0, \]  
(21)

where \( \alpha_{ij} \) are the elements of the identity matrix, i.e., \( \alpha_{ii} = 1 \) if \( i = j \), else \( \alpha_{ij} = 0 \). Defining matrix \( H \) with elements \( h_{ij} = (p_j^U + \gamma p_j^A) b_{ji} \), then Eq. (21) can be described in a matrix form
\[ He = \frac{\sigma\mu}{\beta^U(\sigma + \mu)} \epsilon, \]  
(22)

where \( \epsilon = (\epsilon_1, \epsilon_2, \ldots)^T \). Obviously, the eigenvalues of \( H \) are equal to \( \frac{\sigma\mu}{\beta^U(\sigma + \mu)} \). The epidemic threshold is the minimum value of \( \beta^U \) satisfying Eq. (22). Therefore, the epidemic threshold of the infectious diseases in the proposed model is given as
\[ \beta^U_c = \frac{\sigma\mu}{\Lambda_{\text{max}}(H)(\sigma + \mu)}, \]  
(23)

where \( \Lambda_{\text{max}}(H) \) is the largest eigenvalue of \( H \). Since \( p_j^U + p_j^A = 1 \), matrix \( H \) depends on \( p_j^A \). Combining Eqs. (18) and (19), we can get
\[ p_i^A = \left( 1 - p_i^A \right) [1 - (1 - m)r_i] + p_i^A [1 - (1 - m)\delta]. \]  
(24)
and
\[ r_i = \prod_j \left( 1 - a_{ij} p_j^A \lambda \right), \]  
(25)

which can also be iteratively resolved.

According to Eq. (23), we can clearly find that the epidemic threshold is related to the network topology and various model parameters. In the next part, we will conduct a large number of numerical simulations to verify the above theoretical analysis.

4. Numerical simulations

In this section, we will verify the accuracy of MMCA by carrying out extensive Monte Carlo (MC) simulations and analyze the impact of various parameters on the epidemic spreading and the epidemic threshold. The two-layer networks used by us are set up as follows. In the physical contact layer, we use the Barabási–Albert (BA) scale-free network [41]
with 1000 nodes, which starts with $M_0 = 10$ randomly connected nodes and adds $M = 5$ edges to the existing nodes for every new added node. In the virtual communication layer, we use the same network as the physical contact layer but with 400 extra random links (non-overlapping with previous). According to the above settings, we generate 50 pairs of BA networks (50 in the virtual communication layer and in the physical contact layer). All MMCA results are obtained by averaging over 50 BA networks, and all MC simulation results are obtained by averaging 1000 independent realizations (50 BA networks, 20 MC simulations for each network). For consistency, the networks are the same throughout the paper. Furthermore, the initial fraction of infected individuals (E-state) and aware individuals (A-state) are set as 2% and 0. All of the MC simulation results are obtained by averaging 1000 realizations. Other model parameters are set as follows: $\beta = 0.8$, $\sigma = 0.6$, $\mu = 0.5$, $\lambda = 0.5$, $\delta = 0.3$, $\gamma = 0$ and $m = 0$.

In the UAU-SEIR model, the fraction of A-state individuals and R-state individuals at steady state are the most critical indicator for the information dissemination and epidemic spreading, which are defined as $\rho^A$ and $\rho^R$. For the MC simulations, $\rho^A$ and $\rho^R$ can be expressed as $\rho^A = \frac{N_A}{N}$, $\rho^R = \frac{N_R}{N}$, where $N_A$ and $N_R$ represent the numbers of A-state individuals and R-state individuals, and $N$ represents the total numbers of individuals. For the MMCA, $\rho^A$ and $\rho^R$ can be expressed as $\rho^A = \frac{1}{M} \sum_{i=1}^{N} p_i^A$, $\rho^R = \frac{1}{M} \sum_{i=1}^{N} p_i^R$, where $p_i^A$ and $p_i^R$ represent the probability that node $i$ is in the A-state and the R-state. Moreover, $\rho^S$, $\rho^E$ and $\rho^I$ can be expressed similarly. We record the results of the last time step of MC simulation and MMCA, where the results of MMCA can be obtained by iteratively solving Eqs. (4)–(10).

Firstly, we give the fraction of individuals under different states as the time step $t$ varies by MC simulations. Fig. 4(a) depicts the fraction of U-state and A-state individuals as a function of the time step $t$ in the process of information dissemination. Fig. 4(b) depicts the fraction of S-state, E-state, I-state and R-state individuals as a function of the time step $t$ in the process of information spreading. We find that the fraction of A-state individuals rises to a certain height and then decreases with the increasing of the time step, and then reaches the steady state. In the UAU-SEIR model, since I-state individuals in the physical contact layer will automatically become A-state in the virtual communication layer, the fraction of A-state individuals will exceed the value in the steady state for a period of time. As the time step increases, the UAU model reaches the steady state with its own information spreading rate $\lambda$ and information recovery rate $\delta$.

Secondly, we verify the accuracy of the MMCA theoretical analysis by comparing the MMCA theoretical results with MC simulations. As shown in Fig. 5, we depict the fraction of R-state individuals ($\rho^R$) as a function of the epidemic spreading rate $\beta$ for different values of infection attenuation factor $\gamma$ and give the MMCA and the MC simulation results, respectively. It can be clearly observed that the MMCA has a good consistency with MC simulations. By calculating, when infection attenuation factor $\gamma$ is 0, 0.2 and 0.8, the relative errors of the two results are 0.89%, 0.65% and 0.21%, respectively. Next, we mainly use MMCA to analyze the following results.

Thirdly, we analyze the impacts of main parameters ($\lambda$, $\sigma$, $m$ and $\gamma$) on the epidemic spreading under the proposed model, where $\lambda$ represents the information spreading rate, $\sigma$ represents the transition rate from E-state to I-state, $m$ represents the mass media spreading rate and $\gamma$ represents the infection attenuation factor. Fig. 6 depicts $\rho^R$ as a function of $\beta$ for different values of $\lambda$, $\sigma$, $m$ and $\gamma$, respectively. Fig. 6(a)–(c) show that $\rho^R$ decreases as $\lambda$ or $\sigma$ or $m$ increase, which indicates that the final epidemic prevalence decreases as $\lambda$ or $\sigma$ or $m$ increase. Increase of $\lambda$ and $m$ means speeding up information dissemination. The dissemination of disease-related information can trigger individuals to take preventive measures to reduce the risk of infection, which inhibits the epidemic spreading. For $\sigma$, increasing $\sigma$ means that individuals transition from E-state to I-state more quickly. Since I-state individuals will automatically become A-state, which facilitates the spread of disease-related information in the virtual communication layer, so as to further inhibit the epidemic spreading. Fig. 6(d) shows that the smaller $\gamma$ will lead to the lower $\rho^R$. According to $\beta^A = \gamma \beta^U = \gamma \beta$, the smaller $\gamma$ means that A-state individuals are less likely to be infected, which decreases the epidemic spreading.
Fig. 5. Comparison of the fraction of R-state individuals ($\rho^R$) using the MMCA (solid line) and Monte Carlo (dotted line) simulations as a function of $\beta$ for different values of $\gamma = 0$ (black), $\gamma = 0.2$ (blue) and $\gamma = 0.8$ (red). The initial fraction of infected individuals (E-state) and aware individuals (A-state) are set as 2% and 0. All of the MC simulation results are obtained by averaging 1000 realizations. Other model parameters are set as follows: $\sigma = 0.6$, $\mu = 0.5$, $\lambda = 0.6$, $\delta = 0.2$ and $m = 0.3$.

Fig. 6. The fraction of R-state individuals ($\rho^R$) as a function of $\beta$ for different values of $\lambda$ (a), $\sigma$ (b), $m$ (c) and $\gamma$ (d). Other model parameters are set as follows. (a): $\sigma = 0.5$, $\mu = 0.8$, $\delta = 0.2$, $\gamma = 0$ and $m = 0$. (b): $\mu = 0.8$, $\lambda = 0.8$, $\delta = 0.2$, $\gamma = 0$ and $m = 0$. (c): $\sigma = 0.8$, $\mu = 0.6$, $\lambda = 0.8$, $\delta = 0.2$ and $m = 0$. (d): $\sigma = 0.8$, $\mu = 0.6$, $\lambda = 0.8$, $\delta = 0.2$ and $m = 0$.

Fourthly, we explore the full phase diagram ($\lambda - \beta$) for the coupled dynamics of UAU-SEIR model to further analyze the impacts of main parameters on the epidemic spreading in detail. Figs. 7–9 depict the full phase diagram ($\lambda - \beta$)
for different values of $\sigma$, $\gamma$ and $m$, respectively. We can clearly see that, in the whole range of $\lambda - \beta$ parameters, the larger $\sigma$ or $m$ will lead to the lower $\rho^R$ in Figs. 7 and 9, and the smaller $\gamma$ will lead to the lower $\rho^R$ in Fig. 8. Increase of the transition rate $\sigma$ from E-state to I-state or increase of the mass media spreading rate $m$ or decrease of the infection attenuation factor $\gamma$ can decreases the epidemic spreading, which is consistent with the conclusions reached above.

Finally, we investigate the impacts of different parameters $\lambda$, $\sigma$, $\gamma$ and $m$ on the epidemic threshold $\beta_c$ under the proposed model. Fig. 10 depicts the epidemic threshold $\beta_c$ as a function of $\lambda$ and $\sigma$ for different values of $\sigma$ and $\lambda$, respectively. Combined Fig. 10(a) with Fig. 10(b), we find that the epidemic threshold $\beta_c$ increases with the increase of $\lambda$ for different $\sigma$ and increases with the increase of $\sigma$ for different $\lambda$. Fig. 11 depicts the epidemic threshold $\beta_c$ as a function of $\gamma$ and $m$ for different values of $m$ and $\gamma$, respectively. Combined Fig. 11(a) with Fig. 11(b), we find that the epidemic threshold $\beta_c$ increases with the increase of $m$ for different $\gamma$ and decreases with the increase of $\gamma$ for different $m$. Therefore, increase of the information spreading rate $\lambda$ or increase of the transition rate $\sigma$ from E-state to I-state or decrease of the infection attenuation factor $\gamma$ or increase of the mass media spreading rate $m$ can raise the epidemic threshold.

5. Conclusions and discussions

Taking into account the characteristics of some diseases (SARS, influenza A, COVID-19, etc.) and the impact of both individuals and mass media information dissemination on the epidemic spreading, we introduce mass media into the
multiplex networks framework to study the dynamic interaction between information dissemination and SEIR-based epidemic spreading. In our model, the UAU model and the SEIR model is used to describe the process of information dissemination between individuals and epidemic spreading, respectively. Furthermore, mass media is represented by a single node linked with all individuals. Then, we use MMCA to make a theoretical analysis and derive the epidemic threshold. Finally, we verify the accuracy of MMCA through extensive MC simulations and analyze the impact of model parameters on the epidemic spreading and the epidemic threshold. The results show that reducing the proportion of asymptomatic infections and accelerating the dissemination of information (including information dissemination between individuals and via mass media) can effectively inhibit epidemic spreading and raise the epidemic threshold.

In this work, we have studied the impact of multiple factors (including asymptomatic infections, information dissemination between individuals, and information dissemination via mass media) on epidemic spreading in multiplex networks. The current results help people further understand the characteristics of SEIR-based epidemic spreading when considering the mass media mechanic and provide an important theoretical basis for disease prevention and control. However, in order to simplify the model, we did not consider the UI-state because we assume that I-state individuals will automatically become A-state. In addition, we did not consider the dynamic evolution of the network structure. In reality, both the physical contact layer and the virtual communication layer are not static but time varying. In order to be closer to the actual situation, we will consider adding UI-state and temporal network to improve our model in the future.

**CRediT authorship contribution statement**

Weicai Ma: Methodology, Software, Visualization, Writing – original draft. Peng Zhang: Conceptualization, Writing – review & Editing, Supervision. Xin Zhao: Data curation, Validation. Leyang Xue: Conceptualization, Writing – review & Editing, Formal analysis.
Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgments

This work is supported by the Fundamental Research Funds for the Central Universities, China (Grant No. 2019XD-A10), National Key R&D Program of China (Grant No. 2020YFF0305300).

References

[1] E. Bakshy, I. Rosenn, C. Marlow, L. Adamic, The role of social networks in information diffusion, in: Proceedings of the 21st International Conference on World Wide Web, 2012, pp. 519–528.
[2] F. Chierichetti, S. Lattanzi, A. Panconesi, Rumor spreading in social networks, Theor. Comput. Sci. 412 (24) (2011) 2602–2610.
[3] P. Van Mieghem, J. Omic, R. Kooij, Virus spread in networks, IEEE/ACM Trans. Netw. 17 (1) (2008) 1–14.
[4] V. Tangcharoensathien, N. Calleja, T. Nguyen, T. Purnat, M. D’Agostino, S. Garcia-Saiso, M. Landry, A. Rashidian, C. Hamilton, A. AbdAllah, et al., Framework for managing the COVID-19 infodemic: methods and results of an online, crowdsourced WHO technical consultation, J. Med. Internet Res. 22 (6) (2020) e19659.
[5] N. Ferguson, Capturing human behaviour, Nature 446 (7137) (2007) 733.
[6] Z. Wang, M.A. Andrews, Z.-X. Wu, L. Wang, C.T. Bauch, Coupled disease–behavior dynamics on complex networks: A review, Phys. Rev. E 103 (5) (2021) 052303.
[7] Y. Pan, Z. Yan, The impact of multiple information on coupled awareness-epidemic dynamics in multiplex networks, Physica A 491 (2018) 45–54.
[8] T. Shi, T. Long, Y. Pan, W. Zhang, C. Dong, Q. Yin, Effects of asymptomatic infection on the dynamical interplay between behavior and disease transmission in multiplex networks, Physica A 536 (2019) 121030.
[9] M. Kivelä, A. Arenas, M. Barthelemy, J.P. Gleeson, Y. Moreno, M.A. Porter, Multilayer networks. J. Complex Netw. 2 (3) (2014) 203–271.
[10] S. Boccaletti, G. Bianconi, R. Criado, C. Del Genio, J. Gómez-Gardenes, M. Romance, I. Sendina-Nadal, Z. Wang, M. Zanin, The structure and dynamics of multilayer networks, Phys. Rep. 544 (1) (2014) 1–122.
[11] M. De Domenico, A. Solé-Ribalta, E. Cozzo, M. Kivelä, Y. Moreno, M.A. Porter, S. Gómez, A. Arenas, Mathematical formulation of multilayer networks, Phys. Rev. X 3 (4) (2013) 041022.
[12] J. Gao, S.V. Buldyrev, H.E. Stanley, S. Havlin, Networks formed from interdependent networks, Nat. Phys. 8 (1) (2012) 40–48.
[13] P.J. Mucha, T. Richardson, K. Macon, M.A. Porter, J.-P. Onnela, Community structure in time-dependent, multiscale, and multiplex networks, Science 323 (5915) (2009) 876–878.
[14] Z. Wang, Q. Guo, S. Sun, C. Xia, The impact of awareness diffusion on SIR-like epidemics in multiplex networks, Appl. Math. Comput. 349 (2019) 134–147.
[15] C. Granell, S. Gómez, A. Arenas, Dynamical interplay between awareness and epidemic spreading in multiplex networks, Phys. Rev. Lett. 111 (12) (2013) 128701.
[16] C. Granell, S. Gómez, A. Arenas, Competing spreading processes on multiplex networks: awareness and epidemics, Phys. Rev. E 90 (1) (2014) 012808.
[17] C. Zheng, C. Xia, Q. Guo, M. Dehmer, Interplay between SIR-based disease spreading and awareness diffusion on multiplex networks, J. Parallel Distrib. Comput. 115 (2018) 20–28.
[18] C. Xia, Z. Wang, C. Zheng, Q. Guo, Y. Shi, M. Dehmer, Z. Chen, A new coupled disease-awareness spreading model with mass media on multiplex networks, Inform. Sci. 471 (2019) 185–200.
[19] J. Chen, Y. Liu, M. Tang, J. Yue, Asymmetrically interacting dynamics with multi-source confirmation from multi-source interacting dynamics in multiplex networks, 2021, arXiv preprint arXiv:2101.01340.
[20] L. He, L. Zhu, Modeling the COVID-19 epidemic and awareness diffusion on multiplex networks, Commun. Theor. Phys. 73 (3) (2020) 035002.
[21] Y. Wang, L. Wang, Interplay between complex contagion of awareness and epidemic spreading in two-layer network, in: 2020 39th Chinese Control Conference, CCC, IEEE, 2020, pp. 765–770.
[22] X. Wang, X. Zhu, X. Tao, J. Xiao, W. Wang, Y.-C. Lai, Anomalous role of information diffusion in epidemic spreading, Phys. Rev. Res. 3 (1) (2021) 013157.
[23] H. Wang, C. Ma, H.-S. Chen, H.-F. Zhang, Effects of asymptomatic infection and self-initiated awareness on the coupled disease-awareness dynamics in multiplex networks, Appl. Math. Comput. 400 (2021) 126084.
[24] D. Wang, Y. Zhao, H. Leng, Dynamics of epidemic spreading in the group-based multilayer networks, Mathematics 8 (11) (2020) 1895.
[25] C. Zuo, A. Wang, F. Zhu, Z. Meng, X. Zhao, A new coupled awareness-epidemic spreading model with neighbor behavior on multiplex networks, Mathematics 8 (9) (2020) 134–147.
[26] E. Massaro, F. Bagnoli, Epidemic spreading and risk perception in multiplex networks: A self-organized percolation method, Phys. Rev. E 90 (5) (2014) 052817.
[27] S. Ruan, W. Wang, S.A. Levin, The effect of global travel on the spread of SARS, Math. Biosci. Eng. 3 (1) (2006) 205.
[28] M. Small, C.K. Tse, Small world and scale free model of transmission of SARS, Int. J. Bifurcation Chaos 15 (05) (2005) 1745–1755.
[29] C. Dye, N. Gay, Modeling the SARS epidemic, Science 300 (5627) (2003) 1884–1885.
[30] G. González-Parra, A.J. Arenas, B.M. Chen-Charpentier, A fractional order epidemic model for the simulation of outbreaks of influenza A (H1N1), Math. Methods Appl. Sci. 37 (15) (2014) 2218–2226.
[31] M.M. Saito, S. Imoto, R. Yamaguchi, H. Sato, H. Nakada, M. Kami, S. Miyano, T. Higuchi, Extension and verification of the SEIR model on the 2009 influenza A (H1N1) pandemic in Japan, Math. Biosci. 246 (1) (2013) 47–54.
[32] B. Tang, X. Wang, Q. Li, N.L. Bragazzi, S. Tang, Y. Xiao, J. Wu, Estimation of the transmission risk of the 2019-nCoV and its implication for public health interventions, J. Clin. Med. 9 (2) (2020) 462.
[33] B. Li, D. Saad, Impact of presymptomatic transmission on epidemic spreading in contact networks: A dynamic message-passing analysis, Phys. Rev. E 103 (5) (2021) 052203.
[34] S. He, Y. Peng, K. Sun, SEIR modeling of the COVID-19 and its dynamics, Nonlinear Dyn. 101 (3) (2020) 1667–1680.
[35] S. Anna, M.I. Pratama, M. Rifandi, W. Sanusi, S. Side, Stability analysis and numerical simulation of SEIR model for pandemic COVID-19 spread in Indonesia, Chaos Solitons Fractals 139 (2020) 110072.
[36] R. Bhattacharyya, R. Kundu, R. Bhaduri, D. Ray, L.J. Beesley, M. Salvatore, B. Mukherjee, Incorporating false negative tests in epidemiological models for SARS-CoV-2 transmission and reconciling with seroprevalence estimates, Sci. Rep. 11 (1) (2021) 1–14.

[37] L. Danon, A.P. Ford, T. House, C.P. Jewell, M.J. Keeling, G.O. Roberts, J.V. Ross, M.C. Vernon, Networks and the epidemiology of infectious disease, Interdiscip. Perspect. Infect. Dis. 2011 (2011).

[38] M.J. Keeling, P. Rohani, Modeling Infectious Diseases in Humans and Animals, Princeton University Press, 2011.

[39] S. Gómez, A. Arenas, J. Borge-Holthoefer, S. Meloni, Y. Moreno, Discrete-time Markov chain approach to contact-based disease spreading in complex networks, Europhys. Lett. 89 (3) (2010) 38009.

[40] S. Gómez, J. Gómez-Gardínes, Y. Moreno, A. Arenas, Nonperturbative heterogeneous mean-field approach to epidemic spreading in complex networks, Phys. Rev. E 84 (3) (2011) 036105.

[41] A.-L. Barabási, R. Albert, Emergence of scaling in random networks, Science 286 (5439) (1999) 509–512.