Epidemic spreading with awareness and different time scales in multiplex networks

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One of the major issues in theoretical modeling of epidemic spreading is the development of methods to control the transmission of an infectious agent. Human behavior plays a fundamental role in the spreading dynamics and can be used to stop a disease from spreading or to reduce its burden, as individuals aware of the presence of a disease can take measures to reduce their exposure to contagion. In this paper, we propose a mathematical model for the spread of diseases with awareness in complex networks. Unlike previous models, the information is propagated following a generalized Maki-Thompson rumor model. Flexibility on the time scale between information and disease spreading is also included. We verify that the velocity characterizing the diffusion of information awareness greatly influences the disease prevalence. We also show that a reduction in the fraction of unaware individuals does not always imply a decrease of the prevalence, as the relative time scale between disease and awareness spreadings plays a crucial role in the systems’ dynamics. Our results provide a new view on how information influence disease spreading and can be used for the development of more efficient methods for disease control.

I. INTRODUCTION

Mathematical and computational studies of epidemic models have proven to be very important for understanding real-world disease dynamics [1, 2]. Currently, one of the main motivations behind epidemic modeling is the development of methods and models that allow to control the transmission of an infectious agent [1, 3]. These methods include the optimization of more traditional strategies to control an outbreak, such as vaccination or quarantine [1], but also novel approaches that take into account more accurately human behavioral responses [5, 9].

Individual prevention methods can considerably reduce the overall reach of an epidemic, as verified in previous studies [7, 8]. These works have analyzed the impact of awareness on epidemic spreading through a population [7, 11]. The most recent approaches have benefited from the latest developments in network theory, especially of the multilayer framework [12, 13]. The latter methodology allows to represent the interaction between individuals as a multiplex network, where the propagation of both the disease and information occurs in different layers of the whole network, though each layer encodes the network of contacts associated to the respective dynamics [8]. Other recent works have also studied the interplay between a disease and a social dynamics in multiplex networks, e.g., [13, 15], where it was found that the healthy-endemic phase transition becomes discontinuous.

Here we explore a model in which two processes co-exist: the spreading of a disease and the dissemination of awareness of the disease. Our model includes two new ingredients. First, we increase the complexity of previous models with respect to the dissemination of information by considering the dynamics of the Maki-Thompson rumor model — instead of using the traditional dynamics of disease spreading [8]. Second, we introduce a parameter that allows to control the relative time scales between the disease and rumor propagation processes. Results for scale-free networks show that the rumor dynamics can indeed reduce the epidemic prevalence. However, if we couple the characteristic time for awareness diffusion with the state of individuals, namely, by considering that infected individuals take more time to inform about its own infection, a counterintuitive behavior is revealed: the prevalence increases with the rate at which individuals become aware, despite the fact that fraction of unaware individuals decreases. The latter mechanism is important, as there are many diseases to which a similar behavior can be associated — e.g., HIV transmission, where HIV-positive patients are often reluctant to voluntarily notify their sexual partners [10]. In what follows, we present the model as well as some analytical insights and results from numerical simulations. We round off the paper by discussing our findings and presenting possible applications to the modeling of real diseases.
II. THE MODEL

Our model considers the propagation of a disease in a population, simultaneously to the spreading of information about it, by which individuals become aware of the disease and of prevention methods, reducing their contagion probabilities. These two processes run in a double-layer multiplex network: one layer for the disease spreading and another one for the information awareness to hold the disease. As of the definition of a multiplex [12][13], each layer has the same number of nodes, and there is a one-to-one link between the nodes in different layers. In this sense, we identify each pair of linked nodes from each layer as the same “individual”; the only difference from one layer to the other one lies in the structure of connections inside the layers. The links on the “epidemic layer” represent contacts that can possibly transmit the disease, whereas links on the “informational layer” represent pairs of individuals that share information with each other, like in social online networks.

The model for the epidemic spreading adopted here is a reactive SIS (susceptible-infected-susceptible) compartmental model [1][2] in which, at each time step \( \Delta t = 1 \) of the dynamics, each infected (I) node tries to transmit the disease to each of its susceptible (S) neighbors on the epidemic layer with probability \( \beta \), and then tries to recover with probability \( \mu \).

For the spreading of information awareness to prevent the transmission we use a cyclic Maki-Thomson rumor model in complex networks [17], which we call UARU (unaware-aware-stifler-unaware). Notice that the latter R here is used for the stifler compartment to avoid confusion with the susceptible (S) state in the SIS model. A stifler is an informed node who does not propagate the information anymore. When an aware (spreader) node contacts an unaware (ignorant) neighbor in the informational layer, it tries to pass the rumor about the disease. If the contacted neighbor, however, is an aware or stifler node, the node that makes the contact becomes stifler. A stifler individual can also forget the information about the disease transmission, becoming ignorant about the disease transmission again. We again use a discrete time approach [15] by considering a reactive formulation in which, at each time step \( \Delta t = 1 \), each aware (A) node first tries to inform each of its unaware (U) neighbors with probability \( \gamma \), and then becomes a stifler (R) with probability \( \kappa \). Besides, each stiffer node becomes ignorant (U) with probability \( \alpha \).

By combining the epidemic and the informational states of each node, we can describe the overall state of each individual. In our model, we have six different states, i.e., SU (susceptible and unaware), SA (susceptible and aware), SR (susceptible and stifler), IA (infected and aware) and IR (infected and stifler). Using these overall states, we define the interaction between the epidemics and awareness by adding two new features. First, a susceptible node that is informed (aware or stifler) will reduce its contagion probability by a factor \( \Gamma \) (with \( 0 \leq \Gamma < 1 \)) for each contact, meaning that it will get the disease from each of its infected neighbors with probability \( \Gamma \beta \) (less than \( \beta \)). Such a feature represents the adoption of prevention methods against the disease. Second, an additional transition called self-awareness is considered: if not informed by a neighbor, an infected-unaware (IU) node can, during the same time step, become aware with probability \( \kappa \), by knowing its own condition. This process simulates the case in which an infected subject recognizes the symptoms of the disease and becomes aware of the infection.

The following reaction equations - representing respectively the \( [1] \) infection of an unaware susceptible, \( [2] \) infection of an aware susceptible, \( [3] \) infection of a stifler susceptible and \( [4] \) healing of an infected node - describe all possible epidemic transitions (where \( x \) is used to represent an arbitrary informational state):

\[
SU + Ix \xrightarrow{\beta} IU + Ix, \tag{1}
\]
\[
SA + Ix \xrightarrow{\Gamma \beta} IA + Ix, \tag{2}
\]
\[
SR + Ix \xrightarrow{\Gamma \beta} IR + Ix, \tag{3}
\]
\[
Ix \xrightarrow{\mu} Sx. \tag{4}
\]

The informational transitions - respectively \( [5] \) information of an unaware node, \( [6] \) self-awareness of an infected unaware, \( [7] \) “stifling” of an aware node by contacting another aware node, \( [8] \) “stifling” of an aware via contact with a stifler and \( [9] \) forgetting of the information - are represented by these equations (\( x \) and \( y \) represent arbitrary epidemic states):

![Diagram](image-url)
\[ xU + yA \xrightarrow{\gamma} xA + yA, \]
\[ IU \xrightarrow{\pi} IA, \]
\[ xA + yA \xrightarrow{\sigma} xR + yA, \]
\[ xA + yR \xrightarrow{\mu} xR + yR, \]
\[ xR \xrightarrow{\alpha} xU. \]

Figure 1 presents the possible transitions between the six states, grouped according to the epidemic and informational dynamics.

The timescale of the model is controlled according to a defined probability. With probability \( \pi \), only the rumor transitions (awareness, self-awareness, stifling and forgetting) can happen during the current time step. With the complementary probability \( (1 - \pi) \), the epidemic transitions (infection and recovering) can occur. By setting the value of \( \pi \), it is possible to emulate different timescales between the two processes. For instance, a value of \( \pi \) close to 1.0 means that the rumor propagates much faster than the infectious agent.

### III. RESULTS

We performed extensive Monte Carlo (MC) simulations of the dynamics described in the last section, where we considered a multiplex network composed by two layers with scale-free organization and \( N = 1000 \) nodes each. Each layer was generated independently by using the configuration model \cite{19} with power-law exponent \( \gamma_s = -2.5 \) and minimum degree \( k_{\text{min}} = 4 \), with a resulting average degree \( \langle k \rangle \approx 7.4 \) in each layer. In order to further study the model, we also developed a Markov chain approach that consists of solving a set of fixed point equations that provide the stationary fractions of nodes in each state. The Markov chain method is described in the appendix at the end of this paper.

In figure 2, the stationary density of infected nodes \( \rho_i^\ast \) (disease prevalence) is plotted against the infection probability \( \beta \), for different values of \( \gamma, \Gamma \) and \( \pi \). For \( \pi = 0.5 \), the rumor spreading and the epidemic propagation have the same time scale, whereas for \( \pi = 0.1 \) (0.9) rumor events are slower (faster) than the events of the epidemic process. The solid lines are Markov chain calculations (see appendix), whereas symbols are results from Monte Carlo simulations. Other parameters of the model are set to: \( \mu = 0.9 \), \( \alpha = 0.6 \), \( \kappa = 0.5 \) and \( \sigma = 0.6 \).

Analyzing figure 2, we can first check that the information about the disease helps in both reducing the prevalence and increasing the epidemic threshold, by comparing curves with different values for the information spreading probability \( \gamma \). Moreover, the prevalence is decreased when the immunity provided by the awareness is total \( (\Gamma = 0.0) \) rather than partial \( (\Gamma = 0.5) \). However, the prevalence is also increased if the relative time scale \( \pi \) is greater, i.e., when the transitions of the rumor process are faster than those of the epidemic process. This is an intriguing result as, intuitively, we expect that a faster informational process should be more efficient in preventing the disease spreading. We believe that an insight into this counterintuitive effect can be obtained by...
studying simpler versions of the present model within a mean-field approach, which we left for future work.

To investigate in more detail how the variation of the relative timescales between the two processes affects the prevalence, we consider the behavior of the infected, aware, stifler and unaware stationary densities as a function of the parameter \( \kappa \) (probability of self-awareness for an infected-unaware node). Figure 3 shows these stationary densities for two different values of \( \pi \). Each curve is normalized by its value when there is no self-awareness (i.e., \( \kappa = 0 \)). We notice that, in both cases, the self-awareness is beneficial to the disease prevention, as the densities of aware (A) and stifler (R) nodes increase, thus reducing the density of unaware (U) nodes and the disease prevalence (I). Figure 3 also shows good agreement between Markov chain method and Monte Carlo simulations.

We can observe an unexpected behavior by making a minor modification to the baseline model. We extend the idea of self-awareness to stifler nodes, considering that a stifler, which is also infected by the disease, is less likely to forget the information. That is, a node who knows about its own infection does not inform other nodes and also impair the transmission of other nodes, creating additional stiflers around it. This behavior is approximately
observed in the case of HIV transmission, in which some infected individuals knows about its own infection but do not voluntarily notify their sexual partners \[16\], acting as infected-stiflers. We include this feature by reducing the probability that an infected-stifler node forgets the information by a factor of \((1 - \kappa)\) (so that the self-awareness parameter also reduces the rate at which infected-stiflers become infected-unaware). We refer to this model as \textit{modified model}, whereas the version without this modification is referred to as \textit{baseline model}.

Figure 4 shows the I, A, R and U normalized stationary densities as a function of \(\kappa\), for two values of \(\pi\), using the modified model with the newly considered transition. For \(\pi = 0.1\), as it happens in the base line model, both densities of aware and stifler nodes increase with \(\kappa\). However, for \(\pi = 0.9\), when the rumor propagates faster than the disease, the fraction of aware (A) nodes decreases with \(\kappa\), whereas the fraction of stiflers (R) increases very rapidly with \(\kappa\). This means that the propagation of the information is hindered by the self-awareness of infected stiflers, as they resist to forget the information (unaware) and then become aware (spreaders) again. Notice that, although the stifler population increases in comparison to the aware population, the density of unaware nodes still decreases with \(\kappa\), meaning that less individuals are unprotected from the disease. Nevertheless, the prevalence (I) increases with \(\kappa\) in this case.

This is another counterintuitive result, because the disease prevalence is greater even though the unaware population is smaller. To understand this, we look at the susceptible population: if most susceptible individuals are unaware of the disease, the information is concentrated at infected nodes and thus is not effective in controlling the disease. In figure 5, we study the relative distribution of the susceptible population between unaware (SU), aware (SA), stifler (SR) and the combination of the previous two (SA + SR). For the case of slower information (\(\pi = 0.1\)), the fraction of informed susceptible nodes (SA, SR) increases with \(\kappa\), as expected. However, when \(\pi = 0.9\), the opposite happens: the fraction of SA and SR decreases and the fraction of susceptible-unaware (SU) nodes increases, meaning that the fraction of susceptible nodes that are protected by the information decreases with \(\kappa\) in this case. Therefore, even if the unaware population is reduced with \(\kappa\) (as reported in figure \ref{fig:4}b), the information is actually concentrated at infected individuals, making the protection inefficient. In other words, the fraction of informed individuals always increases with \(\kappa\) but, for large \(\pi\), susceptible individuals become less informed as \(\kappa\) increases.

Hence, we conclude that the timescale, controlled by the parameter \(\pi\), plays a fundamental role on the prevalence, meaning that the relative timescale between epidemics and information determines if the self-awareness is beneficial or not for the disease prevention. We also study how the parameter \(\pi\) changes the behavior of the prevalence with \(\kappa\) on the modified model, by analyzing the prevalence \(\rho_I^\pi\) vs \(\kappa\) curves for four different values of \(\pi\). Figure 6 shows such curves, normalized by the value of the prevalence when \(\kappa = 0\).

By analyzing the plots in figure 6, we can conceive the influence of the timescale. For small \(\pi\) (faster epidemics, slower information), the prevalence exhibits its normal decreasing behavior with \(\kappa\) for both baseline and modified models. On the other hand, for larger \(\pi\) (slower epidemics, faster information), the curves for the modified model flip their slope for larger \(\kappa\) values, whereas they maintain the same behavior for the baseline model. This means that, when the informational processes are considerably faster than the disease transmission, the self-awareness process can generate too many stiflers and impair the information spreading, increasing the prevalence. For both baseline and modified models, the time scale plays an important role on determining the effectiveness of the information on reducing the disease prevalence.
We have verified that the information helps to reduce both the prevalence and the epidemic threshold of the disease. We have also observed that self-awareness, which keeps infected individuals aware of their condition, is a very effective mechanism for reducing the disease prevalence. However, in the case that the information spreads much faster than the disease, large values of self-awareness can lead to the counterintuitive result of a higher prevalence. This happens because the self-awareness can generate such an excessive number of stiflers that impair the propagation of information, with the overall effect of increasing the prevalence. Therefore, the relative time scales between the information and infection processes determines whether the information awareness is beneficial or not for the magnitude of the epidemics. In this way, our work highlights the important role played by infected individuals who help spreading the information about the disease, reducing the disease transmission and the outbreak.

Our investigations can be extended by considering other dynamics for rumor and disease spreading, as well as networks presenting assortativity and community organization. The effect of the topology of the multilayer structure on the spreading dynamics is another possible topic for further studies.

IV. CONCLUSIONS

We have analyzed the effect of information awareness to prevent the transmission of disease in multiplex networks. We have considered the Maki-Thompson rumor model for the propagation of the information, which incorporates a forgetting mechanism not included in previous related models. Besides, the rumor and disease spread at the same time but under different time scales that control the relative speeds of these two processes.

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Appendix A: Markov chain approach for the SIS/UARU model

In order to predict the behavior of the model in a double-layer network, we develop a microscopic Markov chain approach to write dynamical equations for relevant probabilities of our system. For that purpose, we follow the methodology described in [15].

For each node $i$ of the network and for each time stamp $t$, the probabilities that it is in each possible state of the model is defined as $p$. Such state can either be from a single process (e.g., $p^i_1(t)$ is the probability that node $i$ is infected (I) at time $t$) or a “composite” state (e.g., $p_{SU}^i(t)$, where $S$ represents the healthy state and $U$ stands for the social state).
from the epidemic side carry a factor of $1 - \pi$. Therefore, for instance, the probability that an infected-aware (IA) node gets healed and becomes susceptible-aware (SA) is of $(1 - \pi) \cdot \mu$, following the corresponding probability tree on the epidemic group.

The transition probabilities for processes which involve contact with neighboring nodes, namely $q_{IU}^i$ (infection of an unaware node), $q_{IR}^i$ (infection of an aware node), $r_{IU}^i$ (awareness by contacting an aware neighbor) and $r_{IA}^i$ (“stifling” - lost of interest) are defined by the following set of equations:

\[
q_{IU}^i = 1 - \prod_{j} (1 - A_{ij} p_I^j \beta),
\]
\[
q_{IR}^i = 1 - \prod_{j} (1 - A_{ij} p_I^j \Gamma \beta),
\]
\[
r_{IU}^i = 1 - \prod_{j} (1 - B_{ij} p_A^j \gamma),
\]
\[
r_{IA}^i = 1 - \prod_{j} (1 - B_{ij} (p_A^j + p_I^j) \sigma),
\]

where $A_{ij}$ and $B_{ij}$ represent the adjacency matrices for the epidemic and informational layers, respectively. Here, we point out that our goal is to study the stationary state of the system, in which all probabilities do not change in time. Therefore, the time label $t$ of all probabilities defined here (e.g., $p_{SU}^i(t), r_{IU}^i(t)$) were removed.

Based on the transition trees drawn in Fig. 7 we can write down the Markov chain equations for the probabilities of each node $i$ being in each of the six compartments (SU, SA, SR, IU, IA, IR) of the model as a fixed point set of equations, in which the time dependence is already removed:

\[
p_{SU}^i = p_{SU}^i \pi (1 - r_{IU}^i) + (1 - \pi) (1 - q_{IU}^i) + p_{SR}^i \left(\prod_j (1 - A_{ij} p_I^j \beta)\right) + p_{IU}^i (1 - \pi) \mu
\]
\[
p_{SA}^i = p_{SU}^i \pi r_{IU}^i + p_{SR}^i \pi (1 - r_{IU}^i) (1 - \pi) (1 - q_{IU}^i) + p_{IA}^i (1 - \pi) \mu
\]
\[
p_{SR}^i = p_{SA}^i \pi r_{IA}^i + p_{SR}^i \pi (1 - \pi) (1 - q_{IA}^i) + p_{IR}^i (1 - \pi) \mu
\]
\[
p_{IU}^i = p_{SU}^i [(1 - \pi) q_{IU}^i] + p_{IU}^i [\pi (1 - r_{IU}^i) (1 - \pi) + (1 - \pi) (1 - \mu)] + p_{IR}^i [\pi (1 - \alpha)]
\]
\[
p_{IA}^i = p_{SA}^i [(1 - \pi) q_{IA}^i] + p_{IA}^i [\pi (r_{IA}^i + (1 - r_{IU}^i) \alpha)] + p_{IR}^i [\pi (1 - \alpha) + (1 - \pi) (1 - \mu)]
\]
\[
p_{IR}^i = p_{SR}^i [(1 - \pi) q_{IR}^i] + p_{IR}^i [\pi (\alpha \kappa + 1 - \alpha) + (1 - \pi) (1 - \mu)]
\]

FIG. 7. Probability trees with all the possible transitions for each state. The informational group has an associated probability of $\pi$, whereas the epidemic group carries the complementary probability $1 - \pi$. For the modified model, the IR $\rightarrow$ IU transition follows the factors in red (instead of the ones in black).

The probability of each event on the informational side of figure 7 is multiplied by $\pi$, which is the probability that the informational group is chosen to be updated in the current time step. On the other hand, probabilities
Equations A6 to A10 represent the baseline model. For the modified model, in which the IR $\rightarrow$ IU has a modified probability, equations A8 and A10 are respectively replaced by:

\[
p^i_{IU} = p^i_{SU}\left[(1 - \pi)q^i_U\right] +
\quad + p^i_{IU}\left[\pi(1 - r^i_U)(1 - \kappa) + (1 - \pi)(1 - \mu)\right] +
\quad + p^i_{IR}\left[\pi\alpha\right] (A11)
\]

\[
p^i_{IR} = p^i_{SR}\left[(1 - \pi)q^i_A\right] +
\quad + p^i_{IA}\left[\pi r^i_A\right] +
\quad + p^i_{IR}\left[\pi(1 - \alpha) + (1 - \pi)(1 - \mu)\right] (A12)
\]

We solve the system of $6N$ equations (where $N$ is the number of nodes on the network) by the fixed point method, in which the LHS values are updated by applying previous values at the RHS expressions. As explained in the main text, the initial conditions are set to:

$p^0_{IA} = 0.2$, $p^0_{SU} = 0.8$ and $p^i_{IU} = p^i_{IR} = p^i_{SA} = p^i_{SR} = 0$, for $i = 0, 1, ..., N-1$. The solutions of these equations are shown in figures 2, 3 and 4 as solid lines, where we can see a good agreement between the Markov chain predictions and Monte Carlo simulations.

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