Disease and information spreading at different speeds in multiplex networks

Fátima Velásquez-Rojas
Instituto de Física de Líquidos y Sistemas Biológicos (UNLP-CONICET), 1900 La Plata, Argentina

Paulo Cesar Ventura da Silva
Instituto de Física de São Carlos, Universidade de São Paulo, São Carlos, SP, Brazil

Colm Connaughton
Mathematics Institute, University of Warwick, Gibbet Hill Road, Coventry CV4 7AL, UK and Centre for Complexity Science, University of Warwick, Coventry CV4 7AL, UK

Yamir Moreno
Institute for Biocomputation and Physics of Complex Systems (BIFI), University of Zaragoza, 50018 Zaragoza, Spain
Department of Theoretical Physics, University of Zaragoza, 50018 Zaragoza, Spain and ISI Foundation, Turin, Italy

Francisco A. Rodrigues
Instituto de Ciências Matemáticas e de Computação, Universidade de São Paulo, São Carlos, São Paulo, Brazil

Federico Vazquez
Instituto de Cálculo, FCEN, Universidad de Buenos Aires and CONICET, Buenos Aires, Argentina
(Dated: June 4, 2020)

Nowadays, one of the challenges we face when carrying out modeling of epidemic spreading is to develop methods to control the disease transmission. For doing that, we intend to explore how beneficial the information that people manage about a disease is to reduce the risk of an outbreak. In this paper we analyze the interaction between two different processes on multiplex networks: the propagation of an epidemic using the susceptible-infected-susceptible dynamics and the dissemination of information (rumor) about the knowledge of this disease—and its prevention methods—using the unaware-aware-unaware dynamics. Unlike previous related models where disease and information spread at the same time scale, we introduce here a parameter that controls the relative speed between the propagation of the two processes. We study the behavior of this model using a mean-field approach that gives results in good agreement with Monte Carlo simulations on complex networks. We find that increasing the rate of rumor propagation reduces the disease prevalence, as one may expect. However, increasing the speed of the rumor process as compare to the epidemic process has the counter intuitive result of increasing the prevalence. This result opens an interesting discussion about the effects of information spreading on disease propagation.

PACS numbers:

I. INTRODUCTION

Currently, the motivation to model epidemic spreading mathematically and computationally arises from the need to contribute to the understanding of a reality that is not alien to us and that is becoming increasingly present due to the information we handle about diseases [1, 2]. Undoubtedly, this information is part of the influence of human behavior in disease spreading and this has represented an intense research topic [1][3]. The knowledge or information we have about a disease and how this can contribute to epidemic spreading might help to develop more effective prevention methods [2][3]. Some of these methods can significantly reduce the full extent of an epidemic, as shown in previous studies [9][11]. To explore the influence of human behavior on the spread of an epidemic, these works have used a model for the spreading of rumors to simulate the spread of knowledge about the disease (and its methods of prevention) by word of mouth. In this way, the rumors—also called information—and the epidemic are considered as two diffusion processes that interact with each other. Some other works have also analyzed the impact of the information on the spread of epidemics in a population of interacting individuals [12][17].

This system can be studied using the topology of a multiplex network, where the disease and the information to prevent transmission spread in two different layers. The disease layer may represent physical or proximity contacts for the spread of airborne diseases in people who interact regularly (family, coworkers, etc.) or occasionally (people who share public transport). The information layer represents contacts between people who exchange information face-to-face or in a virtual way by means of social networks. To model the spreading of awareness (information) in this entangled epidemic-
In Fig. 1. In the epidemic layer, nodes can be either susceptible (S) or infected (I), while in the information layer nodes are either in the Unaware (U) state (an individual not aware of the disease) or in the Aware (A) state (subjects who are aware of the disease). We represent the composite state of a node with two capital letters, the first one for the epidemic state and the second one for the information state, i.e., Susceptible–Unaware (SU), Susceptible–Aware (SA), Infected–Unaware (IU), and Infected–Aware (IA).

The basic SIS dynamics, in which infected nodes transmit the disease to susceptible neighbors with rate $\beta$ and recover from the disease at rate $\mu$, is modified to introduce the interaction between information and epidemics. The information is considered as the knowledge of the prevention methods that aware individuals have to reduce the probability of contracting the disease. This is modeled as a reduction in the contagion rate by a factor $\Gamma$ ($0 \leq \Gamma \leq 1$) if the susceptible node is aware. Then, an infected node infects an SU neighbor with rate $\beta$, while the infection rate is reduced to $\Gamma \beta \leq \beta$ if the neighbor is in the SA state. The dynamics on the information layer is quite similar to that of the SIS model, i.e., an unaware node becomes aware with rate $\gamma$ by contacting an aware neighbor, and aware nodes forget the information—or simply lose interest in it—and go back to the unaware state at rate $\alpha$. Besides, the existence of infected nodes reinforces the information about the disease, which is included in the model as a “self-awareness” of the infected people, where IU nodes spontaneously become aware at rate $\kappa$.

As mentioned before, in real life it is expected that both the epidemic and information dynamics do not necessarily evolve at the same speed. For this reason we introduce a parameter $\pi$ ($0 \leq \pi \leq 1$) that tunes the relative timescales associated with the disease and rumor propagation processes, by making the information and disease transitions proportional to $\pi$ and $(1 - \pi)$, respectively. That is, $\pi$ increases the speed of the information process.
process as compared to the infection process, so that the final form of state transitions and their rates are:

\[
\begin{align*}
I_x + SU & \xrightarrow{(1-\pi)\beta} I_x + IU, \\
I_x + SA & \xrightarrow{(1-\pi)\Gamma\beta} I_x + IA, \\
I_x & \xrightarrow{(1-\pi)\mu} S_x,
\end{align*}
\]

for the epidemic process, where \(x = U, A\) represent an arbitrary information state, and

\[
\begin{align*}
yU + yA & \xrightarrow{\pi\gamma} yA + yA, \\
yA & \xrightarrow{\pi\alpha} yU, \\
IU & \xrightarrow{\pi\kappa} IA,
\end{align*}
\]

for the information process, where \(y = I, S\) represent arbitrary epidemic state. All these transitions are shown in Fig. 2.

III. NUMERICAL SIMULATION RESULTS

We perform numerical simulations of the model described in section II using a two-layer network made of two Erdős-Rényi networks that represent the information and the epidemic layer, each one with \(N = 1000\) nodes and mean degree \(\langle k \rangle = 20\). The nodes in different layers represent the same individuals but their connections may differ in both layers. We analyze the behavior of two macroscopic magnitudes of the system at the stationary state, i.e., the stationary density of infected nodes \(\rho_i^*\) (disease prevalence) and the stationary density of aware nodes \(\rho_u^*\). We are particularly interested in studying how these two magnitudes are affected by the parameter \(\pi\), which increases the speed of the information process as compared to the infection process. For instance, large values of \(\pi\) (\(\pi > 1/2\)) means fast information spreading and slow disease propagation.

In Fig. 3, we show the simulation results of the average value of \(\rho_i^*\) over \(10^4\) independent realizations of the dynamics as a function of \(\pi\), for various parameter values. By comparing the top-left panel with the bottom-left panel for \(\kappa = 0.5\), we notice that \(\langle \rho_i^* \rangle\) is larger for \(\Gamma = 0.5\) than for \(\Gamma = 0\). We can see a similar behavior if we compare top-right and bottom-right panels for \(\kappa = 1\). In general, we have verified that \(\langle \rho_i^* \rangle\) increases as \(\Gamma\) increases. This is because the infection rate of \(SA\) nodes increases with \(\Gamma\), increasing the overall infection rate and so the disease prevalence. The second and less intuitive result shown in this figure is that the prevalence increases monotonically with \(\pi\) in all panels, which seems to be a quite robust behavior, independently on the parameter values. This is an intriguing result given that \(\pi\) speeds up the information process with respect to the infection process, and thus we expect that a faster information spreading would imply in a larger number of aware people that would lead to a smaller number of infected individuals. In the next section we develop a MF approach that helps to elucidate this apparently contradictory result. Note that a similar behavior was also observed in our previous work [17] using a more complex model, suggesting that this phenomenology may be universal in these type of models.

We also notice that the increase of the prevalence with \(\pi\) is less pronounced for \(\Gamma = 0.5\), and we have verified that the curves become independent of \(\pi\) for \(\Gamma = 1\). This
is because for $\Gamma = 1$ there is no reduction in the infection rate of aware nodes and thus all susceptible nodes are infected at rate $\beta$, independently of its information state. Therefore, the disease dynamics uncouples from the information dynamics, leading to a standard SIS model with infection and recovery rates $\beta$ and $\mu$, respectively, whose stationary density of infected nodes in a MF set up, i.e., fully connected network, is $\rho^*_i = \frac{\delta \beta - \mu}{\delta \eta} = 0.85$.

For $\Gamma = 0$ and $\gamma = 0.3$ the prevalence vanishes for all $\pi$ values (triangles in top panels), and thus the system is reduced to a standard cyclic LSU dynamics akin to that of the SIS model, with transmission and recovery information rates $\gamma$ and $\alpha$, respectively, giving a stationary density of aware nodes in MF $\rho^*_\alpha = \frac{\delta \gamma - \alpha}{\delta \eta} = 0.9$.

In Fig. 4 we show the behavior of the prevalence for two values of $\Gamma$ and three values of $\pi$, as indicated in the legends. Panels (a) and (b) show the prevalence as a function of the self-awareness rate $\kappa$. We observe that the prevalence decreases with $\kappa$, confirming that the self-awareness is an effective method in reducing disease propagation. However, for $\Gamma = 0.5$ the impact of $\kappa$ on the prevalence is very small, and also the prevalence is almost independent on $\pi$ [panel (b)]. Panels (c) and (d) show the prevalence as a function of the infection rate $\beta$. As it happens in panel (b), the prevalence barely varies with $\pi$ for $\Gamma = 0.5$ [panel (d)]. We also observe a transition from a healthy phase to an endemic phase at a threshold value $\beta_c$, which is reminiscent of that found in the SIS model. To explore how the transition value $\beta_c$ depends on the information transmission rate, we calculated $\beta_c$ for $\pi = 0.5$, $\Gamma = 0.1$ and various values of $\gamma$ in the interval $(0, 1)$. Results are shown in the two-dimensional $\beta - \gamma$ phase diagram of Fig. 4 where squares represent the transition values that separate the healthy and endemic phases, calculated numerically. Starting from a population in the endemic phase with $\beta \lesssim 0.35$ and increasing $\gamma$ while keeping $\beta$ fixed, the system undergoes a transition to a healthy phase as $\gamma$ overcomes a threshold value $\gamma_c(\beta)$. However, for $\beta \gtrsim 0.35$ the system remains in the endemic phase for all $\gamma$ values. This means that, as long as the infection rate is low enough, the epidemics can be stopped by increasing the rate at which the information is transmitted between individuals but, strikingly, the information spreading is not able to stop the disease propagation when the infection rate is high enough.

We also run simulations for other values of $\pi$ and $\Gamma$ (not shown). These simulations reveal that the transition lines are independent on $\pi$. Besides, the transition line $(\beta_c, \gamma_c)$ becomes more vertical as $\Gamma$ increases, until for $\Gamma = 1.0$ it becomes the perfect vertical line $\beta_c \simeq 0.05$, independent of $\gamma$ and $\pi$. An insight into these quite remarkable behaviors is given in section IV.

Summarizing the behavior of the model with respect to the parameters we can say that, on the one hand, the disease prevalence decreases when the information spreading rates increase through $\gamma$ and $\kappa$, or when the disease recovery rate $\mu$ increases. On the other hand, the disease prevalence increases when the information recovery rate $\alpha$ decreases, or when the infection rate increases through $\beta$ and $\Gamma$. These results are expected by model construction. However, the prevalence increase with $\pi$ turns to be an unexpected and a striking result that seems harder to understand. In section IV we develop a MF approach that helps to gain an insight into these results.
IV. MEAN-FIELD APPROACH

We study the behavior of the SIS/UAU model using a mean-field approximation that assumes that, at every infinitesimal time step $dt$ of the dynamics, each node interacts with $\eta$ neighbors chosen at random among the nodes of the entire population (annealing approximation). This approach neglects correlations that appear between the states of neighboring nodes in a network, and should work reasonably well for random networks with homogeneous degree distributions and without degree correlations, such as the Erdős-Rényi networks. Then, the densities of nodes in each of the four states evolve according to the following set of coupled rate equations:

\[
\begin{align*}
\frac{d\rho_{su}}{dt} &= (1 - \pi) \beta \eta \rho_{su} \rho_i + \pi \alpha \rho_{ia} - (1 - \pi) \mu \rho_{iu} \\
\frac{d\rho_{su}}{dt} &= (1 - \pi) \mu \rho_{iu} + \pi \gamma \rho_{su} \rho_i \\
\frac{d\rho_{ia}}{dt} &= \pi \gamma \rho_{su} \rho_a + \pi \alpha \rho_{ia} - (1 - \pi) \mu \rho_{ia} \\
\frac{d\rho_{iu}}{dt} &= \pi \gamma \rho_{su} \rho_i + (1 - \pi) \mu \rho_{ia} - \pi \alpha \rho_{sa} \\
\frac{d\rho_{sia}}{dt} &= (1 - \pi) \gamma \beta \rho_{sa} \rho_i,
\end{align*}
\]

where $\rho_{xy}$ is the density of nodes in state $xy$ ($x = i, a$ and $y = u, a$), $\rho_i = \rho_{iu} + \rho_{ia}$ is the density of infected nodes, and $\rho_a = \rho_{ia} + \rho_{sa}$ is the density of aware nodes. Also, the conservation relation for the total number of nodes $\rho_{iu} + \rho_{su} + \rho_{ia} + \rho_{sa} = \rho_i + \rho_s = \rho_a + \rho_u = 1$ holds at any time. The gain and loss terms of Eqs. (1) correspond to the respective incoming and outgoing arrows at each of the four node states of Fig. 2. For instance, the gain term $(1 - \pi) \beta \eta \rho_{su} \rho_i$ in Eq. (1a) describes the fraction of nodes in state $SU$ that make the transition to state $IU$ per unit of time $dt$: an $SU$ node is infected at rate $(1 - \pi) \beta$ by each of its infected neighbors, which are a total of $\eta \rho_i$ in average.

A. Stationary states

In this section we obtain solutions of the system of Eqs. (1) at the stationary state. We are particularly interested in the behavior of $\rho_i^*$ with $\pi$, which is the most intriguing as we showed in section III. Event though Eqs. (1) are non-linear and thus it is hard to obtain closed expressions for the densities as a function of the parameters, we derive parametric equations that relate $\rho_i^*$ and $\pi$ through $\rho_a^*$, which plays the role of the parameter. For that, we obtain expressions for the different stationary densities as a function of $\rho_a^*$, as we show below.

We start by adding Eqs. (1a) and (1c) on one side, and Eqs. (1b) and (1d) on the other side, to arrive to the following rate equations for $\rho_i$ and $\rho_a$, respectively:

\[
\begin{align*}
\frac{d\rho_i}{dt} &= (1 - \pi) [\beta \eta (\rho_{su} + \Gamma \rho_{sa}) - \mu] \rho_i, \\
\frac{d\rho_a}{dt} &= \pi [\gamma \eta (1 - \rho_a) - \alpha] \rho_a + \pi \kappa \rho_{iu}.
\end{align*}
\]

A simple stationary solution of Eqs. (2) is obtained by setting $\rho_i = 0$, which leads to $[\gamma \eta (1 - \rho_a) - \alpha] \rho_a = 0$ for $\pi \neq 0$. Therefore, there are two trivial stationary states corresponding to a totally healthy population ($\rho_{su}^* = \rho_{sa}^* = 0, \rho_{iu}^* = 1$) in which (a) either all individuals are unaware ($\rho_a^* = 0, \rho_{iu}^* = 1$), or (b) there is a fraction $\rho_{iu}^* = \frac{2 \pi - \alpha}{\gamma}$ of aware individuals. This scenario corresponds to a simple UAU dynamics. At the non-trivial stationary state $\rho_i \neq 0$ with $\pi \in (0,1)$ is

\[
\beta \eta (\rho_{su}^* + \Gamma \rho_{sa}^*) - \mu = 0, \text{ and } [\gamma \eta (1 - \rho_a^*) - \alpha] \rho_a^* + \kappa \rho_{iu}^* = 0.
\]

Using the identities $\rho_{su}^* + \rho_{iu}^* = \rho_1^* - 1 - \rho_a^*$, $\rho_{sa}^* + \rho_{iu}^* = \rho_a^*$ and $\rho_i^* = \rho_{iu}^* + \rho_{iu}^*$ we can express $\rho_{su}^*$ and $\rho_{sa}^*$ in terms of $\rho_i^*$, $\rho_a^*$ and $\rho_{iu}^*$ as

\[
\begin{align*}
\rho_{su}^* &= 1 - \rho_a^* - \rho_{iu}^* \text{ and } \\
\rho_{sa}^* &= \rho_a^* - \rho_i^* + \rho_{iu}^*.
\end{align*}
\]

Substituting the expressions Eqs. (3) for $\rho_{su}^*$ and $\rho_{sa}^*$ into Eq. (3a) and solving for $\rho_i^*$ we arrive to

\[
\rho_i^* = \frac{\beta \eta - \mu}{\Gamma \beta \eta} - \frac{(1 - \Gamma)(\rho_{iu}^* + \rho_{su}^*)}{\Gamma}.
\]

Finally, replacing the expression

\[
\rho_{su}^* = \frac{[\alpha - \gamma \eta (1 - \rho_a^*)]}{\kappa} \rho_a^*
\]

for $\rho_{iu}^*$ from Eq. (3b) into Eq. (3) we obtain, after doing some algebra, the following equation that relates $\rho_i^*$ with $\rho_a^*$

\[
\rho_i^* = \frac{\beta \eta - \mu}{\Gamma \beta \eta} - \frac{(1 - \Gamma)[\kappa + \alpha - \gamma \eta (1 - \rho_a^*)]}{\Gamma \kappa} \rho_a^*.
\]

We can also express $\rho_{su}^*$ and $\rho_{sa}^*$ in terms of $\rho_a^*$. Inserting expression Eq. (3a) for $\rho_{iu}^*$ into Eq. (3a) we arrive to

\[
[\kappa + \alpha - \gamma \eta (1 - \rho_a^*)] \rho_a^* - \frac{\beta \eta - \mu}{\Gamma \beta \eta} \rho_a^*.
\]

Then, replacing Eqs. (3a) and (3b) for $\rho_{iu}^*$ and $\rho_{iu}^*$, respectively, into Eq. (4b) we obtain

\[
\rho_{su}^* = \frac{[\kappa + \alpha - \gamma \eta (1 - \rho_a^*)] \rho_a^* - \beta \eta - \mu}{\Gamma \beta \eta}.
\]

Now that we have explicit expressions for the stationary densities $\rho_{iu}^*, \rho_i^*, \rho_{sa}^*$ and $\rho_{iu}^*$ in terms of $\rho_{iu}^*$ given by Eqs. (3), (7), (8) and (9), respectively, we can obtain
an expression that relates \(\pi\) with \(\rho^*_a\) by inserting these expressions into Eq. (11) at the stationary state
\[
(1-\pi)\mu\rho_{iu} + \pi\alpha\rho_{sa} - \left[(1-\pi)\beta\rho_i + \pi\gamma\rho^*_a\right]\eta\rho_{su} = 0,
\]
and solving for \(\pi\). After doing some algebra, we finally obtain the following equation that gives \(\pi\) as a function of the density \(\rho^*_a\) and the other parameters:
\[
\pi = \frac{P(\rho^*_a)}{Q(\rho^*_a)},
\]
where \(P\) and \(Q\) are polynomial of degree two and four in \(\rho^*_a\) given by Eqs. (A3) and (A4), respectively, of Appendix A. In principle, it is possible to transform Eq. (11) into a quartic equation in \(\rho^*_a\) and find its solution, which would give an expression for \(\rho^*_a\) as a function of the model’s parameters and also an expression for \(\rho^*_i\) by inserting this expression for \(\rho^*_a\) into Eq. (11). However, as we can guess, the resulting expression would be highly complicated and not very useful. Instead, we prefer to state the analytical relationship between \(\rho^*_a\) and \(\pi\) in the parametric form \([\pi(\rho^*_a), \rho^*_i(\rho^*_a)]\), where the expressions for \(\pi(\rho^*_a)\) and \(\rho^*_i(\rho^*_a)\) are given by Eqs. (11) and (7), respectively. This parametric solution is plotted by solid lines in Fig. 3 and compared with MC simulation results (symbols). We observe that the agreement between theory and simulations is quite good for \(\Gamma = 0\), but some discrepancies arise for \(\Gamma > 0\).

Even though the analytical solution presented above describes numerical data rather well, its complicated form makes it hard to explore the behavior of the densities with \(\pi\). Instead, to gain an insight into the behavior of \(\rho^*_i\) with \(\pi\) it proves useful to analyze the simplest non-trivial case \(\gamma = 0\) and \(\Gamma = 0\), where \(\rho^*_a\) also exhibits the monotonic increase with \(\pi\) observed for the general case \(\gamma \neq 0\) and \(\Gamma \neq 0\). As we show in Appendix B, the stationary density of infected nodes for \(\gamma = \Gamma = 0\) adopts the rather simple form
\[
\rho^*_a = \frac{\alpha(\beta\eta - \mu)}{\alpha + (1 - \pi)\mu} \left[\pi(\kappa + \alpha) + (1 - \pi)\mu\right],
\]

We can check from expression Eq. (12) that for \(\kappa = 0\) is \(\rho^*_i = \frac{\beta\eta - \mu}{\beta\eta}\), which corresponds to the stationary value of \(\rho_i\) in the SIS model. Indeed, when \(\kappa = 0\) and \(\gamma = 0\) there are no transitions to aware states \(SA\) and \(IA\), and thus all nodes are unaware at the steady state \((\rho^*_a + \rho^*_i = 1)\), and subject to the standard SIS dynamics. For \(\kappa > 1\), the term \(\pi(\kappa + \alpha)\) in the numerator of Eq. (12) grows faster than the term \(\pi\alpha\) in the denominator, and thus \(\rho^*_a\) increases with \(\pi\) as we have seen already for all parameter values analyzed in section III.

This result can be understood intuitively with the help of Fig. 1 by analyzing the stationary flow between states. On the one hand, we expect that \(\rho^*_a\) decreases with \(\pi\). This is because the incoming flow \(F_{ia-sa} = (1 - \pi)\mu\rho^*_a\) (from \(IA\) to \(SA\)) decrease with \(\pi\), while the outgoing flow \(F_{sa-su} = \pi\alpha\rho^*_a\) (from \(SA\) to \(SU\)) increases with \(\pi\).

On the other hand, we proved in Appendix B that \(\rho^*_a\) is independent of \(\pi\) and given by the expression
\[
\rho^*_a = \frac{\mu}{\beta\eta - \mu},
\]

Therefore, the density of susceptible nodes \(\rho^*_a = \rho^*_a + \rho^*_i\) decreases with \(\pi\), and thus \(\rho^*_i\) increases with \(\pi\).

It proves instructive to derive Eq. (13) from the analysis of the flows of Fig. 1. Given that in the steady state the incoming and outgoing flows in any node state is the same, we have that \(F_{ia-sa} = F_{sa-su}\), and thus we can think that there is a net flow from \(IA\) to \(SU\) equal to
\[
F_{ia-su} = (1 - \pi)\mu\rho^*_a.
\]

Therefore, the total incoming flow to \(SU\) from infected states is
\[
F_{i-su} = F_{iu-su} + F_{ia-su} = (1 - \pi)\mu\rho^*_i + (1 - \pi)\mu\rho^*_a = (1 - \pi)\mu\rho^*_i,
\]

while the outgoing flow from \(SU\) to infected nodes is
\[
F_{su-i} = F_{su-ia} = (1 - \pi)\beta\eta\rho^*_su\rho^*_a.
\]

Then, the dynamics of the system corresponds to that of an SU \(\rightarrow I\) \(\rightarrow SU\) model, where we know that the stationary density of \(SU\) nodes equals the ratio between the recovery rate \((1 - \pi)\mu\) and the infection rate\((1 - \pi)\beta\eta\), leading to Eq. (13).

### B. Stability analysis

A relevant feature in models of epidemic and information spreading is the existence of a transition from a healthy phase (\(\rho^*_i = 0\)) to an endemic phase (\(\rho^*_i > 0\)) as the infection probability overcomes a threshold value \(\beta_c\), as we described in section III and showed in Figs. 4 and 5. We want to find an analytical expression for the transition line \(\beta_c(\gamma)\) of Fig. 5 along which the stability of the the healthy phase changes, so that it is stable for \(\beta < \beta_c\) and unstable for \(\beta > \beta_c\). For that, we perform a linear stability analysis of the stable fixed point within the healthy phase, which is
\[
\begin{align*}
\tilde{\rho}_1^* &= (0, 0, 0, 1) \quad \text{for } \gamma\eta < \alpha \\
\tilde{\rho}_2^* &= \left(0, \frac{\gamma\eta - \alpha}{\gamma\eta}, 0, \frac{\alpha}{\gamma\eta}\right) \quad \text{for } \gamma\eta > \alpha.
\end{align*}
\]

where \(\tilde{\rho}_n^* \equiv (\rho^*_iu, \rho^*_sa, \rho^*_ia, \rho^*_su)\), with \(n = 1, 2\). These are the two fixed points corresponding to the healthy phase obtained in section IV A, where the dynamics of aware nodes is given by Eq. (20) with \(\rho_{iu} = 0\)
\[
\frac{d\rho_a}{dt} = \pi\left[\eta\gamma(1 - \rho_a - \alpha)\right]\rho_a.
\]

The linearized form of this equation around \(\rho_a = 0\) corresponding to the fixed point \(\tilde{\rho}_1^*\) is \(d\rho_a/dt = \lambda\rho_a\), with
\( \lambda \equiv \pi (\gamma \eta - \alpha) \). Then, \( \tilde{\rho}_n^* \) is stable (unstable) for \( \lambda < 0 \) \((\lambda > 0)\), as stated in Eqs. (17), assuming \( \pi \neq 0 \).

To better handle calculations, we write the fixed points in the general form \( \tilde{\rho}_n^* = (0, A, 0, 1 - A) \), where
\[
A = 0 \quad \text{for } \gamma \eta < \alpha \quad (n = 1) \text{ and } \\
A = \frac{\gamma \eta - \alpha}{\gamma \eta} \quad \text{for } \gamma \eta > \alpha \quad (n = 2),
\]
and study their stability under a small perturbation by means of Eqs. (11). For that, we linearize Eqs. (11) around the fixed point \( \tilde{\rho}_n^* \) by setting \( \rho_{iu} = \epsilon_1 \), \( \rho_{su} = A + \epsilon_2 \) and \( \rho_{ia} = \epsilon_3 \), with \( |\epsilon_k| < 1 \quad (k = 1, 2, 3) \), and study their time evolution (the evolution of \( \tilde{\rho}_n^* \) is obtained from the other three densities). Neglecting terms of order \( \epsilon^2 \), we obtain
\[
\frac{d\epsilon}{dt} = M \epsilon
\]
where
\[
M = \begin{pmatrix} a & 0 & b \\ c & d & e \\ f & 0 & g \end{pmatrix} \quad \text{and} \quad \epsilon = (\epsilon_1, \epsilon_2, \epsilon_3),
\]
with
\[
a = (1 - \pi) [\beta \eta (1 - A) - \mu] - \pi [\kappa + \eta \gamma A],
b = (1 - \pi) [\beta \eta (1 - A) + \pi \alpha],
c = - [\pi \gamma \eta + (1 - \pi) \Gamma \beta \eta] A,
d = \pi [\gamma \eta (1 - 2A) - \alpha],
e = \pi \gamma \eta (1 - 2A) + (1 - \pi) [\mu - \Gamma \beta \eta A],
f = \pi [\gamma \eta A + \kappa] + (1 - \pi) \Gamma \beta \eta A,
g = (1 - \pi) [\Gamma \beta \eta A - \mu] - \pi \alpha.
\]
At the critical point, the determinant of matrix \( M \)
\[
\det(M) = d(af - gb)
\]
must be zero, from where obtain after doing some algebra the following relation at the transition point:
\[
[(1 - \pi) \mu + \pi (\gamma \eta + \kappa)] [(1 - \Gamma) \beta \eta A + \mu - \beta \eta] = 0. \tag{20}
\]
Given that we considered the rates \( \mu, \gamma \) and \( \kappa \) to be positive in simulations, the first term in brackets of Eq. (20) is positive, thus we have
\[
(1 - \Gamma) \beta \eta A + \mu - \beta \eta = 0.
\]
Replacing the value of \( A \) from Eqs. (18), we finally obtain the following expression for the critical infection rate:
\[
\beta_c = \begin{cases} \frac{\mu}{\gamma \eta - \alpha} & \text{for } \gamma \eta < \alpha \quad \text{and} \\ \frac{\mu}{\gamma \eta - (1 - \Gamma) \beta \eta A - \alpha} & \text{for } \gamma \eta > \alpha.
\end{cases} \tag{21}
\]
In Fig. [6] we observe that the analytical approximation of the transition line \( \beta_c(\gamma) \) from Eq. (21) (solid line) agrees quite well with the transition points obtained from simulations (squares). Given that performing numerical simulations for various values of \( \gamma \) and \( \Gamma \) are very costly, we also implemented Eq. (21) to build a transition plane in the \( \beta - \gamma - \Gamma \) space. Results are shown in the phase diagram of Fig. [6].

V. CONCLUSIONS

We have explored the interplay between the propagation of an epidemic disease using the susceptible-infected-susceptible dynamics and the dissemination of information (rumor) about the knowledge of the disease using the unaware-aware-unaware dynamics, as a simplified model from a recent study [17]. For that, we assumed that the disease and the information spread on two coupled Erdős-Rényi networks where these two processes interact with each other, and whose relative propagation speeds are controlled by an external parameter \( \pi \). We have verified that the information helps to reduce the disease prevalence and increase 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. Surprisingly, the prevalence increases with \( \pi \), that is, as the information spreads faster. This seemingly counterintuitive result was also obtained in a more complex model studied in our previous work [17] and, therefore, it seems to be universal and independent of the model details. However, it was not fully explored and understood. It is interesting to note that these results invites us to make a more extensive interpretation of the information we handle about an epidemic, something very pertinent in the current global pandemic.

In order to gain an insight into this phenomenon, we developed a MF approach to study the dynamics of the model. We found a good agreement between simulations of the model and MF results. We showed that the SIS/UAU dynamics in MF exhibits a behavior that is qualitatively the same to that found in the SIS/UAU and SIS/UARU models using the Markov chain approach and Monte Carlo simulations [17], in particular, the increase of the prevalence with \( \pi \). Besides, the MF approach al-
The trivial fixed point of this system of equations is $\rho^* = 1.0$, corresponding to a totally healthy and unaware population. The non-trivial fixed point corresponds to the stationary densities

$$\rho^*_{iu} = \frac{\alpha(\beta \eta - \mu)}{(k + \alpha) \beta \eta}$$

(B2)

$$\rho^*_{ia} = \frac{\pi \alpha \kappa (\beta \eta - \mu)}{(k + \alpha) \beta \eta [\pi \alpha + (1 - \pi) \mu]}$$

(B3)

$$\rho^*_{su} = \frac{\mu}{\beta \eta}$$

(B4)

$$\rho^*_{sa} = \frac{(1 - \pi) \mu \kappa (\beta \eta - \mu)}{(k + \alpha) \beta \eta [\pi \alpha + (1 - \pi) \mu]}$$

(B5)

The expression for the disease prevalence is

$$\rho^* = \frac{\alpha(\beta \eta - \mu)}{(k + \alpha) \beta \eta [\pi \alpha + (1 - \pi) \mu]}$$

(B6)

Equation (B6) predicts that the prevalence takes the value $\rho^* = \alpha(\beta \eta - \mu)/[(1+ \alpha) \beta \eta] = 0.1875$ and $\rho^* = (\beta \eta - \mu)/\beta \eta = 0.5$ in the $\pi = 0$ and $\pi = 1$ limits, respectively. However, these extreme cases are pathological because the above limiting values do not correspond to the value of $\rho^*_i$ at those points. That is, $\rho^*_i$ exhibits a discontinuity at $\pi = 0$ and at $\pi = 1$. To see that we rewrite Eqs. (B1)

\[
\frac{d\rho^*_{su}}{dt} = (1 - \pi) \beta \eta \rho^*_{su} \rho^*_{i} + \pi \alpha \rho^*_{ia} - (1 - \pi) \mu \rho^*_{iu} - \pi \rho^*_{iu},
\]

(B1a)

\[
\frac{d\rho^*_{ia}}{dt} = (1 - \pi) \mu \rho^*_{iu} + \pi \alpha \rho^*_{sa} - (1 - \pi) \beta \eta \rho^*_{su} \rho^*_{i},
\]

(B1b)

\[
\frac{d\rho^*_{ia}}{dt} = \pi \rho^*_{iu} - \pi \alpha \rho^*_{sa} - (1 - \pi) \mu \rho^*_{ia},
\]

(B1c)

\[
\frac{d\rho^*_{sa}}{dt} = (1 - \pi) \mu \rho^*_{ia} - \pi \alpha \rho^*_{sa}.
\]

(B1d)
whose stationary solution is \( \rho_{iu} = C_0 - \mu / \beta \eta \), \( \rho_{ia} = 0 \), \( \rho_{su} = \mu / \beta \eta \) and \( \rho_{sa} = 1 - C_0 \), where \( C_0 = \rho_a(t = 0) \) is a constant. Assuming that all individuals are unaware initially, \( C_0 = 1 \), leads to a prevalence \( \rho_i^* = (\beta \eta - \mu) / \beta \eta = 0.5 \) at \( \pi = 0 \), which is higher by a factor \((1 + \alpha) / \alpha = 2.66\) than the limit \( \pi \to 0 \) from Eq. (B6).

For \( \pi = 1 \) Eqs. (B1) are reduced to

\[
\frac{d \rho_{ia}}{dt} = \alpha \rho_{ia} - \rho_{iu}, \\
\frac{d \rho_{su}}{dt} = \alpha \rho_{sa}, \\
\frac{d \rho_{iu}}{dt} = \rho_{iu} - \alpha \rho_{ia}, \\
\frac{d \rho_{sa}}{dt} = -\alpha \rho_{sa},
\]

(B8)

whose non-trivial stationary solution is \( \rho_{iu} = \frac{C_0 - \mu / \beta \eta}{1 + \alpha} \), \( \rho_{ia} = \frac{C_1}{(1 + \alpha)} \), \( \rho_{su} = 1 - C_1 \) and \( \rho_{sa} = 0 \), where \( C_1 = \rho_i(t = 0) \). That is, the fraction of infected nodes stays constant over time. If there is one infected individual initially, then the prevalence is \( \rho_i^* = 1/N \ll 1 \) for large \( N \).

We note that the stationary density of aware nodes \( \rho_a = (\beta \eta - \mu) / [\beta \eta (1 + \alpha)] \) is independent on \( \pi \), while \( \rho_i^* \) does depend on \( \pi \). This means that both SIS and UAU dynamics are cyclic but not equivalent. This equivalence is broken by the term \( \kappa \pi \) in the spontaneous transition \( IU \to IA \). Indeed, for the \( \kappa = 0 \) case we obtain that \( \rho_i^* = (\beta \eta - \mu) / \beta \eta \) independent on \( \pi \). This gives an insight into the non-intuitive behavior of \( \rho_i^* \), as we describe in section IV A.

---

[1] R. Pastor-Satorras, C. Castellano, P. Van Mieghem, and A. Vespignani, Rev. Mod. Phys. 87, 925 (2015).
[2] G. Ferraz de Arruda, F. A. Rodrigues, and Y. Moreno, Physics Reports 756, 1 (2018).
[3] Z. Wang, C. T. Bauch, S. Bhattacharyya, A. d’Onofrio, P. Manfredi, M. Perc, N. Perra, M. Salathé, and D. Zhao, Physics Reports 664, 1 (2016).
[4] S. Funk, M. Salathé, and V. A. Jansen, Journal of the Royal Society Interface p. rsif20100142 (2010).
[5] P. Manfredi and A. D’Onofrio, Modeling the interplay between human behavior and the spread of infectious diseases (Springer Science & Business Media, 2013).
[6] S. Meloni, N. Perra, A. Arenas, S. Gómez, Y. Moreno, and A. Vespignani, Scientific Reports 1 (2011).
[7] Z. Wang, M. A. Andrews, Z.-X. Wu, L. Wang, and C. T. Bauch, Physics of Life Reviews 15, 1 (2015).
[8] S. Funka, S. Bansal, C. T.Bauch, K. T.D.Eames, W. J. Edmunds, A. P. Galvani, and P. Klepac, Epidemics 10, 21 (2015).
[9] S. Funk, E. Gilad, C. Watkins, and V. A. A. Jansen, Proceedings of the National Academy of Sciences 106, 6872 (2009).
[10] C. Granell, S. Gómez, and A. Arenas, Phys. Rev. Lett. 111, 128701 (2013).
[11] C. Granell, S. Gómez, and A. Arenas, Physical Review E 90, 012808 (2014).
[12] W. Wang, M. Tang, H. Yang, Y. Do, Y.-C. Lai, and G. Lee, Scientific Reports 4, 5097 (2014).
[13] W. Wang, Q. H. Liu, M. Cai, S. M. and. T ang, L. A. Braunstein, and H. E. Stanley, Scientific Reports 6, 29259 (2016).
[14] Q. Guo, X. Jiang, Y. Lei, M. Li, Y. Ma, and Z. Zheng, Physical Review E 91, 012822 (2015).
[15] Q. Wu, X. Fu, M. Small, and X.-J. Xu, Chaos: An Interdisciplinary Journal of Nonlinear Science 22, 013101 (2012).
[16] F. Velásquez-Rojas and F. Vazquez, Physical Review E 95, 052315 (2017).
[17] P. C. V. da Silva, F. Velásquez-Rojas, C. Connaughton, F. Vazquez, Y. Moreno, and F. A. Rodrigues, Physical Review E 100, 032313 (2019).
[18] D. P. Maki and j a. Thompson, Maynard, Mathematical models and applications : with emphasis on the social, life, and management sciences (Englewood Cliffs, N.J : Prentice-Hall, 1973), ISBN 0135616700, includes bibliographies.