Disease spreading with social distancing: a prevention strategy in disordered multiplex networks

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Abstract

The continue emerging of diseases that have the potential to become threats at local and global scales, such as influenza A(H1N1), SARS, MERS, and COVID-19, makes it relevant to keep designing models of disease propagation and strategies to prevent or mitigate their effects in populations. Since isolated systems are very rare to find in any context, specially in human contact networks, here we examine the susceptible-infected-recovered model of disease spreading in a \textit{multiplex network} formed by two distinct networks or layers that are interconnected through a fraction $q$ of shared individuals. We model the interactions between individuals in each layer through a weighted network, because person-to-person interactions are diverse (or \textit{disordered}); weights represent the contact times of these interactions and we use a distribution of contact times to assign an individual disorder to each layer. Using branching theory supported by simulations, we study a social distancing strategy where we reduce the average contact time in one layer (or in both of them, if necessary). We find a set of disorder parameters — associated with average contact times — that prevents a disease from becoming an epidemic. When the disease is very likely to spread, the system is always in an epidemic phase, regardless of the disorder parameters and the fraction of shared nodes. However we find that it is still possible to protect a giant component of susceptible individuals, which is crucial to keep the functionality of the system composed by the two interconnected layers.

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I. INTRODUCTION

Due to the increasing levels of population migration, travel, and relocation from rural to urban regions, infectious diseases that were once under control, such as Ebola [1] and measles [2], have begun to resurface [3, 4]. An outbreak of such a disease may become an epidemic if no preventive measures are undertaken, nor mitigation strategies are implemented. Worse still, the high interconnection degree between cities or countries extremely favors the spreading of a disease throughout the entire world, which may become a pandemic in a matter of months, weeks, or even days. This is what recently happened with the COVID-19 disease, declared as a pandemic on March 11, 2020, by the World Health Organization (WHO) [5, 6]. Thus, nowadays researchers across multiple disciplines model disease propagation to develop strategies that could prevent or at least curtail epidemics (and pandemics, in the worst cases). Infectious diseases usually propagate through physical contacts among individuals [3, 7], and researchers have found that modeling these contact patterns [8, 9] is best achieved using complex networks [10–13], in which individuals and their interactions are represented by nodes and links, respectively. Numerous disease propagation models have made use of complex networks, including the susceptible-infected-susceptible (SIS) [7, 10, 13] and susceptible-infected-recovered (SIR) [3, 10, 13–16] models. In these epidemic models, individuals can be in different states. For example, infected (I) individuals carry the disease and can transmit it to susceptible (S) neighbors that are not immune to the disease, while recovered (R) individuals do not participate in the propagation process because they have either recovered from a previous infection or because they have died. The SIR model, in which individuals acquire permanent immunity after recovering from an illness, is the simplest and most used to study non-recurrent diseases. In the discrete-time version of this model [7], at each time step I individuals spread the disease to their S neighbors with the same probability \( \beta \in [0, 1] \), and switch to the R state \( t_r \) time steps after being infected, where \( t_r \) is the recovery time of the disease. The propagation reaches the final stage when the number of I individuals goes to zero. At this stage, the fraction \( R \) of recovered individuals indicates the extent of the infection, since all recovered individuals were once infected. In this model, the spreading is controlled by the effective probability of infection \( T = 1 - (1 - \beta)^{t_r} \), the transmissibility, with \( T \in [0, 1] \). When \( T \) is below a critical value \( T_c \), also called the epidemic threshold, the fraction \( R \) of recovered individuals, which is
the order parameter of a continuous phase transition, is negligible compared to the system size $N$, and the system is in a non-epidemic phase. On the other hand, above $T_c$ the fraction $R$ is comparable to $N$ and thus it is said that the disease becomes an epidemic [10–19].

Different strategies have been proposed for preventing or mitigating the impact of diseases in healthy populations, the most common being vaccination [13, 20–27] and partial or complete lockdown. Vaccination is a pharmacological strategy that provides immunity against a disease to individuals, who are then unable to get infected or infect neighbors. However in emergency situations such as the COVID-19 pandemic, a new disease rapidly spreads throughout the population and, since to this date a vaccine is yet to be developed, it is necessary to take other types of measures. In contrast, lockdown is a non-pharmacological strategy which consists in isolating individuals to prevent disease propagation. Nevertheless, a major drawback of this strategy is that it may cause a significant disruption in the economy of a region, because it implies a massive cut of social interactions. In addition, large-scale implementation of this mitigation strategy is challenging. On the other hand, there are other less extreme measures than lockdown with the same spirit. “Social distancing” strategies [28–34] are a set of measures for reducing contact between individuals (keep a minimum physical distance, work from home when possible, avoid crowded places, etc.), thus decreasing the probability of disease transmission. This approach assumes that an infection is more likely to be transmitted between people if they spend more time together, and thus the goal is to shorten the duration of interaction times. Social distancing strategies, along with partial or complete lockdown, have been undertaken in many countries to face the COVID-19 pandemic due to the absence of a vaccine and the fast propagation of the disease. These kind of measures respond to the direct transmission mechanism of the virus: it is expelled in the form of droplets from an infected individual through mouth and nose (when talking, exhaling, or coughing), and can get into a healthy individual (located within a 1 meter range, approximately, and facing the infected individual) through the mucous (eyes, nose, and mouth) [35]; as individuals spend more time together the probability of infection increases.

Contact times in real-world networks usually span a broad distribution [9, 36, 37]. These kind of systems are known as disordered networks, which are characterized by the existing diversity in the strength or intensity of the interactions among the different parts of the system, and have been receiving much attention recently [11, 31, 33, 34]. In Ref. [34] disorder
is implemented using a weighted complex network [11], in which the weights associated with
links represent the normalized contact times $\omega$ of the interaction between two individuals,
and they follow a power law distribution, $W(\omega) = \frac{1}{a\omega}$ that resembles experimental
results [9, 36, 37]. The parameter $a$ is the disorder intensity that controls the range of
allowable contact times in the distribution and their average value. Also, the interactions
between individuals are categorized as either close (larger average contact time) or distant
(shorter average contact time), each representing complementary fractions ($f_1$ and $1 - f_1$,
respectively) of the total number of interactions. This is carried out by controlling the
corresponding disorder intensity of the contact times distributions. Researchers have found
that for a system in an epidemic phase ($R > 0$), when the fraction $f_1$ of close contacts is
sufficiently small, increasing the disorder intensity of the distant interactions to decrease
their average contact time may switch the system to a non-epidemic phase ($R = 0$).

A very relevant magnitude to also consider is the size of the giant cluster of susceptible
individuals (GCS), or biggest connected cluster, at the final stage. This cluster is formed
by all the remaining susceptible (healthy) individuals that are connected with each other
and it is the network that sustains the functionality of a society, e.g., the economy of a
region. Using a generating function formalism, Newman [38] showed that in the SIR model
there exists a second threshold $T^*$ above which the giant cluster of susceptible individuals
vanishes at the final stage. On the other hand, Valdez et al. [32] showed that $T^*$ is an
important parameter to determine the efficiency of a mitigation or control strategy, because
any strategy that manages to decrease the transmissibility below $T^*$ can protect a large and
connected cluster of susceptible individuals, even when the system is in an epidemic state.

The previously mentioned studies were carried out using isolated networks, i.e., networks
that do not interact with other different networks. Researchers have noted that isolated
network models ignore the “external” connections that real-world systems use to communicate
with their environment, which usually affect the behavior of the dynamical processes that
take place on complex systems [39–42]. Thus, the modeling of interconnected networks, i.e.,
networks of networks (NoN) or multilayer networks, has become utterly relevant as it allows
a more accurate representation of real systems. The ubiquity of the NoN has encouraged
researchers to use them in the study of several topics such as cascading failure [43–45],
social dynamic [46, 47], and disease propagation [48–50]. Particularly, the SIR model was
simulated and solved theoretically in an overlapped multiplex network [51] system consisting
of two individual networks or layers, in which a fraction \( q \) of shared nodes (\( q \) overlapping) is present in both layers. These shared individuals connect the different layers, and their presence makes diseases more likely to spread as \( q \) increases [51].

In this paper we study a disease spreading process using the SIR model in an overlapped two-layer multiplex network. The layers have particular disorder intensities, which represent the average contact time between individuals in each layer, and are connected through a fraction \( q \) of shared nodes. We use the branching theory, supported by extensive simulations, to study a social distancing strategy in which we increase the disorder intensity in one of the layers (or in both of them) to reduce the average contact time between individuals, with the purpose of preventing the onset of an epidemic. In addition, the ultimately goal is to protect the total giant component of susceptible individuals, formed by the healthy individuals which are in contact through both layers, that will keep the economy of a region running.

II. MODEL AND RESULTS OF THE STOCHASTIC SIMULATION

We use an overlapped multiplex network formed by two layers, \( A \) and \( B \), in which a fraction \( q \) of nodes are present in both layers, called shared nodes. For the construction of the layers, both of which are of size \( N \), we use the Molloy-Reed algorithm [52]. Each layer has its own uncorrelated degree distribution \( P_i(k) \), which gives the probability that a node has \( k \) neighbors in layer \( i = A, B \). Here we use a Poisson distribution \( P(k) = e^{-(k)} (k)^k / k! \) which is homogeneous, being the average connectivity \( \langle k \rangle \) the most probable value, and allows to easily obtain some analytical results, and a power law distribution with exponential cutoff \( k_c \), \( P(k) \propto k^{-\lambda} e^{-k/k_c} \), which is heterogeneous and is more representative of real-word networks, where some individuals may have a high number of contacts while the majority has only a few. We consider the exponential cutoff \( k_c \) in the power law distribution as real-world networks are finite and the maximum number of connections of a node is limited by the size of the system [16]. Additionally, each layer is a weighted network in which links have associated weights \( \omega \), where \( \omega \) is a normalized contact time that defines the intensity of the interaction between two individuals. The \( \omega \) values are taken from the theoretical distribution \( W_i(\omega) = 1/(a_i \omega) \) [34], \( \omega \epsilon [e^{-a_i}, 1] \), where \( a_i \) is the disorder intensity of layer \( i = A, B \). Therefore, each link of the network is assigned a weight \( \omega = e^{-a_i r} \) [53], where \( r \) is a random
number uniformly distributed within the interval \([0, 1]\).

To simulate the disease spread, we use the SIR model taking into account that the probability of infection depends not only on the type of disease, but also on the disorder intensity. All individuals are initially susceptible, except for one that randomly becomes infected, called the patient zero. At each time step, infected individuals spread the disease to susceptible neighbors with a probability \(\beta \omega\), where \(\beta\) is the intrinsic infectivity of the disease, and infected individuals recover after \(t_r\) time steps. The propagation stops when the number of infected individuals is zero in both layers. By using the model described above, we can write the transmissibility of the disease in layer \(i = A, B\) as

\[
T_{a_i} = \sum_{t=1}^{t_r} \frac{(1 - \beta e^{-a_i})^t - (1 - \beta)^t}{(a_i t)} \quad [54].
\]

Note that \(T_{a_i}\) is a decreasing function of the disorder intensity \(a_i\) because, since \(\omega \epsilon [e^{-a_i}, 1]\), for higher \(a_i\) values shorter contact times become more probable, and hence the disease is less likely to propagate. This allows us to implement a social distancing strategy by increasing the disorder intensity \(a_i\) in layer \(i\). On the other hand, in the limit \(a_i \rightarrow \infty\) we have that \(T_{a_i} \rightarrow 0\), which is a complete lockdown scenario where each individual is isolated from the rest. On the other hand, in the limit \(a_i \rightarrow 0\) there is no disorder in layer \(i\), then the infection probabilities throughout layer \(i\) are all simply equal to \(\beta\), recovering the original SIR model in which the transmissibility is \(T_{a_i} \rightarrow T = 1 - (1 - \beta)^{t_r}\).

Next, we examine the disease parameters \(\beta\) and \(t_r\) so that the system enters an epidemic phase (i.e., \(R > 0\), where \(R\) is the fraction of recovered individuals in the multiplex network) without disorder, i.e., \(a_A = a_B = 0\) (thus the interactions in both layers have the largest temporal duration). We want to analyze whether or not we can find a pair of disorder intensity values \((a_A, a_B)\) (or a set of pairs of values) that prevent the disease from becoming an epidemic, and examine how they depend on the fraction \(q\) of shared nodes. In addition, we look for disorder intensity values that can prevent the total giant component of susceptible individuals GCS (composed of S nodes connected through both layers) to fall apart, which would certainly cause a significant disruption in the economy of a region or country. We remark that the social distancing strategy is applied before the disease starts spreading and during its evolution. This could represent an optimistic scenario, where the authorities of a particular region (e.g., city or country) are well informed about the existence of an infectious disease, and they immediately undertake strict measures to get the most out of them. In order to implement social distancing strategies, the structure of a population may have to be taken into consideration. For instance, the population could be divided into two layers: one
layer representing essential workers (food supply and distribution systems, sanitary system, public transport, etc.), where social distancing is certainly difficult to apply (lower disorder intensity), while the other layer could represent people who stay at their homes and only go out to make necessary purchases, such as food and cleaning supplies, for medical care or to do paperwork, where it is usually easier for the individuals to keep a safe distance from the others (higher disorder intensity). In this structured system, it is reasonable that a fraction $q$ of individuals may belong to both groups of people.

In Fig. 1 we show the simulation results for the total fraction $R$ of recovered individuals and the size $GCS$ of the giant component of susceptible individuals that span the entire multiplex network as functions of the disorder intensity $a_B$ in layer $B$. Note that in Fig. 1 (a) the system can be either in an epidemic phase, where $R > 0$, or in a non-epidemic phase, where only small outbreaks take place and hence it is considered that $R = 0$. When $a_B$ is below a critical value $a_{Bc}$, so that $T_B > T_{Bc}$, the average contact time in layer $B$ is longer and hence the interactions between individuals in that layer. Therefore, the disease is more likely to propagate and switch the system to an epidemic phase. Conversely, when $a_B \geq a_{Bc}$ ($T \leq T_{Bc}$), shortening the average contact time limits the spread of the disease down to a negligible fraction of individuals, which avoids an epidemic (non-epidemic phase). Also, we observe that $GCS$ increases with $a_B$, as the giant cluster of susceptible individuals is less affected due to the curtail of the spread of the disease. On the other hand, in Fig. 1 (b) the distancing between individuals is not enough to prevent an epidemic, but it can preserve the GCS if $a_B$ is above a critical value $a_B^*$.

In this section we see that it is definitely possible to switch the system from an epidemic phase to a non-epidemic phase by increasing the social distancing between individuals in one layer, i.e., by controlling the temporal duration of their interactions. Besides, if there is an epidemic in the system, social distancing may prevent the GCS to fall apart, thus keeping the system functional. In addition to the computational simulations, in the next section we introduce a theoretical approach that facilitates the analysis of the phase space for $R$ and $GCS$, at the final stage of the process.
FIG. 1: Total fraction $R$ of recovered individuals (red circles) and the size $GCS$ of the giant component of susceptible individuals (green squares) at the final stage, as functions of the disorder intensity $a_B$. (a) We observe that the increase of $a_B$, which decreases the transmissibility $T_B$ by reducing the average contact time in layer $B$, can bring the system from an epidemic phase ($R > 0$) to a non-epidemic phase ($R = 0$), while keeping the integrity of the largest connected cluster of susceptible individuals — $\beta = 0$. (b) The system always seems to be in an epidemic phase ($R > 0$) and despite starting off with a null GCS, the increase of the disorder intensity can make the system functional ($GCS > 0$) — $\beta = 0.1$, $t_r = 5$, $a_A = 0$ and $q = 0.4$. The results correspond to two layers with Poisson degree distributions, with $\langle k_A \rangle = \langle k_B \rangle = 4$, $k_{\text{min}} = 0$ and $k_{\text{max}} = 40$, where $k_{\text{min}}$ and $k_{\text{max}}$ are the minimum and maximum connectivity of a node, respectively. Simulations, shown in symbols, were averaged over $10^4$ realizations and using layers of size $N = 10^5$. We only consider realizations where the number of recovered individuals in each layer is above a threshold $s_c = 200$ [19]. The full black lines correspond to theoretical results.

III. THEORY AND RESULTS AT THE FINAL STAGE

A. Phase space for $R$

It has been demonstrated that in isolated networks the final stage of the SIR model [3, 10, 13, 16] maps exactly into link percolation [16, 53, 55] in which links between nodes are occupied with probability $p$. Thus, the relevant magnitudes of this model can be obtained theoretically. The mapping holds in the thermodynamic limit, where $N \to \infty$, and
considering that the number of recovered individuals is zero unless they are above a threshold $s_c$ \cite{19}, which distinguishes between an epidemic and a small outbreak. In isolated complex networks, the critical transmissibility for which the system switches from an epidemic to a non-epidemic phase is $T_c = 1/(\kappa - 1)$, where $\kappa = \langle k^2 \rangle / \langle k \rangle$ is the branching factor of the network, and $\langle k \rangle$ and $\langle k^2 \rangle$ are the first and second moments of the degree distribution $P(k)$, respectively \cite{15,16}.

Next, we proceed to map the final stage of our social distancing model into link percolation using the branching theory and the generating functions framework \cite{16,53,55–57}. Given a two-layer multiplex network with overlapping $0 < q \leq 1$, we can write a system of transcendental coupled equations for $f_i(T_A, T_B) \equiv f_i$, which is the probability that a branch of infection (formed by recovered individuals) that originates from a random link in layer $i = A, B$ expands infinitely through any of the layers,

\begin{align}
    f_A &= (1 - q)(1 - G_A^1(1 - T_A f_A)) + q(1 - G_A^1(1 - T_A f_A)G_B^0(1 - T_B f_B)), \\
    f_B &= (1 - q)(1 - G_B^1(1 - T_B f_B)) + q(1 - G_B^1(1 - T_B f_B)G_A^0(1 - T_A f_A)),
\end{align}

where $G_i^0(x) = \sum_k P_i(k)x^k$ and $G_i^1(x) = \sum_k (kP_i(k)/\langle k \rangle)x^{k-1} = G_i^0'(x)/G_i^0'(1)$ (with $G' \equiv dG/dx$ and $G_0'(1) = \langle k \rangle$) are the generating functions of the degree and the excess degree distributions, respectively \cite{16,53,55–57}. Note that the factor $G_i^1(x)$, with $x = 1 - T_i f_i$, is the probability that in layer $i$ a branch of infection reaches a node with connectivity $k$, so that it cannot keep extending through its $k - 1$ remaining connections. In a similar way, $G_i^0(x)$ is the probability that a randomly chosen node is not reached by a branch of infection through its $k$ connections in layer $i$. Thus, $f_A$ is the sum of two main terms. First, the probability of reaching an individual only present in layer $A$ (with probability $1 - q$) so that the branch of infection expands through any of the $k - 1$ remaining connections of the individual in that layer, and second, the probability of reaching one of the shared nodes (with probability $q$) so that the branch of infection expands through any of its $k - 1$ contacts in layer $A$ or through any of its $k$ connections in layer $B$ (see Fig. 2). An analogous interpretation holds for $f_B$. Once we calculate the non-trivial roots of Eqs. (1) and (2), then the fractions $R_A$, $R_B$ and $R$ of recovered individuals (i.e., those reached by the branches of
FIG. 2: Scheme of a disordered multiplex network formed by two partially overlapped layers, A and B. The size of the layers is $N_A = N_B = 15$, and the fraction of nodes present in both layers is $q = 3/15 = 0.2$ (vertical lines are used as a guide to show the shared nodes, which are represented by boxes). The thickness of the segments represents the diversity of the normalized contact times $\omega$ between individuals. (a) Initially, all the individuals are in the susceptible (S) state, except for an infected (I) node, which kickstarts the propagation of the disease. (b) At the final stage, the recovered (R) individuals are connected by the branches of infection, which originate from the link denoted by a red arrow. One of the branches, denoted by dotted lines, corresponds to the spread of the disease only through layer A, and is represented by the first term in Eq. (1). The other branch, denoted by dash-dotted lines, is a branch of infection that spreads through both layers and is represented by the second term in Eq. (1). Infection) can be obtained from

\begin{align*}
R_A &= (1 - q)(1 - G_A^0(1 - T_Af_A)) + \xi_R, \\
R_B &= (1 - q)(1 - G_B^0(1 - T_Bf_B)) + \xi_R, \\
R &= (R_A + R_B - \xi_R)/(2 - q),
\end{align*}

where $\xi_R = q(1 - G_A^0(1 - T_Af_A)G_B^0(1 - T_Bf_B))$ is the fraction of shared recovered nodes at the final stage. The factor $2 - q$ in Eq. (5) is present because the total number of individuals in the system is $(2 - q)N$. Fig. [I] shows the results for the total fraction $R$ of recovered individuals obtained from Eq. (5) (full lines), which agree with the simulation results (symbols). We observe that the system undergoes a phase transition between epidemic ($R > 0$) and non-epidemic ($R = 0$) phases when we vary the disorder intensity $a_B$. Generally, if there is a critical disorder intensity $a_{Bc}$ for a given $a_A$ value, it can be computed by solving the
equation \( \det(J^f - I) = 0 \) evaluated at \( f_A = f_B = 0 \) (since at the critical point none of the branches of infection expands infinitely). Here \( I \) is the identity matrix and \( J^f \) is the Jacobian matrix of the system of Eqs. (1) and (2), \( J^f_{i,k} = \partial f_i / \partial f_j \),

\[
J^f|_{f_A=f_B=0} = \begin{pmatrix}
T_A(\kappa_A - 1) & qT_B\langle k_B \rangle \\
qT_A\langle k_A \rangle & T_B(\kappa_B - 1)
\end{pmatrix},
\]

where \( \kappa_i \) and \( \langle k_i \rangle \) are the branching factor and the average connectivity in layer \( i = A, B \). Then, the critical transmissibility \( T_{Bc} \) is given by

\[
T_{Bc} = \frac{T_A(\kappa_A - 1) - 1}{[T_A(\kappa_A - 1) - 1](\kappa_B - 1) - q^2 T_A\langle k_A \rangle \langle k_B \rangle}.
\]

This result differs from the one obtained in Ref. \[51\], where both layers of the multiplex network have the same transmissibility \( T \), which yields a quadratic equation for \( T_c \) with only one stable solution. Inverting \( T_{Bc} \) (and \( T_A \)) from Eq. (6), we can calculate the critical disorder intensity \( a_{Bc} \) for a given \( a_A \) value.

In Fig. 3 we show the phase diagram for \( R \) on the plane \((a_A, a_B)\), for two layers with power law degree distribution and exponential cutoff, and a disease with \( \beta = 0.1 \) and \( t_r = 5 \). Each curve shows the critical value \( a_{Bc} \) as a function of \( a_A \) for a given fraction \( q \) of shared nodes, separating the epidemic phase (\( R > 0 \) below the curve) from the epidemic-free phase (\( R = 0 \) above the curve). As \( a_A \) increases \( a_{Bc} \) decreases, indicating that for shorter contacts in layer \( A \), the critical average duration of interactions in layer \( B \) becomes larger. The effect of the shared nodes is reflected on the increase of the \( a_{Bc} \) values for fixed \( a_A \) as \( q \) increases, because these nodes facilitate the propagation between layers, which in addition widens the epidemic phase. Note that in the limit \( a_A \to \infty \), \( a_{Bc} \) approximates to the critical disorder intensity in an isolated network, \( a_{B\infty} \), as \( T_{Bc} \to 1/(\kappa_B - 1) \)—see Eq. (6)—for all \( q \) values (in Ref. \[34\], this result corresponds to a single layer with \( f_1 = 0 \)). This is because as \( a_A \to \infty \) there is complete isolation between the individuals in layer \( A \), and thus the spread of the disease in only possible in layer \( B \). An analogous analysis holds for \( a_{A\infty} \), which is the critical disorder intensity in layer \( A \) when it is isolated. Note also that for \( a_A < a_{A\infty} \) there is no critical value \( a_{Bc} \) (gray-colored region in Fig. 3), and therefore an epidemic cannot be avoided even if we completely isolate the individuals in layer \( B \). This analysis shows that the negative effect of the interconnection between layers means that, to prevent an epidemic in the system, the disorder intensities in both layers must be above the critical values corresponding to the isolated-layer case.
FIG. 3: Phase space that shows the extent of an epidemic on the \((a_A, a_B)\) plane for a disease with \(\beta = 0.1\) and \(t_r = 5\). The black full curves represent the critical intensity \(a_{Bc}\) as a function of \(a_A\), for different fractions of shared nodes \(q\) (\(q\) goes from 1 to 0.1 at intervals of 0.1, from top to bottom). Below each curve the system is in an epidemic phase \((R > 0)\) while it is in a non-epidemic phase above \((R = 0)\). Note that the epidemic phase widens as \(q\) increases, since the shared nodes ease the spreading of the disease. The results correspond to two layers with a power law degree distribution with \(\lambda_A = \lambda_B = 2.35\), exponential cutoff \(k_c = 20\), \(k_{\text{min}} = 2\), and \(k_{\text{max}} = 250\), so that the single layer critical disorder intensity is \(a_{A\infty} = a_{B\infty} \approx 2.3\).

**B. Phase space for GCS**

In what follows, we present a set of equations that allow us to calculate the size \(GCS\) of the giant component of susceptible individuals of the multiplex network at the final stage of the process. It is straightforward to write the fraction of nodes of layers \(A\) and \(B\) that belong to the GCS as

\[
GCS_A = (1-q)(G^A_0(1 - T_A f_A) - G^A_0(\nu_A)) + \xi_S,  \\
GCS_B = (1-q)(G^B_0(1 - T_B f_B) - G^B_0(\nu_B)) + \xi_S.
\]  

The first terms takes into account the probability that a node that is part of only one of the layers (with probability \(1 - q\)) belongs to the GCS. This can be written as the probability that a node of layer \(i\) is susceptible \((G^i_0(1 - T_i f_i))\) minus the probability of the node being susceptible but not belonging to the GCS \((G^i_0(\nu_i))\). On the other hand, \(\xi_S = q(G^A_0(1 - T_A f_A)G^B_0(1 - T_B f_B) - G^A_0(\nu_A)G^B_0(\nu_B))\) is the fraction of shared nodes that
belong to the GCS. A node is susceptible and does not belong to the GCS if none of its links lead to susceptible nodes that do belong to the GCS. But, if one of these links connects to a R node, in order to be susceptible, the node cannot have been infected by this R node. Thus, we define \( \nu_i \) as the probability that a random link from layer \( i \) leads to a susceptible node that does not belong to the GCS, or to a R node. However, note that in the last case the link must be unoccupied, with probability \( 1 - T_i \). Note that similar to Eqs. (1) and (2), we can write a set of coupled transcendental equations for the probabilities \( \nu_A(T_A, T_B) \equiv \nu_A \) and \( \nu_B(T_A, T_B) \equiv \nu_B \),

\[
\nu_A = (1 - T_A)f_A + (1 - q)G^A_1(\nu_A) + qG^A_1(\nu_A)G^B_0(\nu_B),
\]

\[
\nu_B = (1 - T_B)f_B + (1 - q)G^B_1(\nu_B) + qG^B_1(\nu_B)G^A_0(\nu_A).
\]

From left to right, the first term is the probability that a random link in layer \( i \) leads to a recovered node, which is \( f_i \) but considering that the link is unoccupied, with probability \( 1 - T_i \). The second is the probability that the random link connects to a node that only belongs to layer \( i \) (with probability \( 1 - q \)), so that none of its outgoing links lead to susceptible nodes belonging to the GCS, nor any of its outgoing links leads to a recovered node and is occupied. Finally, the last term is similar to the second, but the random link in layer \( i \) leads to a shared node (with probability \( q \)) so that besides its outgoing links in layer \( i \), none of its links in layer \( j \) connect to susceptible nodes that are part of the GCS, nor any of its links connects to a recovered node and is occupied. Once the values of \( \nu_i \) and consequently \( GCS_i \) for \( i = A, B \) are obtained from Eqs. (7-10), the size of the GCS can be computed

\[
GCS = (GCS_A + GCS_B - \xi_S)/(2 - q),
\]

where the factor \( 2 - q \) accounts for the total number of nodes in the system, which is \( (2 - q)N \).

Fig. 1 shows the results of the size \( GCS \) of the total giant component of susceptible individuals obtained from Eq. (11) (full lines), which agree with the simulation results (symbols). We observe that the system undergoes a phase transition between functional \( (GCS > 0) \) and non-functional \( (GCS = 0) \) phases when we vary the disorder intensity \( a_B \). To study the phase space for the GCS, we define \( \mu_i \) as the probability that a random link in layer \( i = A, B \) connects to a node belonging to the GCS (similar to what was done in Sec. III A with \( f_i \) for the recovered individuals). Recall that \( \nu_i \) is the probability that a random link connects to an S node which does not belong to the GCS or that it connects
to an R node but considering that the link is unoccupied with probability $1 - T_i$. Then we have that $\mu_i = 1 - (v_i + T_i f_i)$ and we obtain a system of equations for $\mu_A$ and $\mu_B$

$$\begin{align*}
\mu_A &= 1 - f_A - (1 - q)G_A^A(u_A - \mu_A) - qG_A^A(u_A - \mu_A)G_B^B(u_B - \mu_B), \\
\mu_B &= 1 - f_B - (1 - q)G_B^B(u_B - \mu_B) - qG_B^B(u_B - \mu_B)G_A^A(u_A - \mu_A),
\end{align*}$$

(12)

(13)

where $u_i \equiv 1 - T_i f_i$. Given a disorder intensity value $a_A$, the condition for the existence of a critical value $a_B^*$ for the GCS is that $\det(J^\mu - I) = 0$ evaluated at $\mu_A = \mu_B = 0$, where $J^\mu$ is the Jacobian matrix of the system of Eqs. (12) and (13), $J_{i,k}^\mu = \partial \mu_i / \partial \mu_j$,

$$J^\mu|_{\mu_A=\mu_B=0} = \begin{pmatrix}
(1 - q)G_A^A(u_A) + qG_A^A(u_A)G_B^B(u_B) & qG_A^A(u_A)G_B^B(u_B) \langle k_B \rangle \\
qG_B^B(u_B)G_A^A(u_A) \langle k_A \rangle & (1 - q)G_B^B(u_B) + qG_B^B(u_B)G_A^A(u_A)
\end{pmatrix}.$$

In this case we obtain an implicit equation for $f_A$, $f_B$ and $a_B^*$, with $a_A$ fixed. Then we proceed to solve it together with Eqs. (1) and (2). Fig. 4 shows the phase diagram for GCS on the plane $(a_A, a_B)$, for a disease with $\beta = 0.1$ and $t_r = 5$. Each curve shows the critical value $a_B^*$ as a function of $a_A$ for a given fraction $q$ of shared nodes, separating the non-functional phase (GCS $= 0$ below the curve) from the functional phase (GCS $> 0$ above the curve).

We observe that as $a_A$ increases $a_{BC}$ decreases, indicating that for shorter contacts times in layer $A$, the critical average duration of interactions in layer $B$ grows. The effect of the shared nodes is reflected on the increase of $a_B^*$ values for fixed $a_A$ as $q$ increases, because these nodes facilitate the propagation between layers, which makes the GCS to decrease. Thus, the non-functional phase widens for larger $q$ values. In this case, regardless of the fraction $q$ of shared nodes present in the multiplex network, $a_B$ decreases until it becomes zero. From there on, for any $a_A$ value the system is in a functional phase independently of $a_B$. This means that if the average contact time is short enough in one layer, then the GCS will not vanish even if the duration of interactions in the other layer is extremely high.

In Fig. 5 we show the critical curve for the GCS together with the critical curve for $R$. We observe that the phase space projected in the $(a_A, a_B)$ plane is now divided into three regions, which is the most general scenario. Region I corresponds to a non-epidemic and functional phase (NE-F), the best possible scenario, in which the disorder intensities in both layers are high enough, i.e., social distancing measures are undertaken intensively. In region II the distancing is moderate and the epidemic can not be avoided, but there is still a GCS, which means that the system remains functional (E-F). Finally, in region III the disease
FIG. 4: Phase space that shows the integrity of the giant component of susceptible individuals (GCS) on the \((a_A, a_B)\) plane for a disease with \(\beta = 0.1\) and \(t_r = 5\). The black full curves represent the critical intensity \(a_B^*\) as a function of \(a_A\), for different fractions of shared nodes \(q\) \((q\) goes from 1 to 0.4 at intervals of 0.1, from top to bottom). The system is in a non-functional phase \((GCS = 0)\) below each curve, while is in a functional phase above it \((GCS > 0)\). Note that the non-functional phase widens as \(q\) increases, because the shared nodes facilitate the propagation of the disease. The results correspond to two layers with a power law degree distribution, with \(\lambda_A = \lambda_B = 2.35\), exponential cutoff \(k_c = 20\), \(k_{\text{min}} = 2\), and \(k_{\text{max}} = 250\).

is certainly likely to spread because the disorder intensities are quite low, which not only fails to prevent an epidemic but also makes the GCS to fall apart, causing the system to collapse (E-NF). Considering this, even though the social distancing efforts may not prevent that a particular disease extends through a significant portion of the population, they could serve to the purpose of keeping the integrity of the system. It is important to note that, if a GCS remains functional after the end of an epidemic, it is highly recommended not to relax the set of measures undertaken to prevent the transmission of the disease, since there is always the possibility of a second outbreak (originating, for instance, from an imported or undetected case).

We want to point out that our overlapped multiplex network model is a simplification of what usually happens in real social networks. For instance, it is known that nowadays passenger traffic is one of the main causes of the dissemination of a disease across different regions (cities, states, countries). A way to tackle this issue is to isolate individuals once
FIG. 5: Division of the phase space on the \((a_A, a_B)\) plane, according to the outcomes of \(R\) and \(GCS\) in the final stage. The full and dashed lines correspond to the critical curves for \(R\) and \(GCS\), respectively. A strict social distancing policy (i.e., high disorder intensities in both layers) ensures the system to lie on a non-epidemic and functional phase (region I, NE-F phase). As the distancing policies are relaxed, the emergence of an epidemic becomes more likely (region II, E-F phase), and if the policies are rather week even the GCS falls apart, disrupting the functionality of the system (region III, E-NF phase). The results correspond to two layers with a power law degree distribution, with \(\lambda_A = \lambda_B = 2.35\), exponential cutoff \(k_c = 20\), \(k_{\text{min}} = 2\), \(k_{\text{max}} = 250\), and \(q = 0.3\). Also we consider \(\beta = 0.1\) and \(t_r = 7\).

they arrive to its destination, which is currently being implemented during the COVID-19 pandemic. This way, it is expected that the passengers cannot spread the disease within the region. To take into account this possibility, instead of using an overlapped multiplex network, we could devise a model in which shared nodes do not belong to both layers, but rather they connect to nodes from other layers according to an inter-layer degree distribution. These nodes would represent individuals that travel to other regions or countries, which are isolated with a certain probability. Such a model may provide more realistic and widely applicable results, and may encourage researchers to devise more suitable and efficient strategies for preventing/mitigating the spread of a disease. In addition, it would be relevant to study the temporal evolution of a disease in this scenario, and how it would respond to mitigation measures that are undertaken with some delay.
IV. CONCLUSIONS

We apply the SIR model to study the spread of a disease in a partially overlapped multiplex network composed of two layers, in which a fraction $q$ of individuals belong to both layers and also each layer has its own distribution of contact times. We propose a social distancing strategy that reduces the average contact time of the interactions between individuals within each layer, by increasing its respective disorder intensity. When the disorder intensity in a layer is below its critical value for $q = 0$ (the isolated-layer case), an epidemic cannot be avoided, even when the individuals in the other layer are completely isolated. However, we find that it is still possible to protect the functional network of susceptible individuals through social distancing policies, i.e., by increasing the disorder intensity. This is fundamental to keep running the economy of a society. In the best case scenario, when the disorder intensity in a layer is above its critical value for $q = 0$, we find that there is a critical disorder intensity in the other layer that can reduce the epidemic size to zero. All the critical values increase with the overlapping $q$ because the individuals that are shared by the layers ease the spread of disease, so that more social distancing measures must be undertaken to prevent an epidemic. All in all, the control of contact times between individuals can serve as a prevention strategy that overcomes the overlapping effect in multiplex networks, preventing not only an epidemic, but also the economic collapse of a region or country, which might be equally bad.

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