May the Best Meme Win!: New Exploration of Competitive Epidemic Spreading over Arbitrary Multi-Layer Networks

Faryad Darabi Sahneh and Caterina Scoglio

Electrical and Computer Engineering Department, Kansas State University

I. INTRODUCTION

Multiple viral spreading within a single population involves very rich dynamics [1], attracting substantial attention [2–4]. Applications of these types of models extend beyond physiological viruses, as ‘virus’ may refer to products [5], memes [6], pathogens [7], etc. Multiple virus propagation is a mathematically challenging problem. This problem becomes particularly much more complicated if the network through which viruses propagate are distinct. Current knowledge of how hybridity of underlying topology influences fate of the pathogens is very little and limited. These systems are usually mathematically intractable, hindering conclusive results on spreading of multiple viruses on multi-layer networks.

Another source of complexity for this problem are multiple interaction possibilities among viruses. For example, viruses may be reinforcing [8], weakening [9], exclusive [10], or asymmetric [3,11]. Newman [10] employed bound percolation to study the spread of two SIR viruses in a host population through a single contact network, where a virus takes over the network, then a second virus spreads through the resulting residual network. The paper proved a coexistence threshold above the classical epidemic threshold, indicating the possibility of coexistence in SIR model. Karrer and Newman [1] extended the work to the more general case where both viruses spread simultaneously. For SIS epidemic spreading, Wang et al. [12] studied competitive viruses and proved exclusive, competitive SIS viruses cannot coexist in scale-free networks.

Multilayer networks generate interesting results for competitive viral spreading. This type of models have implications in several applications like product adoption (e.g. Apple vs. Android smart phones), virus-antidote propagation, meme propagation, opposing opinions propagation, and etc. In competitive spreading scenario, if infected by one virus, a node (individual) cannot be infected by the other virus. Funk and Jansen [2] extended the bond percolation analysis of two competitive viruses to the case of a two-layer network, investigating effects of layer overlapping. Granell et al. [9] studied the interplay between disease and information co-propagation in a two-layer network consisting of one physical contact network spreading the disease and a virtual overlay network propagating information to stop the disease. They found a meta-critical point for the epidemic onset leading to disease suppression. Importantly, this critical point depends on awareness dynamics and the overlay network structure. Wei et al. [13] studied SIS spreading of two competitive viruses on an arbitrary two-layer network, deriving sufficient conditions for exponential die-out of both viruses. They introduced a statistical tool, Eigen-Predict, to predict viral dominance of one competitive virus over the other [4].

In this paper, we address the problem of two competitive viruses propagating in a host population where each virus has distinct contact network for propagation. In particular, we study an S1IS1S2S model as the simplest extension from SIS model for single virus propagation to competitive spreading of two viruses on a two-layer network. From topology point of view, our study is comprehensive because our multilayer network is allowed to have any arbitrary structure.

Our paper is most relevant to [13] and [4]. Wei et
al. conjectured in [13] and numerically observed in [3] that “the meme whose first eigenvalue$^1$ is larger tends to prevail eventually in the composite networks.” We challenge this argument from two aspects: First, the definition of viral dominance in [3] is related to comparison of fractions of nodes infected by each virus. However, when comparing two viruses with two different contact networks, having a larger eigenvalue is not a direct indicator of a higher final fraction of infected nodes. In fact, it is possible to create two distinct network layers where a meme spreading in the population with smaller eigenvalue takes over a much larger fraction of the population. We find the definition of viral dominance presented in [3] cannot be corroborated with eigenvalues without severe restriction to a specific family of networks.

Second, and of paramount interest in this paper, largest eigenvalue is a graph property$^2$ of the layers in isolation and thus does not have the capacity to discuss the joint influence of the network topology, unless some sort of symmetry or homogeneity is assumed. In fact, the generation of one layer in their synthetic multi-layer network via the Erdos Reiny model [4] dictated a homogeneity in their multilayer networks, creating a biased platform for further observations of layer interrelations. Our work more accurately addresses network interrelation than presented by Wei et al. [4] in moving beyond viral aggressivity in isolation. We derived formulae more accurately and fully describing effect of individual network layers and their interrelatedness.

We quantitate interrelations of contact layers in terms of spectral properties of a set of matrices. Therefore, our results are not limited to any homogeneity assumption or degree distribution and network model arguments. We find analytical results determining extinction, mutual exclusion, and coexistence of the viruses by introducing concepts of survival threshold and winning threshold. Furthermore, we show possibility of coexistence in SIS-type competitive spreading over multilayer networks. Not only do we prove a coexistence region rigorously, we quantitate it via interrelation of central nodes across the network layers. None or small overlapping of central nodes of each layer is the key determinant of coexistence. We employ a novel multilayer network generation framework to obtain a set of networks so that individual layers have identical graph properties while the interrelation of network layers varies. Therefore, any difference in outputs is purely the result of interrelation. This makes ours a paradigmatic contribution to shed light on topology hybridity in multilayer networks.

II. COMPETITIVE EPIDEMICS IN MULTI-LAYER NETWORKS

In this paper, we study a continuous time $SI_1SI_2S$ model of two competitive viruses propagating on a two-layer network, initially proposed in discrete time$^3$ [13].

A. Multilayer Network Topology

Consider a population of size $N$ among which two viruses propagate, acquiring distinct transmission routes. Represented mathematically, the network topology is a multi-layer network because two link types are present; one type allows transmission of virus 1 and the other type, allows transmission of virus 2. We represent this multilayer network as $G(V, E_A, E_B)$, where $V$ is the set of vertices (nodes) and $E_A$ and $E_B$ are set of edges (links). By labeling vertices from 1 to $N$, adjacency matrices $A \triangleq [a_{ij}]_{N \times N}$ and $B \triangleq [b_{ij}]_{N \times N}$ correspond to edge sets $E_A$ and $E_B$, respectively, where $a_{ij} = 1$ if node $j$ can transmit virus 1 to node $i$, otherwise $a_{ij} = 0$, and similarly $b_{ij} = 1$ if node $j$ can transmit virus 2 to node $i$, otherwise $b_{ij} = 0$. We assume the network layers are symmetric, i.e., $a_{ij} = a_{ji}$ and $b_{ij} = b_{ji}$. Corresponding to adjacency matrices $A$, we define $d_A$ as the node degree vector, i.e., $d_{A,i} = \sum_{j=1}^{N} a_{ij}$, $\lambda_1(A)$ as the largest eigenvalue (or spectral radius) of $A$, and $\mathbf{v}_A$ as the normalized dominant eigenvector, i.e., $A\mathbf{v}_A = \lambda_1(A)\mathbf{v}_A$ and $\mathbf{v}_A^T\mathbf{v}_A = 1$. We similarly define $d_A$, $\lambda_1(A)$, and $\mathbf{v}_A$ for adjacency matrix $B$.

Unlike simple, single-layer graphs, multilayer networks have not been studied much in network science. We define simple graphs $G_A(V, E_A)$ and $G_B(V, E_B)$ to refer to each isolated layer of the multilayer network $G(V, E_A, E_B)$. This allows us to argue multilayer network $G$ in terms of simple graphs $G_A$ and $G_B$ properties and their interrelation. FIG. 1 shows a schematics of the two-layer network.

B. $SI_1SI_2S$ Model

The $SI_1SI_2S$ model is an extension of continuous-time SIS spreading of a single virus on a simple graph [14][15] to modeling of competitive viruses on a two-layer network. In this model, each node is either ‘Susceptible,’ ‘$I_1$–Infected,’ or ‘$I_2$–Infected’ (i.e., infected by virus 1 or 2, respectively), while virus 1 spreads through $E_A$ edges and virus 2 spreads through $E_B$ edges.

In this competitive scenario the two viruses are exclusive: a node cannot be infected by virus 1 and virus 2

---

$^1$ Wei et al. [2] defined first eigenvalue of of a meme as $\beta \lambda_1 - \delta$, where $\beta$ is infection probability, $\delta$ is curing probability, and $\lambda_1$ is spectral radius of the underlying graph layer.

$^2$ A graph property is any property on a graph which is invariant under relabeling of nodes. Eigenvalues, degree moments, graph diameter, etc. are examples of graph property.

$^3$ Wei et al. [13] referred to their model as $SI_1I_2S$. We prefer $SI_1SI_2S$ as a better candidate to emphasize impossibility of direct transition between $I_1$ and $I_2$ in this model.
The $SI_1SI_2S$ model is essentially a coupled Markov process. For a network with arbitrary structure, this model becomes mathematically intractable due to exponential explosion of its Markov state space size \cite{10}. To overcome this issue with coupled Markov processes, applying closure techniques results in approximate models with much smaller state space size, however at the expense of accuracy. Specifically, a first order mean-field type approximation \cite{10} suggests the following differential equations for the evolution of infection probabilities of virus 1 and 2, denoted by $p_{1,i}$ and $p_{2,i}$ for node $i$, respectively:

$$
\dot{p}_{1,i} = \beta_1 (1 - p_{1,i} - p_{2,i}) \sum_{j=1}^{N} a_{ij} p_{1,j} - \delta_1 p_{1,i}, \quad (1)
$$

$$
\dot{p}_{2,i} = \beta_2 (1 - p_{1,i} - p_{2,i}) \sum_{j=1}^{N} b_{ij} p_{2,j} - \delta_2 p_{2,i}, \quad (2)
$$

for $i \in \{1, \ldots, N\}$, with the state-space size of $2N$. This model is an extension of NIMFA model \cite{13} for SIS spreading on simple graphs.

Our competitive virus propagation model \cite{12} exhibits rich dynamical behavior depending on epidemic parameters and contact network multi-layer structure. Values of effective infection rates $\tau_1 \triangleq \frac{\beta_1}{\delta_1}$ and $\tau_2 \triangleq \frac{\beta_2}{\delta_2}$ of virus 1 and 2 yields several possible outcomes for $SI_1SI_2S$ model \cite{12}. In particular, both viruses may extinct ultimately, or one removes the other one, or both coexist.

C. Problem Statement

Linearization of our $SI_1SI_2S$ model \cite{12} at the healthy equilibrium (i.e., $p_{1,i} = p_{2,i} = 0, i \in \{1, \ldots, N\}$) demonstrates the exponential extinction condition for both viruses. When $\tau_1 < 1/\lambda_1(A)$ and $\tau_2 < 1/\lambda_1(B)$, any initial infections exponentially die out. In this paper, we refer to such critical value as no-spreading threshold because a virus with a lower effective infection rate is too weak to spread in the population even in the absence of any viral competition.

Wei et al. \cite{13} detailed the no-spreading condition as: If $\tau_1 < 1/\lambda_1(A)$, virus 1 does not spread, exponentially dying out. Importantly, exponential extinction of both viruses occurs only if $\tau_1 < 1/\lambda_1(A)$ and $\tau_2 < 1/\lambda_1(B)$ simultaneously. Dynamical interplay between the competitive viruses does not affect the no-spreading thresholds $\tau_1^0 = 1/\lambda_1(A)$ and $\tau_2^0 = 1/\lambda_1(B)$ for virus 1 and virus 2. These thresholds remain independent of viral aggressivity of competitive viruses and network layers interrelation. Exponential extinction is the only analytical outcome in Wei \cite{13}. Our paper addresses two scenarios where for both viruses $\tau_1 > 1/\lambda_1(A)$ and $\tau_2 > 1/\lambda_1(B)$.

**Problem:** Assume the effective infection rates of each virus is larger than their no-spreading threshold, i.e., $\tau_1 > 1/\lambda_1(A)$ and $\tau_2 > 1/\lambda_1(B)$:

1. Will both viruses survive (coexistence) or will one virus completely remove the other (mutual exclusion)?
2. Which characteristics of multi-layer network structure allow for coexistence?

These questions pertain to long term behaviors of competitive spreading dynamics. To address these questions, we perform a steady-state analysis of \( S1S1S2S \) model. Specifically, bifurcation techniques are used to find two critical values: survival threshold and winning threshold to determine if a virus will survive and whether it can completely remove the other virus. Significantly, we go beyond these threshold conditions and examine interrelations of network layers. Using eigenvalue perturbation, we find interrelations of dominant eigenvectors and node-degree vectors of network layers are critical determinants in ultimate behaviors of competitive viral dynamics.

### III. MAIN RESULTS

Given our stated objective to study long-term behavior of \( S1S1S2S \) model for competitive viruses, we use bifurcation analysis to study the steady-state behavior of \( S1S1S2S \) model. Application of bifurcation analysis to the SIS model of a single virus on a simple graph determines the critical value at which a non-healthy equilibrium emerges [14], determining a survival threshold for the virus. Interestingly, no-spreading threshold and survival threshold coincide for this SIS model. However, we expect these two critical values are distinct for \( S1S1S2S \) because a virus may initially spread in an almost entirely susceptible population but then die out from competition with a simultaneous virus having a sufficiently stronger infection rate.

In fact, the survival threshold is larger than the no-spreading threshold, monotonically increasing with the aggressivity of the other competitive virus. Furthermore, a surviving virus can even be so aggressive to completely remove the other virus. Consequently, competitive spreading induces an additional threshold concept-the winning threshold-determining the critical value of effective infection rate for a virus to prevail as sole survivor.

The determination of the two thresholds for each virus involves four quantities. We are able to deduce winning thresholds from survival thresholds, which then become our sole focus. Furthermore, with no loss of generality, we only find survival threshold of virus 1 because of expressions duality.

Unfortunately, any conclusive understanding of the system is hindered by the complex interdependency of survival threshold of one virus on the multilayer network topology and the aggressiveness of the competitive virus. While complete analytical solution of survival threshold appears impossible, we characterize possible solutions with explicit analytical expressions. This step is a unique contribution to current understanding of competitive spreading over multi-layer networks with solid and quantitative implications on role of multilayer network topology.

### A. Threshold Equations

Bifurcation analysis of \( S1S1S2S \) model equilibriums finds the survival threshold. Our competitive virus propagation model \((12)\) yields the equilibrium equations:

\[
\frac{p_{1,i}^*}{1-p_{1,i}^* - p_{2,i}^*} = \tau_1 \sum a_{ij} p_{1,j}^*,
\]

\[
\frac{p_{2,i}^*}{1-p_{1,i}^* - p_{2,i}^*} = \tau_2 \sum b_{ij} p_{2,j}^*,
\]

for \( i \in \{1, ..., N\} \). The healthy equilibrium (i.e., \( p_{1,i}^* = p_{2,i}^* = 0, \forall i \)) is always a solution to the above equilibrium equation \((3)\). Long term persistence of infection in the population is associated with non-zero solution for the equilibrium equations \((14)\). We use bifurcation theory to identify critical values for effective infection rates \( \tau_1 \) and \( \tau_2 \) such that a second equilibrium, aside from the healthy equilibrium, emerges. The critical value for one virus is a function of the effective infection rate of the other virus. Without loss of generality, we determine the survival threshold for virus 1 by finding the critical effective infection rate \( \tau_{1c} \) as a function of \( \tau_2 \).

**Definition:** Given virus 2 effective infection rate \( \tau_2 \), the survival threshold value \( \tau_{1c} \) is the smallest effective infection rate that virus 1 steady state infection probability of each node is positive for \( \tau_1 > \tau_{1c} \). For \( \tau_2 \in [0, +\infty) \) as an independent variable, \( \tau_{1c} \) constitutes a survival threshold curve, monotonically increasing function of \( \tau_2 \), denoted by \( \Phi_1(\tau_2) \).

The above definition for survival threshold value indicates that exactly at the threshold value, \( p_{1,i}^*|_{\tau_1=\tau_{1c}} = 0 \) and \( \frac{dp_{1,i}^*}{d\tau_1}|_{\tau_1=\tau_{1c}} > 0 \) for all \( i \in \{1, ..., N\} \). Taking the derivative of equilibrium equations \((3)\) with respect to \( \tau_1 \), and defining

\[
w_i \triangleq \frac{dp_{1,i}^*}{d\tau_1}|_{\tau_1=\tau_{1c}}, \quad y_i \triangleq p_{2,i}^*|_{\tau_1=\tau_{1c}},
\]

we find the survival threshold \( \tau_{1c} \) is the value for which nontrivial solution exists for \( w_i > 0 \) in

\[
w_i = \tau_{1c}(1 - y_i) \sum a_{ij} w_j,
\]

where \( y_i \) is the solution of:

\[
\frac{y_i}{1-y_i} = \tau_2 \sum b_{ij} y_j,
\]

according to equilibrium equation \((4)\).

Equation \((6)\) is an eigenvalue problem. Among all the possible solutions, only

\[
\tau_{1c} = \frac{1}{\lambda_1(diag(1-y_i)A)}
\]

is acceptable; according to Perron-Frobenius Theorem, only the dominant eigenvector of the matrix \( diag(1-
threshold according to (7). For values of \( \tau_2 \) close to \( 1/\lambda_1(B) \), we use eigenvalue perturbation technique and study sensitivity of threshold equation (6) respective to deviation in \( \tau_2 \) from \( 1/\lambda_1(B) \). As detailed in the Appendix, we find

\[
\frac{d\tau_{1c}}{d\tau_2} \bigg|_{\tau_2=\tau_{1c}} = \frac{\lambda_1(B)}{\lambda_1(A)} \frac{\sum v_{A,i}^2 v_{B,i}}{\sum v_{B,i}^2}, \tag{10}
\]

expressing the dependency of virus 1 survival threshold \( \tau_{1c} \) to effective infection rate of virus 2 \( \tau_2 \) for values of \( \tau_2 \) close to \( 1/\lambda_1(B) \). Expression (10) consists of two components: \( \frac{\lambda_1(B)}{\lambda_1(A)} \), the spectral radius ratio of each network layers in isolation, and \( \frac{\sum v_{A,i}^2 v_{B,i}}{\sum v_{B,i}^2} \), which determines the influence of interrelations of the two layers. Significantly, if \( \sum v_{A,i}^2 v_{B,i} \) is small, expression (10) suggests the virus 1 survival threshold is not influenced by virus 2 infection rate. This has very interesting interpretations: when spectral central nodes of \( G_A \) (those nodes with larger element in dominant eigenvector of \( G_A \)) are are spectrally insignificant in \( G_B \), the virus 1 survival threshold does not increase much by \( \tau_2 \). In other words, virus 2 does not compete over accessible resources of virus 1, therefore, virus 1 is not affected much by the co-propagation. On the other hand, if spectral central nodes of \( G_A \) have significant spectral centrality in \( G_B \), then \( \sum v_{A,i}^2 v_{B,i} \) is maximal indicating considerable dependency of survival threshold of virus 1 on aggressiveness of the other virus. From (10), the die-out threshold curve \( \Phi_1(\tau_2) \) can be approximated close to \( (\tau_2, \tau_1) = \left( \frac{1}{\lambda_1(B)}, \frac{1}{\lambda_1(A)} \right) \) as

\[
\Phi_1(\tau_2) \simeq \frac{1}{\lambda_1(A)} \left\{ 1 + \frac{\sum v_{A,i}^2 v_{B,i}}{\sum v_{B,i}^2} (\lambda_1(B)\tau_2 - 1) \right\}. \tag{11}
\]

Studying threshold equations (6) - (7) for \( \tau_2 \rightarrow \infty \), we find \( \tau_{1c} \rightarrow \infty \) is the inverse of the spectral radius of \( D_B^{-1}A \) (see Appendix for detailed derivation):

\[
\frac{\tau_{1c}}{\tau_2} \bigg|_{\tau_2=\infty} = \frac{1}{\lambda_1(D_B^{-1}A)} = \frac{1}{\lambda_1(D_B^{-1/2} AD_B^{-1/2})}, \tag{12}
\]

expressing the dependency of virus 1 survival threshold \( \tau_{1c} \) on effective infection rate of virus 2 \( \tau_2 \) for large values of \( \tau_2 \). This expression (12) directly highlights the influence of interrelations of the two layers. Significantly, if \( \lambda_1(D_B^{-1}A) \) is large, expression (10) suggests that virus 1 survival threshold does not increase significantly by virus 2 infection rate. Similar arguments about interpretation of (10) apply to aggressive competitive viruses where \( \tau_1 \) and \( \tau_2 \) are relatively large. The main difference in case of aggressive competitive spreading is that node degree is the determinant of centrality. From (12), the die-out threshold curve \( \Phi_1(\tau_2) \) asymptotically becomes

\[
\Phi_1(\tau_2) \simeq \frac{1}{\lambda_1(D_B^{-1}A)} \tau_2. \tag{13}
\]
for aggressive competitive propagation. FIG. 3 depicts survival threshold curves for non-aggressive (left) and aggressive (right) competitive spreading.

We prove conditions for coexistence by showing there is overlapping between regions where viruses survive.

**Theorem 1** In $SI_1SI_2S$ model \( \{12\} \) for competitive epidemics over multi-layer networks, if the two network layers $G_A$ and $G_B$ are identical, coexistence is impossible, i.e., a virus with even a slightly larger effective infection rate dominates and completely removes the other virus. Otherwise, if node-degree vectors of $G_A$ and $G_B$ are not parallel, i.e., $d_A \neq c_B$, or dominant eigenvectors of $G_A$ and $G_B$ do not completely overlap, i.e., $v_A \neq v_B$ the multi-layer structure of the underlying topology allows a nontrivial coexistence region.

**Proof.** If $G_A = G_B$, then equation \( \{1\} \) suggests $\tau_{c1} = \tau_2$ solves threshold equation \( \{6\} \). Similarly $\tau_{c2} = \tau_1$, suggesting $\tau_{2c} = \tau_2$ according to \( \{0\} \), i.e., survival and winning thresholds coincide. Therefore, the virus with even a slightly larger effective infection rate dominates and completely removes the other virus if the two network layers are identical.

In order to show possibility of coexistence for non-aggressive competitive viruses, we show the survival regions overlap by proving

$$\frac{d\tau_{1c}}{d\tau_2} \bigg|_{(\tau_1, \tau_2) = (\frac{1}{\lambda(A)}, \frac{1}{\lambda(B)})} \leq 1. \quad \{14\}$$

Using expression \( \{10\} \) and its counterpart for $\frac{d\tau_{2c}}{d\tau_1}$ (see Appendix), we find condition \( \{14\} \) is always true except for the special case where dominant eigenvectors of $G_A$ and $G_B$ completely overlap, i.e., $v_A = v_B$.

In order to show possibility of coexistence for aggressive competitive viruses, we show the survival regions overlap by proving

$$\frac{\tau_{1c}}{\tau_2} \bigg|_{\tau_2 \to \infty} \times \frac{\tau_{2c}}{\tau_1} \bigg|_{\tau_1 \to \infty} < 1. \quad \{15\}$$

Using expression \( \{12\} \) and its counterpart for $\frac{d\tau_{2c}}{d\tau_1}$ (see Appendix), we find that condition \( \{15\} \) is always true except for the special case where node-degree vectors of $G_A$ and $G_B$ are parallel, i.e., $d_A = c_B$.

When dominant eigenvectors of $G_A$ and $G_B$ are not identical, condition \( \{14\} \) indicates non-aggressive viruses can coexist. When propagation of competitive viruses is aggressive, condition \( \{15\} \) indicates viruses can coexist if node-degree vectors of $G_A$ and $G_B$ are not parallel. However, the rare scenario where $G_A$ and $G_B$ are not identical and $d_A = c_B$ and $v_A = v_B$ hold simultaneously demands further exploration.

The above theorem and equations \( \{10\} \) and \( \{12\} \) prove the importance of interrelation of network layers. As will be discussed in the simulation section, one approach capturing only the effect of interrelation is generating multilayer networks from two graphs $G_A$ and $G_B$ through simple relabeling vertices of $G_B$. We thus have a set of multilayer networks whose layers have identical graph properties but correspondence of nodes in one layer to the nodes of the other varies.

In the context of competitive spreading, whether memes, opinions, or products, the population under study serves as the ‘resource’ for the competitive entities, relating nicely to the concept of ‘competing species’ in ecology. Long-term study of competing species in ecology centers on the ‘competitive exclusion principle’ \[17\]: Two species competing for the same resources cannot coexist indefinitely under identical ecological factors. The species with the slightest advantage or edge over another will dominate eventually. Our $SI_1SI_2S$ model also predicts when the network layers are identical, coexistence is not possible. Significantly, different propagation routes break this ‘ecological symmetry,’ allowing coexistence. Not only have we rigorously proved a coexistence region, we quantitated this ecological asymmetry via interrela- tion of central nodes across the network layers. None or small overlapping of central nodes of each layer is the key determinant of coexistence. Excitingly, this conclusion nicely relates to ‘niche differentiation’ in ecology and yet is built upon network science rigor.

**C. Standardized Threshold Diagram and a Global Approximate Formula**

Exploring efficient characterization of threshold curves using extreme scenarios, we propose a standardized threshold diagram, where threshold curves are plotted in a $[0, 1] \times [0, 1]$ plane for $(x, y) = \left( \frac{1}{\lambda_1(1/2)}, \frac{1}{\lambda_1(1/2)} \right)$. axes
scaled by layer spectral radius and inverted. Curves in standardized threshold diagram start from origin to point \((1, 1)\). From (11) and (12) the slopes of the survival curve of virus 1 at \((0, 0)\) and \((1, 1)\) are

\[
\begin{align*}
m_0 &= \frac{\lambda_1(B)}{\lambda_1(A)} \lambda_1(D_B^{-1} A), \\
m_1 &= \frac{\sum v_{A,i}^2 v_{B,i}}{\sum v_{B,i}},
\end{align*}
\]

respectively. Importantly, these slopes help creating a parametric approximation for the survival threshold curve \(\tau_1 = \Phi_1(\tau_2)\) for the full range of \(\tau_2\). We use a quadratic Bezier curve

\[
\begin{bmatrix} x \\ y \end{bmatrix} = 2\sigma(1 - \sigma) \begin{bmatrix} a \\ b \end{bmatrix} + \sigma^2 \begin{bmatrix} 1 \\ 1 \end{bmatrix},
\]

connecting \((x, y) = (0, 0)\) to \((x, y) = (1, 1)\) for \(\sigma \in [0, 1]\), and satisfying the slope constraints (10) and (17), if \(a\) and \(b\) are chosen as:

\[
a = \frac{1 - m_1}{m_0 - m_1}, \quad b = \frac{m_0(1 - m_1)}{m_0 - m_1}.
\]

Therefore, the Bezier curve (18) approximates the standardized threshold curve diagram for the whole range of \(\tau_1 > 1/\lambda_1(A)\) and \(\tau_2 > 1/\lambda_1(B)\) using only spectral information of a set of matrices.

D. Multi-layer Network Metric for Competitive Spreading

Proving coexistence is one of the key contributions of this paper. We go further to define a topological index \(\Gamma_s(G)\) quantifying possibility of coexistence in a multi-layer network \(G = (V, E_A, E_B)\) for the case of non-aggressive spreading as

\[
\Gamma_s(G) = 1 - \frac{\left(\sum v_{B,i}^2 v_{A,i}\right)\left(\sum v_{A,i} v_{B,i}^2\right)}{\left(\sum v_{B,i}\right)\left(\sum v_{A,i}\right)}.
\]

Values of \(\Gamma_s(G)\) vary from 0 (corresponding to the case where \(v_A = v_B\)) to 1. Values of \(\Gamma_s(G)\) close to zero imply coexistence is rare and any survived virus is indeed the absolute winner. \(\Gamma_s(G)\) closer to 1 indicates coexistence is very possible on \(G\). Therefore, \(\Gamma_s(G)\) can be used to discuss coexistence of non-aggressive competitive viruses.

Similar to non-aggressive competitive spreading, we can define a topological index \(\Gamma_l(G)\) to quantify coexistence possibility in a multi-layer network \(G = (V, E_A, E_B)\) as

\[
\Gamma_l(G) = 1 - \frac{1}{\lambda_1(D_B^{-1} A)} - \frac{1}{\lambda_1(D_A^{-1} B)}.
\]

Values of \(\Gamma_l(G)\) vary from 0 (corresponding to the case where \(d_A = \varepsilon d_B\)) to 1. Values of \(\Gamma_l(G)\) close to zero imply coexistence is rare and any survived virus is indeed the absolute winner. \(\Gamma_l(G)\) closer to 1 indicates coexistence is very possible on \(G\). Therefore, \(\Gamma_l(G)\) can be used to discuss coexistence of aggressive competitive viruses.

E. Numerical Simulations

Multi-layer network generation: Our objective for numerical simulations is not only to test our analytical formulation, but also to investigate our prediction of cross-layer interrelation effect on competitive epidemics. This demands a set of two-layer networks for which isolated layers have identical graph properties but how these layers are interrelated is different, hence capturing the pure effect of interrelation. Specifically, in the following numerical simulations, the contact network \(G_A\) through which virus 1 propagates is a random geometric graph with \(N = 1000\) nodes, where pairs less than \(r_c = \sqrt{\frac{3\log(N)}{\pi N}}\) apart connect to ensure connectivity. For the contact graph of virus 2 \((G_B)\), we first generated a scale-free network according to the Barabási–Albert model. We then used a randomized greedy algorithm to associate the nodes of this graph with the nodes of \(G_A\), approaching a certain degree correlation coefficient \(\rho\) with \(G_A\), i.e., each iteration step permutes nodes when the degree correlation coefficient

\[
\rho(G) = \frac{\sum(d_{A,i} - \bar{d}_A)(d_{B,i} - \bar{d}_B)}{\sqrt{\sum(d_{A,i} - \bar{d}_A)^2} \sqrt{\sum(d_{B,i} - \bar{d}_B)^2}}
\]

is closer to the desired value. Specifically, we obtained three different permutations where the generated graphs are negatively \((\rho = -0.47)\), neutrally \((\rho = 0)\), and positively \((\rho = 0.48)\) correlated with \(G_A\). These three graphs have identical graph properties, yet they are distinct respective to \(G_A\). FIG. 4 depicts a graph \(G_A\) and three graphs of \(G_B\) with \(N = 100\) nodes to improve conceptualization.

Steady-state infection fraction: When the spreading of a single virus is modeled as SIS, the steady-state infection fraction \(\bar{p}_s\) illustrates a threshold phenomena respective to effective infection rates: steady-state infection fraction \(\bar{p}_s\) is zero for effective infection rates less than a critical value but becomes positive for larger values. When two viruses compete to spread, steady state infection fraction \(\bar{p}_s\) in the SI1SI2S model exhibits a threshold behavior at \(\tau_1 = \tau_1c\), for a given \(\tau_2\). FIG. 5 depicts the steady state infection fraction curve of virus 1 in the SI1SI2S competitive spreading model. In this simulation, effective infection rate of virus 2 is fixed at \(\tau_2 = 6\frac{1}{\lambda_1(B)}\) and \(G_B\) is positively correlated with \(G_A\) \((\rho = 0.48)\). In order to obtain a unified form, we normalized the horizontal axis to \(\tau_1 = \lambda_1(A)\). The steady state infection fraction of virus 1, \(\bar{p}_s\), is zero for \(\tau_1 \leq \tau_1c \simeq 3\frac{1}{\lambda_1(A)}\), identifying this range as an extinction region for virus 1, while \(\bar{p}_s\) is positive for \(\tau_1 > \tau_1c\) indicating survival of virus 1. Interestingly, aside from the survival threshold \(\tau_1c\), the winning threshold \(\tau_1^w\) appears in the figure when plotted against a single virus case: \(\bar{p}_s\) takes the same values as the single virus case for effective infection rates larger than the winning threshold \(\tau_1^w\). For example FIG. 5 shows \(\bar{p}_s\) in the competitive sce-
The contact network $G_A$ through which virus 1 propagates is a random geometric graph where pairs of nodes with a distance less than $r_c$ are connected to each other. For visualization convenience, the number of nodes is $N = 1000$, which is different from the actual $N = 1000$ used for numerical simulation results. For the contact graph of virus 2 ($G_B$), we first generated a scale-free network according to the B-A model, associating the nodes of this graph with the nodes of $G_A$ to achieve a certain degree correlation coefficient with $G_A$. Specifically, we obtained three different permutations such that the generated graphs are negatively, neutrally, and positively correlated with $G_A$. These three graphs are the same if isolate, and distinct in their interrelation with $G_A$. The high degree nodes in the positively correlated $G_B$ (lower right) have also high degree in $G_A$ (upper left), while the high degree nodes in the negatively correlated $G_B$ (upper right) have low degree size in $G_A$. The uncorrelated $G_B$ (lower left) shows no clear association.

\[ \bar{p}_{1}^{ss} = \frac{1}{\lambda_{1}(A)} \]

The steady-state infection fraction of virus 1 ($\bar{p}_{1}^{ss}$) is zero for $\tau_1 \leq \tau_{1c} \approx 0.55$, an extinction region for virus 1. Interestingly, for $\tau_1 > 1\times \lambda_{1}(A)$, $\bar{p}_{1}^{ss}$ for the competitive scenario (red curve) is identical to the case of single virus propagation (black curve), suggesting extinction of virus 2, hence marking this region as the winning range for virus 1. For $\tau_1 \in (\tau_{1c}, 1\times \lambda_{1}(A))$, virus 1 and virus 2 both persist in the population, marking this range as the coexistence region.

The contact network $G_B$ is positively correlated with that of virus 1 ($G_A$), it is more difficult for virus 1 to survive, making the survival threshold $\tau_{1c}$ relatively larger for positively correlated $G_B$. Negatively correlated contact network layers impede virus 1 from completely suppressing virus 2, making winning threshold $\tau_1^*$ larger for negatively correlated $G_B$. Hence, this region is identified as the absolute winning range for virus 1. For $\tau_1 \in (\tau_{1c}, 1\times \lambda_{1}(A))$, virus 1 and virus 2 persist in the population, marking this range as the coexistence region.

**FIG. 4:** Two-layer network generation for numerical simulations is generated here. The contact network $G_A$ through which virus 1 propagates is a random geometric graph where pairs of nodes with a distance less than $r_c$ are connected to each other. For visualization convenience, the number of nodes is $N = 1000$, which is different from the actual $N = 1000$ used for numerical simulation results. For the contact graph of virus 2 ($G_B$), we first generated a scale-free network according to the B-A model, associating the nodes of this graph with the nodes of $G_A$ to achieve a certain degree correlation coefficient with $G_A$. Specifically, we obtained three different permutations such that the generated graphs are negatively, neutrally, and positively correlated with $G_A$. These three graphs are the same if isolate, and distinct in their interrelation with $G_A$. The high degree nodes in the positively correlated $G_B$ (lower right) have also high degree in $G_A$ (upper left), while the high degree nodes in the negatively correlated $G_B$ (upper right) have low degree size in $G_A$. The uncorrelated $G_B$ (lower left) shows no clear association.

**FIG. 5:** Steady state infection fraction curve of virus 1 in the $S_I S_I S_S$ competing spreading model (red). While increasing $\tau_1$, steady state infection fraction of virus 1 in the the $S_I S_I S_S$ model becomes nonzero at the survival threshold $\tau_{1c}$, while it coincides with that of the SIS model (black curve) at the winning threshold $\tau_1^*$. In this simulation, the steady-state infection fraction of virus 1 ($\bar{p}_{1}^{ss}$) is zero for $\tau_1 \leq \tau_{1c} \approx 3.0$, an extinction region for virus 1. Interestingly, for $\tau_1 > 1\times \lambda_{1}(A)$, $\bar{p}_{1}^{ss}$ for the competitive scenario (red curve) is identical to the case of single virus propagation (black curve), suggesting extinction of virus 2, hence marking this region as the winning range for virus 1. For $\tau_1 \in (\tau_{1c}, 1\times \lambda_{1}(A))$, virus 1 and virus 2 both persist in the population, marking this range for coexistence region.

**FIG. 6:** Comparison of steady-state infection fraction curves of virus 1 in the $S_I S_I S_S$ competitive spreading model. Survival threshold $\tau_{1c}$ is larger for positively correlated $G_B$, indicating it is more difficult to survive positively correlated $G_B$, while $\tau_1^*$ is larger for negatively correlated $G_B$, indicating it is more difficult to completely suppress the other virus in negatively correlated $G_B$. 

This page contains diagrams illustrating the relationship between the contact network layers and virus propagation, along with mathematical expressions for the steady-state infection fraction curves.
Analytical approximation formula (18) finds the threshold survival regions.

White curves represent theoretical threshold curves derived from the solution to (18). Standardized threshold diagram shows three survival regions: mutual extinction region I, where only virus 1 survives and virus 2 dies out, mutual extinction region II, where only virus 2 survives and virus 1 dies out, and finally coexistence region III, where both viruses survive and persist in the population.

Survival diagram: Allowing variation of $\tau_2$, the steady-state infection curve extends to the steady-state infection surface. FIG. 7 plots steady-state infection fraction for virus 1 and virus 2 as a function of $\tau_1$ and $\tau_2$. White curves represent theoretical threshold curves derived from the solution to (18), accurately separating the survival regions.

FIG. 8 plots standardized threshold diagram where $G_B$ is negatively correlated with $G_A$ (left) and the case where $G_B$ is positively correlated with $G_A$ (right). Dashed lines are the predictions from analytical approximation formula explicitly expressed in (18). Standardized threshold diagram shows three survival regions: mutual extinction region I, where only virus 1 survives and virus 2 dies out, mutual extinction region II, where only virus 2 survives and virus 1 dies out, and finally coexistence region III, where both viruses survive and persist in the population.

IV. DISCUSSION AND CONCLUSION

Competitive multi-virus propagation shows very rich behaviors, beyond those of single virus propagation. This type of modeling is suitable for co-propagation of exclusive entities, for example, opposing opinions about a subject, where people are for, against, or neutral; spreading of a disease through physical contact and viral propagation of antidote providing absolute immunity to the disease, or marketing penetration of competitive products like Android versus Apple smart phones. Aside from its potential applications, the problem of competitive spreading over multilayer networks is technically challenging. In particular, compared to single layer networks, science of multilayer networks is still in its infancy. There are yet numerous unknowns about this complex problem.

In this paper, we study $SI_1SI_2S$ model, the simplest extension of $SIS$ model to competitive spreading over a two-layer network, focusing on long-term behaviors in relation to multilayer network topology. In brief, the major contributions of this paper are: (a) identification and quantification of extinction, coexistence, and mutual exclusion via defining survival thresholds and winning thresholds, (b) proving a region of coexistence and quantitating it through overlapping of layers central nodes, (c) developing an explicit approximation formula to globally find threshold values, and (d) proposing a novel multilayer network generation scheme to capture influence of layers interrelation. We believe our methodology has great potentials for application to broader classes of multi-pathogen spreading over multi-layer and interconnected networks.

Acknowledgement: This work was partially supported by National Science Foundation under Award DMS-1201427. Any opinions, findings, and conclusions or recommendations expressed in this paper are those of the authors and do not necessarily reflect the views of the National Science Foundation.

Appendix: Selected Proofs

1. Derivation of Eigenvalue Perturbation Formulae

Here, we detail the derivations of (10) and (12).

At $\tau_2 = 1/\lambda_1(B)$, (7) finds $y_i = 0$ for all nodes. Equation (7) is indeed the steady state equation for infection probabilities in NIMFA model. Van Mieghem [14] found for SIS model the derivative with respect to effective infection rate, suggesting

$$\frac{dy_i}{d\tau_2} = cBv_{B,i},$$

$$w_i = c_A v_{A,i},$$

where

$$c_A = \frac{\lambda_1(A)}{\sum_i v_{A,i}} , \quad c_B = \frac{\lambda_1(B)}{\sum_i v_{B,i}},$$

where $v_A$ and $v_B$ are the normalized dominant eigenvectors of $A$ and $B$, respectively.
Differentiating \( \frac{dw_i}{dt} \) with respect to \( \tau_2 \) yields:

\[
\frac{dw_i}{d\tau_2} = \frac{d\tau_{1c}}{d\tau_2}(1 - y_i) \sum a_{ij}w_j + \tau_{1c}(-\frac{dy_i}{d\tau_2}) \sum a_{ij}w_j + \tau_{1c}(1 - y_i) \sum \frac{dw_j}{d\tau_2}.
\]

Inserting \( \tau_{1c} = 1/\lambda_1(A) \), \( w_i = c_AV_{A,i} \), \( y_i = 0 \), and \( dy_i/d\tau_2 = c_Bv_{B,i} \), the above equation changes to:

\[
(I - \frac{1}{\lambda_1(A)}A) \frac{dw}{d\tau_2} = (\frac{\tau_{1c}}{d\tau_2})\lambda_1(A)c_AV_A - c_Bc_A(v_B \circ v_A)
\]

in the collective form, where the Hadamard product \( \circ \) acts entry-wise. Multiplying both sides by \( v_i^T \) from left yields:

\[
\frac{d\tau_{1c}}{d\tau_2}|_{\tau_2 = \frac{1}{\lambda_1(A)}} = \frac{1}{\lambda_1(A)}c_Bv_{A}^T(v_B \circ v_A)
\]

\[
= \lambda_1(B) \sum v_{B,i}^2v_{B,i} \quad (A.6)
\]

obtaining (10). Finding \( \frac{d\tau_{1c}}{d\tau_2} \) at \( \tau_2 = 1/\lambda_1(B) \) obtains the dependence of \( \tau_{1c} \) on \( \tau_2 \) close to \( 1/\lambda_1(B) \).

Replacing for \( 1 - y_i = \frac{\tau_i}{\tau_2} + \sum b_{ij}y_j \) from (7) into (6) yields

\[
w_i = \frac{\tau_{1c}}{\tau_2}(\frac{1}{\tau_2 + \sum b_{ij}y_j}) \sum a_{ij}w_j.
\]

When effective infection rate \( \tau_2 \) is enormous \( \tau_2^{-1} \rightarrow 0 \) and \( y_1 \rightarrow 1 \), suggesting

\[
w_i = (\frac{\tau_{1c}}{\tau_2}) \sum a_{ij}w_j.
\]

where \( d_{B,i} \) is the \( B \)--degree of node \( i \). Therefore, \( \frac{dw_i}{d\tau_2} \rightarrow \infty \) for large values of \( \tau_2 \).

### 2. Coexistence Proofs

**Coexistent region non-aggressive competitive viruses:**

To investigate the coexistence region for non-aggressive viruses we show that (14) is true. From (10), we find

\[
\frac{d\tau_{1c}}{d\tau_2} = \frac{d\tau_{2c}}{d\tau_1} = \frac{(\sum v_{B,i}v_{A,i}^2)(\sum v_{A,i}v_{B,i}^2)}{(\sum v_{B,i}^2)(\sum v_{A,i}^2)}
\]

From Hölder’s inequality

\[
\sum v_{B,i}v_{A,i}^2 \leq \left( \sum v_{B,i}^3 \right)^{1/3} \left( \sum v_{A,i}^3 \right)^{2/3}
\]

\[
= \left( \sum v_{B,i}^3 \right)^{1/3} \left( \sum v_{A,i}^3 \right)^{2/3},
\]

and the equality happens iff \( v_A = v_B \). Similarly,

\[
\sum v_{A,i}v_{B,i}^2 \leq \left( \sum v_{B,i}^3 \right)^{2/3} \left( \sum v_{A,i}^3 \right)^{1/3}.
\]

Multiplying sides of (A.10) and (A.11) yields

\[
(\sum v_{B,i}v_{A,i}^2)(\sum v_{A,i}v_{B,i}^2) \leq \left( \sum v_{B,i}^3 \right)(\sum v_{A,i}^3),
\]

proving (A.9) is true.

**Coexistent region for aggressive competitive viruses:**

To investigate the coexistence region for non-aggressive viruses we shown that (14) is true. Substituting from (12) yields

\[
\frac{\tau_{1c}}{\tau_2} \rightarrow \infty \Rightarrow \frac{\tau_{2c}}{\tau_1} \rightarrow \infty = \frac{1}{\lambda_1(D_B^{-1}A)} \cdot \frac{1}{\lambda_1(D_A^{-1}B)}
\]

\[
= \frac{1}{\lambda_1(D_B^{-1}A \circ A^{-1}B)}
\]

\[
= \frac{1}{\lambda_1([(D_B^{-1} \circ A^{-1}B)(A \circ B)]}
\]

\[
= \frac{1}{\lambda_1[(D_B \circ D_A)^{-1}(A \circ B)]},
\]

according to properties of Kronecker product.

The degree diagonal matrix of \( (A \circ B) \) is \( (D_A \circ D_B) \). Therefore, \( (D_B \circ D_A) \) is a diagonal permutation of the degree diagonal matrix of \( (A \circ B) \). According to Lemma 1, presented in the following, \( \lambda_1[(D_B \circ D_A)^{-1}(A \circ B)] \geq 1 \), thus

\[
\frac{\tau_{1c}}{\tau_2} \rightarrow \infty \Rightarrow \frac{\tau_{2c}}{\tau_1} \rightarrow \infty \leq 1,
\]

and equality holds only if \( D_B \otimes D_A = D_A \otimes D_B \), which holds only if ratio of \( B \)--degree and \( A \)--degree of each node is same for all nodes.

**Lemma 1** If \( H = \pi(D_C)^{-1}C \), where \( \pi(D_C) \) is a diagonal permutation of degree diagonal matrix of symmetric matrix \( C \), then \( \lambda_1(H) \geq 1 \). Furthermore, equality holds only if \( \pi(D_C) = D_C \).

**Proof.** The largest eigenvalue maximizes Rayleigh quotient, therefore,

\[
\lambda_1(H) = \lambda_1(\pi(D_C)^{-1}C) = \lambda_1(\pi(D_C)^{-1/2}C\pi(D_C)^{-1/2})
\]

\[
= \max_x \frac{x^T(\pi(D_C)^{-1/2}C\pi(D_C)^{-1/2}x)}{x^Tx} \geq \frac{1^TC1}{1^T\pi(D_C)1} = \frac{\sum d_{C,i}}{\sum c_{D,i}} = 1,
\]

where \( d_{C,i} \) is the degree of node \( i \) map. Therefore, \( \lambda_1(H) \geq 1 \). Equality holds only if \( x = \pi(D_C)^{-1/2}1 \) is the dominant eigenvector of \( \pi(D_C)^{-1/2}C\pi(D_C)^{-1/2} \), i.e., \( \pi(D_C)^{-1/2}C1 = \pi(D_C)^{1/2}1 \), which only holds if \( d_{C,i} = d_{C,i} \).
3. Steady State Numerical Solution

Given \( \tau_2 > 1/\lambda_1(B) \), (6) and (7) numerically find \( \tau_{1,c} \). We now define \( x_i \triangleq \frac{y_i}{1 + y_i} \), given the recursive iteration law:

\[
x_i(k + 1) = \tau_2 \sum b_{ij} \frac{x_j(k)}{1 + x_j(k)} \tag{A.15}
\]

to prove they converge exponentially, numerically solving (7) as \( \frac{x_i(k)}{1 + x_i(k)} \to y_i \). The main advantage of finding equilibrium values using recursive law (A.15) instead of solving ordinary differential equations of the model is recursive law (A.15) does not require incremental time increase, making computations drastically faster.

Furthermore, the steady-state infection probabilities in (3)-(4) can be found via the recursive iteration law:

\[
x_i(k + 1) = \tau_1 \sum a_{ij} \frac{x_j(k)}{1 + x_j(k) + z_j(k)}, \tag{A.16}
\]
\[
z_i(k + 1) = \tau_2 \sum b_{ij} \frac{z_j(k)}{1 + x_j(k) + z_j(k)}, \tag{A.17}
\]
as \( \frac{x_i(k)}{1 + x_i(k) + z_i(k)} \to p_{1,i}^* \) and \( \frac{z_i(k)}{1 + x_i(k) + z_i(k)} \to p_{2,i}^* \).

[1] B. Karrer and M. Newman, Physical Review E, 84(3), 036106 (2011). Competing epidemics on complex networks.
[2] S. Funk and V. A. Jansen, Physical Review E, 81(3), 036118 (2010). Interacting epidemics on overlay networks.
[3] Y.-Y. Ahn, H. Jeong, N. Masuda, and J. D. Noh, Physical Review E, 74(6), 066113 (2006). Epidemic dynamics of two species of interacting particles on scale-free networks.
[4] X. Wei, N. C. Valler, B. A. Prakash, I. Neamtiu, M. Faloutsos, and C. Faloutsos, Selected Areas in Communications, IEEE Journal on, 31(6), 1049–1060 (2013). Competing memes propagation on networks: A network science perspective.
[5] S. Aral and D. Walker, Management Science, 57(9), 1623–1639 (2011). Creating social contagion through viral product design: A randomized trial of peer influence in networks.
[6] L. Weng, A. Flammini, A. Vespignani, and F. Menczer, Scientific Reports, 2 (2012). Competition among memes in a world with limited attention.
[7] S. Shrestha, A. A. King, and P. Rohani, PLoS computational biology, 7(8), e1002135 (2011). Statistical inference for multi-pathogen systems.
[8] M. Newman and C. Ferrario, arXiv preprint arXiv:1305.4648 (2013). Interacting epidemics and coinfection on contact networks.
[9] C. Granell, S. Gomez, and A. Arenas, arXiv preprint arXiv:1306.4136 (2013). On the dynamical interplay between awareness and epidemic spreading in multiplex networks.
[10] M. E. Newman, Physical review letters, 95(10), 108701 (2005). Threshold effects for two pathogens spreading on a network.
[11] Q. Wu, M. Small, and H. Liu, Journal of Nonlinear Science, 23(1), 113–127 (2013). Superinfection behaviors on scale-free networks with competing strains.
[12] Y. Wang, G. Xiao, and J. Liu, New Journal of Physics, 14(1), 013015 (2012). Dynamics of competing ideas in complex social systems.
[13] X. Wei, N. Valler, B. A. Prakash, I. Neamtiu, M. Faloutsos, and C. Faloutsos, ACM SIGCOMM Computer Communication Review, 42(5), 5–12 (2012). Competing memes propagation on networks: a case study of composite networks.
[14] P. Van Mieghem, J. Omic, and R. Kooij, IEEE/ACM Transactions on Networking, 17(1), 1–14 (2009). Virus spread in networks.
[15] A. Ganesh, L. Massoulié, and D. Towsley in Proceedings IEEE INFOCOM, Vol. 2, pages 1455–1466, 2005.
[16] F. D. Sahneh, C. Scoglio, and P. Van Mieghem, IEEE/ACM Transaction on Networks, to appear (2013). Generalized epidemic mean-field model for spreading processes over multi-layer complex networks.
[17] G. Hardin et al., Science, 131(4400), 1292–1297 (1960). The competitive exclusion principle.