Superinfection in networks

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***Abstract*—We introduce an extension of the SIS epidemic model that describes infection, mutation and curing for a whole hierarchy of viruses, resembling a nested spreading process. In our model, high level viruses are only allowed to spread to nodes that have acquired a lower level of infection before. The simplest case of two viruses, in which one "superinfects" the other, shows already rich dynamics that are difficult to predict by common mean-field approximation techniques in certain cases. We derive an exact Markovian description for superinfection in the complete network and the star network showing that the steady state of the epidemic process is highly sensitive to the spreading rate of both viruses. Taking the spreading rates into account, we outline conditions for epidemic outbreaks, coexistence of both viruses and extinction cycles.**

I. Introduction

Epidemic spreading in networks is a process that models the propagation of information, the spread of rumors, the contagion with diseases and similar phenomena. SIS-like models [1], [15] are amenable to analytical understanding and enable us to describe and study effects like endemic outbreaks and extinction of viruses. We interpret a virus as a mere metaphor for the entity or property that is transferred from one node to others in the network.

Superinfection is a medically inspired term that originally described the process of infecting an already infected individual with a second, usually more severe virus. This can be due to a weakness of the immune system caused by the first virus that allows for the infection of the second virus, which in return will dominate the first one. Hence, superinfection can be regarded as an interaction of competing viruses, in which the second virus is dominant, but also strongly dependent on the (pre)-infection of the first virus.

The interactions between HIV and the Herpex Simplex Virus type 2 (HSV-2) are a well known medical example of superinfection: Acquisition of HSV-2 significantly increases the chances of getting superinfected with HIV and to transmit HIV to others [22].

Apart from the medical context, we see superinfection as an embedded epidemic spreading process. This view is applicable if two conditions hold: first, there needs to be a pre-condition for the infection with the dominant virus and second, the pre-condition itself spreads like a virus.

We believe that several applications can be better understood if described as a superinfection. One example is marketing: The knowledge about features (or the mere existence) of a product is a pre-condition for people to make a buying decision. This knowledge spreads via advertisement in social networks, increasing brand-awareness, but not necessarily inducing a buying decision for each infected individual. However, once several persons buy the actual product, first-hand experiences start spreading via self-written reviews over social networks as well. A possible consequence is that people predisposed by advertisement will now make a buying decision and start to actively promote the product as well.

A different use case is observed in cybersecurity, in which a security vulnerability is the precondition to acquire a piece of malware like a computer worm. Sometimes, even a computer worm can be a regarded as a precondition to become infected by another worm. For example, the Blaster computer worm [2] was able to spread in late 2003 to over 100.000 Windows machines, where it caused significant damage. Shortly after Blaster, another computer worm named "Welchia" [3] appeared. It spread by using the same system vulnerability as Blaster, but was designed to eliminate Blaster and then to patch the whole system. While most-likely released with best intentions, Welchia ultimately might have caused more damage than Blaster, which shows how easy the interactions of computer worms are misjudged.

While Welchia was not a full success, the general idea of *active defense* provides a promising alternative to traditional countermeasures that rely on classical anti-virus software only. Instead of deploying a resource-hungry scanner on each machine on the network, a network operator might rather design an *antiworm* as an epidemic control unit, which allows the removal of undesired software. This worm would only spread to infected machines and would eventually restore these systems back to a healthy state once protection is no longer needed.

In order to realize these applications, our work introduces the fundamental theory of superinfection by the use of epidemic models. After discussing related work in Section II, we describe the standard SIS-model and our model of superinfection in Section III. In Section IV, we show that the interaction of 2 viruses is already so complex that standard analysis techniques like mean-field approximation produce deviations. In order to understand the interactions between the viruses and their conditions for spreading and survival, we analyze the exact Markov process for small complete networks and small star networks in Section V in order to conclude in Section VI.

II. Related Work

Nowak and May are among the first authors who focused on modeling superinfection in their pioneering work [14].

Similar to our work, a virus hierarchy is introduced in which stronger viruses dominate less virulent strains. In particular, only the strongest virus is considered active, which means that it is the only virus that spreads. In contrast to our work, mixed populations are assumed and no underlying contact network is taken into account.

The case where multiple viruses are active is known as *coinfection* and was also explored and modeled by Nowak and May [9]. Mosquera et al. [11] show that superinfection can be a limit case for a coinfection process and give conditions for coexistence of multiple viruses. Multiple other works exist that investigate the existence and interaction of multiple viruses in the setting of competing viruses [5], [6], [18], [21], in which infection with one virus provides immunity to the other (*cross-immunity*).

Newman and Ferrario [13] study an SIR-like epidemic model, where the infection with one virus is a prerequisite for the infection with a second virus. The authors use a general configuration network model and evaluate their spreading process on two examples: one network with a Poisson degree distribution and one with a power-law degree distribution. Their model is different from ours as they consider a sequential process, in which both viruses spread at well separated times rather than in parallel. Also, our work is a generalization of the SIS-model and not of the SIR-model.

Wu et al. [28] study a different superinfection model by means of linear stability analysis and extensive computer simulations on networks with power-law degree distributions. Conditions for coexistence in terms of the reproductive numbers of both viruses are given.

Superinfection has been used as a feature in many models in the field of computational biology. Prado et al. [17] show - based on computer simulations - how coevolutionary cycles between pathogen virulence and sociality for hosts in contact networks are influenced by the possibility of superinfection. Leventhal et al. [8] show analytically and with simulations how the topology of an underlying contact network may impact the spread of competing viruses in the SIS-model. In their work, the second virus appears after the first virus has reached an endemic state, but can only spread to the subpopulation of still susceptible nodes, as they assume cross-immunity. Our work can be regarded as complementary, as we instead restrict the virus to spread only in the subpopulation of nodes already infected by the first virus.

A similar effect to superinfection appears in information diffusion processes between different pieces of information (contagions) that traverse the network. Myers and Leskovec [12] show that interaction between contagions can change their relative spreading properties, having a major impact on the diffusion process. Similar effects might trigger information cascades [7] and are observable as interacting waves in networks [10].

III. Modeling superinfection

This section is divided in two parts: First, we present the standard Markovian SIS-model of epidemic spreading and point out some of its properties. Second, we show how this model can be generalized to describe superinfection by introducing mutations and additional viruses.

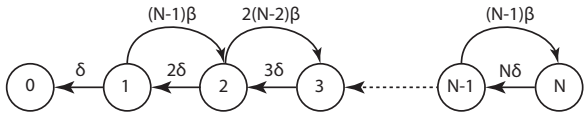


Fig. 1. Markov graph of the SIS-model for the complete network of N nodes. Each vertex here represents one possible state of the Markov chain and is labeled with the total number of infected nodes of the underlying network. Edges represent the transition rates between neighboring states. State 0 is an absorbing state, as there are no new infections possible once all nodes are cured.

A. The SIS-model

The standard SIS-model is a Markov chain in which each node in a network can be in two possible compartments: I for *infected* or S for *susceptible* (to infection). Two underlying Poisson processes govern the transition between these compartments: the *curing process* is a nodal transition that changes the node's compartment from I to S (curing) with a fixed rate of δ. The *infection process* is a link based transition and changes the compartment of a node from S to I (infecting) with a fixed rate of β for each link of a susceptible node to an infected neighbor. Both rates β and δ are assumed to be fixed and global constants for the whole network. The fraction $\tau = \beta/\delta$ is called the *effective infection rate*.

Depending on τ, different behaviors of the SIS process are observed. A very low τ (for a specific topology) results in a relatively short survival time of the virus, as the average hitting time of the absorbing state of the Markov process is very low as well. However, there exists an epidemic threshold τ_c for which the virus becomes *endemic* and infects a constant fraction of nodes for a considerably longer period of time [15].

Given N nodes and two compartments, the network can be in 2^N different states, which define the *state space* of the Markov chain. The exponential growth of the state space makes an exact computation infeasible for larger networks. To deal with this issue, graph-automorphisms of the network can be used to "lump" certain states together (see Simon et al. [20]). This allows for polynomial sized representation for networks with symmetric infectious states like the complete network (shown in Figure 1) or the star network. However, this approach is not feasible for all networks. In order to analyze the epidemic process in these networks as well, mean-field approximations are frequently used, which we describe further in Section IV.

B. The superinfection SIkS-model

We propose a natural extension of the standard SIS-model which we call the SIkS-model. In this model, there exists a total number of k infection compartments $I_1, \ldots, I_k$ linked to viruses 1 to k, which constitute an *infection hierarchy*: S is only susceptible to infections by virus 1, nodes in compartment I_j are only susceptible to infections by the next higher virus $j+1$, for $j = 1, \ldots, k-1$. The infection rates $\beta_1, \ldots, \beta_k$ describe the rate at which these infections occur. See Figure 2a for an illustration of these link based transitions.

Depending on the current compartment, there are k curing rates $\delta_1, \ldots, \delta_k$ which are nodal transitions back to S. We also introduce nodal *mutation* with rates $\mu_1, \ldots, \mu_k$ as a second force of infection, which allows a node to switch to the next

higher infection level without exposure to the next higher virus by a neighbor. Figure 2b illustrates these node based transitions.

The SIkS-model reduces to the SIS-model for $k = 1$ and $\mu_1 = 0$ and to the ε-SIS-model [25], [26] (which is an SIS-model that allows for self-infection) for $\mu_1 = \varepsilon$ for some small $\varepsilon > 0$. The size of the state space for the SIkS-model is $(k+1)^N$.

In order to study the effect of superinfection, we confine ourselves to $k = 2$ for the remainder. In this case, the complete process is governed by 6 parameters, namely: $\beta_1, \beta_2, \delta_1, \delta_2, \mu_1$ and μ_2. We will occasionally call β_2 *superinfection rate*, in contrast to β_1 which we simply call *infection rate*.

Finding a succinct representation of the Markov chain for the complete network in the SIkS-model for $k = 2$ is more complex than for the SIS-model. As we have two different viruses in the network, we can no longer identify a state with the total number of infected nodes. Additionally, there exist new transitions due to the introduction of mutation. Figure 3 shows a succinct representation of these transitions in a Markov chain of size $\frac{1}{2}(N+1)(N+2)$, where N is the total number of nodes. The state space counts all possible combinations for the total number of infected nodes in the network per virus.

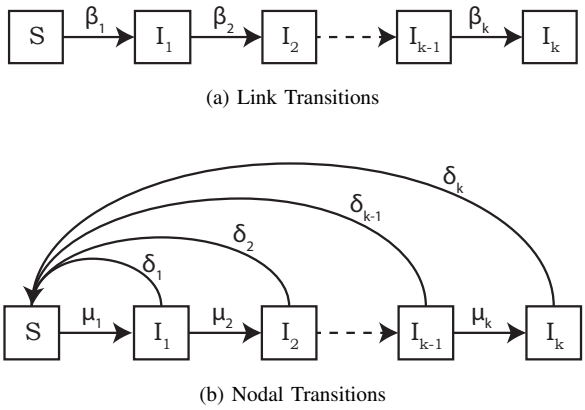
In the star network, the spread of viruses is dominated by the compartment of the center node. If the center node is in compartment S, mutation is the only way of infecting new leaf nodes, as the only neighbor of a each leaf node is susceptible. Similarly, the only way of virus 2 to spread towards leaf nodes is to infect the center node, which is only possible once it is in compartment I_1. The compartment of the center node is like a switch that determines which virus may currently spread towards leaf nodes, which are the majority of all nodes in the star network.

Hence, the state space of the Markov chain of the star network is partitioned into 3 groups according to the 3 possible compartments of the center node. For each group, the states are further ordered similarly to the complete network by all possible combinations of the number of infected leaf nodes by each virus. Figure 4 shows a succinct representation of all possible transitions in the star network. The size of the state space for this Markov chain is $\frac{3}{2}N(N+1)$, where N is the number of leaf nodes (thus the total number of nodes is $N+1$).

IV. Mean-Field Approximation

The exponentially growing state space of the Markov process imposes a hard challenge for the analysis of epidemics, especially for larger networks. In order to reduce the size of the governing equations, mean-field approximations are frequently applied in literature [1], [16], [23]. The use of mean-field approximations allowed for the discovery of some interesting results for the standard SIS-model, like a lower bound for the epidemic threshold by the inverse of the spectral radius of the adjacency matrix [27].

The recently introduced GEMF-model [19] is a generalized mean-field approximation of epidemic processes with multiple compartments in multilayer networks. We adopt the following GEMF-notations: each compartment is labelled by a number



(a) Link Transitions

(b) Nodal Transitions

Fig. 2. Transition rate graphs for the SIkS-model.

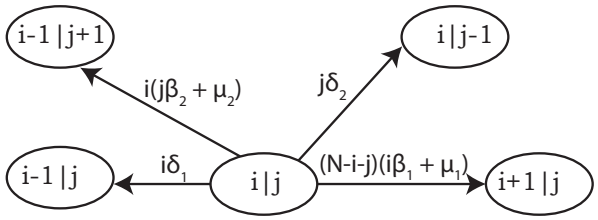


Fig. 3. Superinfection in the complete network: the state space of the Markov process consists of tuples (i, j), where i nodes are infected with virus 1 and j nodes are infected by virus 2, leaving $N - (i + j)$ nodes in compartment S. Shown are all the possible outgoing state transitions from state (i, j) with the according rates.

from 1 to M. The state of a node i at time t is $x_i(t) = e_m$, if i is in compartment m at time t. The vector e_m is a unit-vector, which is 0 at every position besides position m, where it is 1. As each entry of $x_i(t)$ is a Bernoulli random variable, the expected value of $x_i(t)$ is given by the compartment occupancy probability vector:

$$v_i(t) = E[x_i(t)] = [\Pr[x_i(t) = e_1], \ldots, \Pr[x_i(t) = e_M]]^T$$

We are particularly interested in the *average fraction of nodes* that belong to a compartment m. This is equivalent to $v_m(t)$ for any time t for the complete network.

In our model of superinfection, we have $M = 3$ compartments labelled with S, I_1, I_2. We use two layers[1] to describe the spreading process of the viruses. Each layer has an *influencer compartment*, which determines the spreading condition for the link based transitions. The influencer compartment of layer 1 is I_1, meaning that a node which has a neighbor in compartment I_1 on layer 1 undergoes a link transition with the specific rates in the transition matrix of layer 1, which we name A_{β_1}. Similarly, the influencer compartment of layer 2 is I_2, so any node with a neighbor in compartment I_2 on layer 2 undergoes a state transition with the specific rates in the transition matrix A_{β_2} of layer 2. In addition, there exists a third transition matrix A_δ, which describes all the nodal transitions and thus needs no influencer compartment. For our model of

[1]The GEMF-model allows for different contact networks on different layers, so it is possible to have two viruses that spread in the same population of nodes but over different links. In our case, both viruses use the same network.

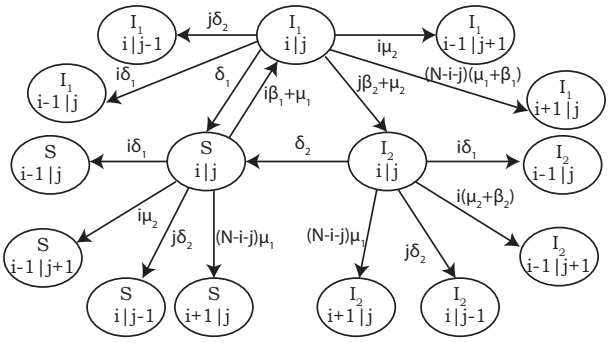


Fig. 4. Superinfection in the star network with N leaf nodes: the state space of the Markov process consists of triplets (C, i, j), where C describes the compartment of the central node, i the number of leaf nodes that are infected by virus 1 and j the number of leaf nodes that are infected by virus 2. Shown are all the possible outgoing state transitions from the states $(S, i, j), (I_1, i, j)$ and (I_2, i, j) with their corresponding rates.

the superinfection, these matrices are given by

$$A_{\beta_2} = \begin{bmatrix} 0 & 0 & 0 \\ 0 & 0 & \beta_2 \\ 0 & 0 & 0 \end{bmatrix}, \quad A_{\beta_1} = \begin{bmatrix} 0 & \beta_1 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

$$\text{and} \quad A_{\delta} = \begin{bmatrix} 0 & \mu_1 & 0 \\ \delta_1 & 0 & \mu_2 \\ \delta_2 & 0 & 0 \end{bmatrix}.$$

The GEMF-equations [19] are a set of non-linear differential equations that describe the behaviour of the epidemic over time for $i = 1, \ldots, N$:

$$\frac{dv_i}{dt} = -Q_{\delta}^T v_i - \sum_{l=1}^{L} \left(\sum_{j=1}^{N} (a_l)_{ij} v_{j,q_l} \right) Q_{\beta_l}^T v_i \qquad (1)$$

where L is the number of layers and A_l with elements $(a_l)_{ij}$ is the adjacency matrix of the contact network of layer l with q_l being the corresponding influencer compartment. The matrices Q_{δ}, Q_{β_1} and Q_{β_2} are the Laplacians matrices of the matrices A_{δ}, A_{β_1} and A_{β_2}, where in general the Laplacian matrix Q of a matrix A is defined as:

$$Q = \text{diag}\left(\sum_{i,j=1}^{N} (a_{ij}) \right) - A. \qquad (2)$$

Solving (1) gives a first order approximation for the average fraction of nodes in each compartment for the steady state of the underlying Markov process. For the complete network, the GEMF-equation reduces to the following:

$$\begin{aligned} \frac{d}{dt} \begin{bmatrix} v_S \\ v_{I_1} \\ v_{I_2} \end{bmatrix} = &- \begin{bmatrix} \mu_1 & -\delta_1 & -\delta_2 \\ -\mu_1 & \delta_1 + \mu_2 & 0 \\ 0 & -\mu_2 & \delta_2 \end{bmatrix} \begin{bmatrix} v_S \\ v_{I_1} \\ v_{I_2} \end{bmatrix} \\ &- (N-1) v_{I_1} \begin{bmatrix} \beta_1 & 0 & 0 \\ -\beta_1 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \begin{bmatrix} v_S \\ v_{I_1} \\ v_{I_2} \end{bmatrix} \qquad (3) \\ &- (N-1) v_{I_2} \begin{bmatrix} 0 & 0 & 0 \\ 0 & \beta_2 & 0 \\ 0 & -\beta_2 & 0 \end{bmatrix} \begin{bmatrix} v_S \\ v_{I_1} \\ v_{I_2} \end{bmatrix}. \end{aligned}$$

The quality of the GEMF-approximation, is assessed by a comparison between the solution of (3) and the exact process for the complete network of $N = 100$ nodes. The values of the fractions of the exact process were determined by continuously applying the transition matrix of the Markov process until the rate of change in the fraction of nodes in each compartment was less than $\varepsilon = 10^{-6}$ for the last 10 iterations. We assume that the steady state has been reached if this condition is true. The parameters were set to $\delta_1 = \delta_2 = 1$ and $\mu_1 = \mu_2 = 0.001$, while values ranging from 0.01 to 0.1 for β_1 and β_2 were investigated. The results are shown in Figure 9.

There exists a discrepancy in the number of infected nodes by virus 1, which is most of the time underestimated by GEMF especially for higher superinfection rates. The number of infected nodes by virus 2, however, seems to be overestimated by GEMF, which is most apparent for higher superinfection rates. Figure 5 shows the convergence of both, the exact process and the GEMF-approximation for $\beta_1 = \beta_2 = 0.1$. These parameters corresponds to the upper right corner of the plots from Figure 9, where the discrepancy is observable.

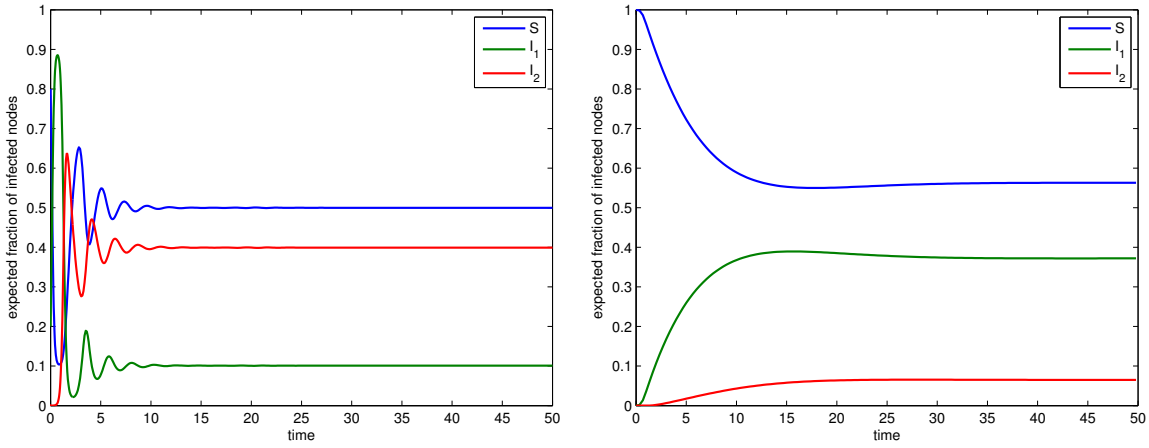
Apart from giving a rather inaccurate estimation on the fractions for the steady state, GEMF shows a damped oscillation not observable for the exact process for the first 10 time units. These oscillations might arise in cases where the average fraction of virus 1 nodes approaches 0, indicating a near-extinction event from which the viruses recover. A similar artifact is known from differential equations for predator prey models, and is sometimes coined the *atto-fox problem* [4], where a population of less than one individual, which would be practically extinct, is able to resurrect. As a side-effect, the proportion of infected nodes by virus 2 is overestimated, as it would inevitably extinct soon after virus 1 as well.

These observations lead us to the conclusion that the GEMF-approximation is reasonably accurate except for cases in which the infection rate β_1 is above the epidemic threshold together with a relatively high superinfection rate β_2. The next section will elaborate on the interaction of both viruses in general.

V. The course of superinfection

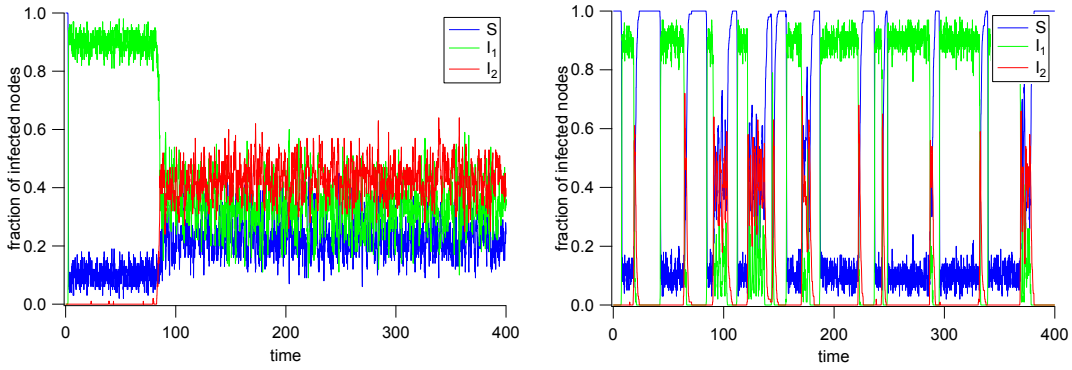
The course of a superinfection is divided in 3 phases: phase 1 begins with all nodes being susceptible, so only mutations can move a node from compartment S to I_1. The nodal mutations are necessary, but are set intentionally at small rates ($\mu_1 = \mu_2 = 0.001$) in comparison to the nodal curing $\delta_1 = \delta_2 = 1$. This assures that mutation has only a minor impact on the overall rate of newly infected nodes compared to their spreading over links. We want to outline the impact of the spreading rates β_1 and β_2 on the process, Therefore, we keep $\delta_1, \delta_2, \mu_1$ and μ_2 at those default values for all our analysis.

Once enough nodes are infected by mutations, the spreading rate β_1 determines, whether virus 1 becomes *endemic* (which means that it infects a larger part of the network) or not. If β_1 is too low, the process remains at phase 1 and apart from tenuous mutations, there is only a tiny number of infected nodes by virus 1 observable. Thus, an occurrence of virus 2



(a) GEMF (b) exact

Fig. 5. The exact process (right) and the GEMF approximation (left) over 50 time units with $\beta_1 = \beta_2 = 0.1$. At the beginning of the process, GEMF shows strong oscillations.



(a) acyclic superinfection ($\beta_2 = 0.03$) (b) cyclic superinfection ($\beta_2 = 0.08$)

Fig. 6. A single simulation of the superinfection process in the complete network with $N = 100, \beta_1 = 0.1, \delta_1 = \delta_2 = 1.0$ and $\mu_1 = \mu_2 = 0.001$. All 3 phases of the superinfection repeat rapidly in the cyclic case while they occur only once in the time interval of 400 for the acyclic case.

is unlikely[2].

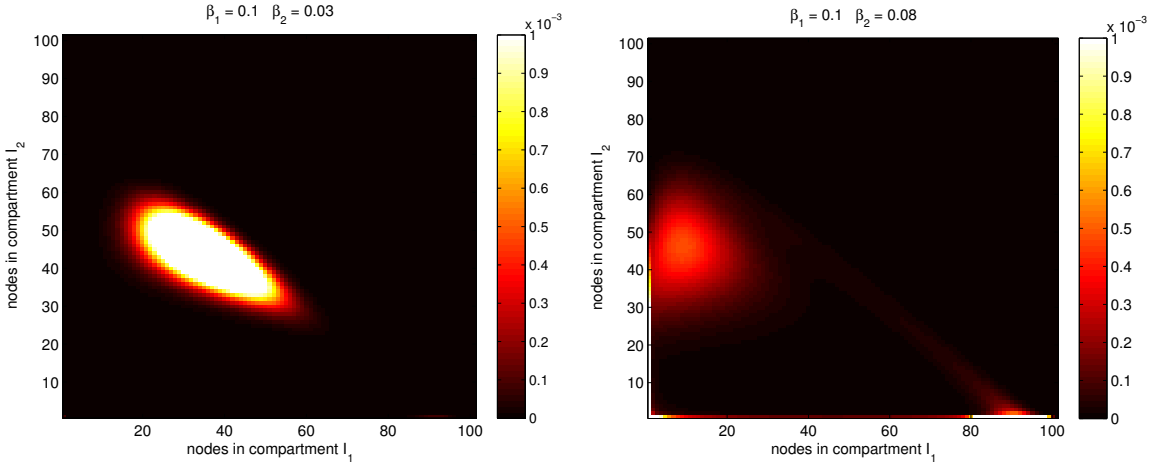
If β_1 is higher than the epidemic threshold of the corresponding network, virus 1 becomes endemic, eventually acquiring a larger fraction of the network. This marks a transition into phase 2, which is characterized by the first mutation events that will move single nodes from I_1 to I_2. Similar to virus 1 and β_1, the rate of β_2 determines, whether virus 2 will become endemic as well: If β_2 is too low to infect enough other nodes via spreading, virus 2 is only observed in tiny numbers and the process remains in Phase 2 with virus 1 persistent in the network. If β_2 is above a certain threshold, which we call σ_l, virus 2 becomes endemic and spreads inside the population that was previously infected, effectively reducing the fraction of infected nodes of virus 1.

The outbreak of virus 2 marks the transition to phase 3 in which a *coexistence* of both viruses in the network is established. In coexistence, both viruses infect (on average) a constant fraction of nodes in the network. The duration of the coexistence depends on another threshold, which we call σ_u. If β_2 is above σ_u, the spread of virus 2 is so strong that virus 1 can no longer infect enough new susceptible nodes to survive. If there are no more nodes in compartment I_1 left, the number of nodes in compartment I_2 goes down to zero as well, as the tenuous mutations are a much weaker force than the nodal curing. We call the extinction of both viruses in the network an *extinction event*. After an extinction event, the superinfection restarts at phase 1 and repeats until the next extinction event. Thus, we call this process *cyclic*.

If β_2 is in between σ_l and σ_u, a stable coexistence is maintained. Extinction events are unlikely and the process remains in phase 3. We call this process *acyclic*. Figure 7 illustrates the course of a cyclic and an acyclic superinfection in comparison, obtained by simulation.

The probability distribution of the Markov state space reveals the difference between acyclic and cyclic superinfection: In the former case, the probability mass is concentrated at network states in which a mixture of both viruses exist (Figure 7a). However, if β_2 approaches σ_u, the probability mass shifts to network states, where only one or even none (extinction)

[2]As the majority of the nodes remains in compartment S, virus 2 would also not be able to spread effectively.



(a) acyclic ($\beta_2 = 0.03$) (b) cyclic ($\beta_2 = 0.08$)

Fig. 7. Distribution of probabilities over the state space of the Markov process of superinfection in a complete network of 100 nodes. The rates are $\mu_1 = \mu_2 = 0.001, \delta_1 = \delta_2 = 1.0$ and $\beta_1 = 0.1$. In an acyclic coexistence (left) the probability mass is divided between states of mixed compartments. In a cyclic coexistence (right), the border states in which only one sort of virus exists have a considerably higher amount of probability mass than the mixed states.

viruses exist (Figure 7b).[3]

To further investigate the extinctions, we simulated superinfection in the complete network and counted the number of simulations that resulted in at least one extinction event. More precisely, for each combination of β_1 and β_2 over a certain range, we started 10 simulations for a fixed amount of time $T = 400$, which we assume to be long enough to observe possible extinctions in most cases. We count a simulation as extinct if two conditions are fulfilled: there is a time t_1 at which at least 10% of all nodes are infected with virus 2 and there is a time $t_2 > t_1$ at which all nodes are cured.

We observe that in this experiment the extinctions seem to be related to the second largest eigenvalue ζ_2 of the infinitesimal generator Q of the Markov process. Contrary to the standard SIS-model [24], the eigenvalue ζ_2 can be complex in the superinfection model. Figure 8 hints that the number of extinct simulations is higher for pairs of β_1 and β_2 that have a complex ζ_2. Although a real-valued ζ_2 does not necessarily imply an acyclic superinfection, the experiment suggests that a complex ζ_2 results most likely in a cyclic superinfection. Furthermore, discrepancies in Figure 9 seem to happen mostly when ζ_2 is complex, suggesting that GEMF has difficulties to capture extinctions.

Although the interactions of the 6 parameters of the superinfection model are complex, a qualitative description on their influence on the average number of nodes for each compartment can be devised as shown in Table I. The superinfection rate β_2 is a noteworthy exception as this parameter is particularly sensitive. Since virus 2 spreads inside a network that dynamically changes its size in response to the infection, we assume that σ_l and σ_u might depend on β_1 and (to a lesser extent) on μ_1.

Another strong influence is the topology of the underlying contact network. Figure 10 shows the exact process of the

[3]For a short video on the transition visit http://www.nas.ewi.tudelft.nl/index.php/maertens.

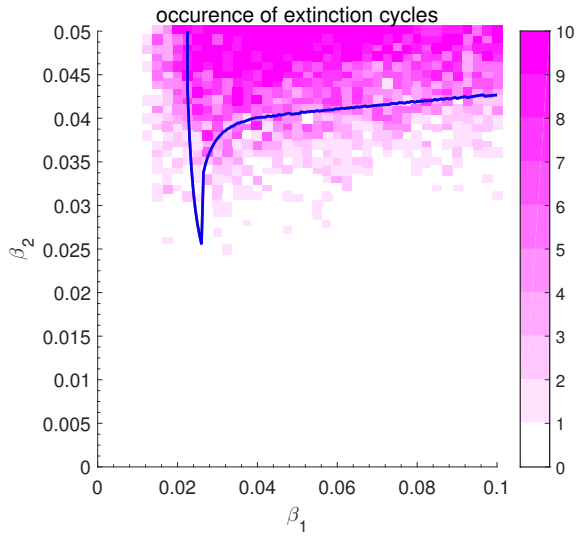


Fig. 8. Each pink square corresponds to a fixed value of β_1 and β_2. Its saturation is proportional to the number of simulations that resulted in at least one extinction event, with white indicating no extinctions at all. The blue curve interpolates for each β_1 the smallest β_2 for which the second largest eigenvalue of the infinitesimal generator of the Markov process is no longer real. The underlying network is the complete network of $N = 100$ nodes.

Markov chain from Figure 4 and its GEMF-approximation in the star network of $N = 100$ nodes. In comparison to the complete network shown in Figure 5, there seems to be no combination of β_1 and β_2 that allows for a larger support of compartment I_2. However, β_2 has an influence on the number of nodes infected by virus 1, which diminishes for higher values due to possible extinction events.

VI. Conclusions

In summary, we proposed a natural extension of the standard SIS-model to analyze nested epidemic processes in which a dominant virus spreads within the population of a another virus. We presented a succinct representation of the Markov

TABLE I. QUALITATIVE INFLUENCE OF THE PARAMETERS ON THE AVERAGE NUMBER OF THE NODES BELONGING TO THE THREE COMPARTMENTS.

	μ_1	μ_2	β_1	β_2	δ_1	δ_2
S	-	+	-	+	+	+
I_1	+	-	+	-	-	+
I_2	+	+	+	*	-	-

A $+$-sign indicates that a high rate of the corresponding parameters will increase the average number of nodes in that compartment while a $-$-sign indicates a decrease. The $*$-sign for the superinfection rate β_2 on compartment I_2 is a special case: in general, the value has to be high to infect more nodes, but a too high value results in an extinction event, negatively influencing the average number of superinfected nodes.

chain for the complete network and the star network that we used to evaluate the quality of the GEMF-approximation. In particular, we observed that for high superinfection rates β_2, the GEMF model shows damped oscillations and overestimates the fraction of infected nodes of the stronger virus.

A closer look at the exact process revealed rich and complex dynamics, ranging from endemic behaviour, stable coexistence to extinction cycles triggered by the occurrence of extinction events. It has been shown that the superinfection processes in 3 different phases that are dependent on the parameters. In particular, β_2 is the most sensitive parameter as it determines whether the process repeats itself in cycles or maintains a state of stable coexistence.

If β_2 is higher than a certain threshold σ_u, an extinction event will eventually eradicate both viruses. We expect σ_u to be strongly dependent on the infection rate β_1 of the weaker virus and the topology of the underlying network. As we focused our analysis on β_1 and β_2, it remains open to which extent the nodal parameters $\delta_1, \delta_2, \mu_1$ and μ_2 influence the process. However, their influence is reflected in the infinitesimal generator Q of the Markov process for which we observe that complex eigenvalues may occur. In particular, a complex second largest eigenvalue ζ_2 seems to be a sufficient condition to observe extinction cycles.

So far, we were unable to find conditions for coexistence or extinctions for the star network, which seems to be more robust against superinfection. We further conjecture that the inaccuracies in the GEMF model are correlated to the occurrence of extinction events.

Considering our model, a superinfection that cures from compartment I_2 back to I_1 instead of S might be worth investigating as well, with respect to real-world applications. Finally, an investigation of bigger hierarchies of 3 or more viruses in the SIkS-model promises even more complex interactions.

REFERENCES

[1] Roy M Anderson and Robert M May. *Infectious diseases of humans*, volume 1. Oxford University Press, Oxford, 1991.

[2] Michael Bailey, Evan Cooke, Farnam Jahanian, and David Watson. The blaster worm: Then and now. *Security & Privacy, IEEE*, 3(4):26–31, 2005.

[3] Gene Bransfield. The Welchia Worm. *Global Information Assurance Certification*, page 27, 2004.

[4] Fabien Campillo and Claude Lobry. Effect of population size in a prey-predator model. *arXiv preprint arXiv:1111.6460*, 2011.

[5] Faryad Darabi Sahneh and Caterina Scoglio. Competitive epidemic spreading over arbitrary multilayer networks. *Phys. Rev. E*, 89:062817, June 2014.

[6] Brian Karrer and Mark EJ Newman. Competing epidemics on complex networks. *Physical Review E*, 84(3):036106, 2011.

[7] Jure Leskovec, Lars Backstrom, and Jon Kleinberg. Meme-tracking and the dynamics of the news cycle. In *Proceedings of the 15th ACM SIGKDD international conference on Knowledge discovery and data mining*, pages 497–506. ACM, 2009.

[8] Gabriel E Leventhal, Alison L Hill, Martin A Nowak, and Sebastian Bonhoeffer. Evolution and emergence of infectious diseases in theoretical and real-world networks. *Nature communications*, 6, 2015.

[9] Robert M May and Martin A Nowak. Coinfection and the evolution of parasite virulence. *Proceedings of the Royal Society of London. Series B: Biological Sciences*, 261(1361):209–215, 1995.

[10] Atieh Mirshahvalad, Alcides Viamontes Esquivel, Ludvig Lizana, and Martin Rosvall. Dynamics of interacting information waves in networks. *Physical Review E*, 89(1):012809, 2014.

[11] Javier Mosquera and Frederick R Adler. Evolution of virulence: a unified framework for coinfection and superinfection. *Journal of Theoretical Biology*, 195(3):293–313, 1998.

[12] Seth A Myers and Jure Leskovec. Clash of the contagions: Cooperation and competition in information diffusion. In *2012 IEEE 12th International Conference on Data Mining (ICDM)*, pages 539–548. IEEE, 2012.

[13] Mark EJ Newman and Carrie R Ferrario. Interacting epidemics and coinfection on contact networks. *PloS one*, 8(8):e71321, 2013.

[14] Martin A Nowak and Robert M May. Superinfection and the evolution of parasite virulence. *Proceedings of the Royal Society of London. Series B: Biological Sciences*, 255(1342):81–89, 1994.

[15] Romualdo Pastor-Satorras, Claudio Castellano, Piet Van Mieghem, and Alessandro Vespignani. Epidemic processes in complex networks. *arXiv preprint arXiv:1408.2701*, 2014.

[16] Romualdo Pastor-Satorras and Alessandro Vespignani. Epidemic dynamics and endemic states in complex networks. *Phys. Rev. E*, 63:066117, May 2001.

[17] Federico Prado, Alyssa Sheih, Jevin D West, and Benjamin Kerr. Coevolutionary cycling of host sociality and pathogen virulence in contact networks. *Journal of theoretical biology*, 261(4):561–569, 2009.

[18] B Aditya Prakash, Alex Beutel, Roni Rosenfeld, and Christos Faloutsos. Winner takes all: competing viruses or ideas on fair-play networks. In *Proceedings of the 21st international conference on World Wide Web*, pages 1037–1046. ACM, 2012.

[19] Faryad Darabi Sahneh, Caterina Scoglio, and Piet Van Mieghem. Generalized epidemic mean-field model for spreading processes over multilayer complex networks. *IEEE/ACM Transactions on Networking (TON)*, 21(5):1609–1620, 2013.

[20] Péter L Simon, Michael Taylor, and Istvan Z Kiss. Exact epidemic models on graphs using graph-automorphism driven lumping. *Journal of mathematical biology*, 62(4):479–508, 2011.

[21] Ruud van de Bovenkamp, Fernando Kuipers, and Piet Van Mieghem. Domination-time dynamics in susceptible-infected-susceptible virus competition on networks. *Phys. Rev. E*, 89:042818, Apr 2014.

[22] Philippe Van de Perre, Michel Segondy, Vincent Foulongne, Abdoulaye Ouedraogo, Issouf Konate, Jean-Marie Huraux, Philippe Mayaud, and Nicolas Nagot. Herpes simplex virus and hiv-1: deciphering viral synergy. *The Lancet infectious diseases*, 8(8):490–497, 2008.

[23] Piet Van Mieghem. Epidemic phase transition of the SIS type in networks. *EPL (Europhysics Letters)*, 97(4):48004, 2012.

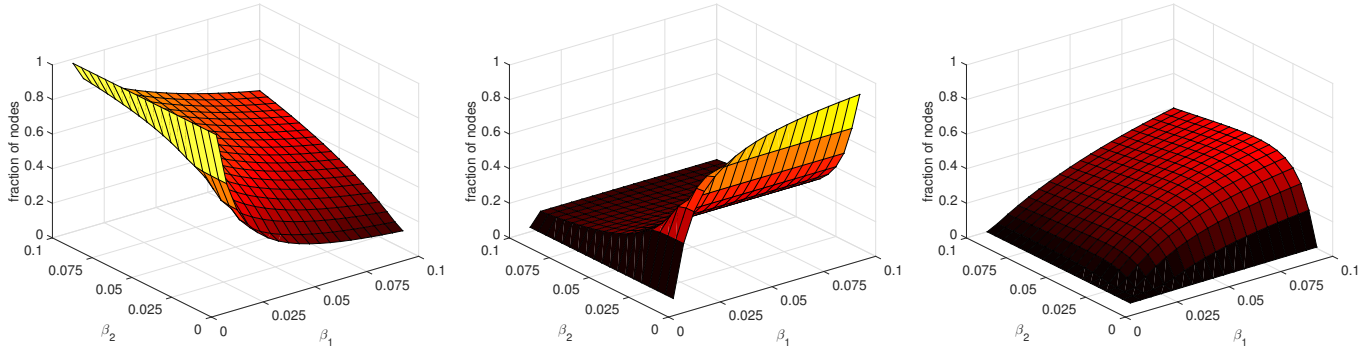
[24] Piet Van Mieghem. Decay towards the overall-healthy state in sis epidemics on networks. *arXiv preprint arXiv:1310.3980*, 2013.

[25] Piet Van Mieghem. *Performance Analysis of Complex Networks and Systems*. Cambridge University Press, 2014.

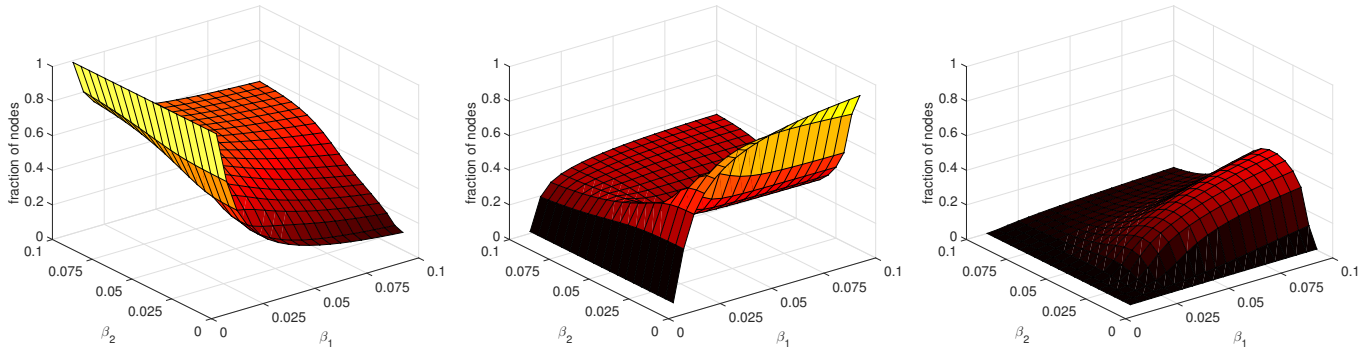
[26] Piet Van Mieghem and Eric Cator. Epidemics in networks with nodal self-infection and the epidemic threshold. *Phys. Rev. E*, 86:016116, July 2012.

[27] Piet Van Mieghem, Jasmina Omic, and Robert Kooij. Virus spread in networks. *IEEE/ACM Transactions on Networking*, 17(1):1–14, 2009.

[28] Qingchu Wu, Michael Small, and Huaxiang Liu. Superinfection behaviors on scale-free networks with competing strains. *Journal of nonlinear science*, 23(1):113–127, 2013.

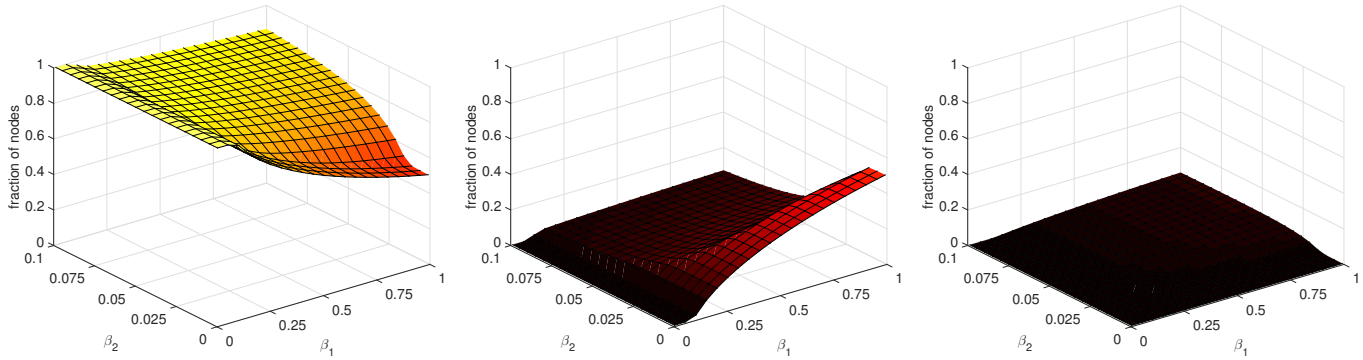


(a) GEMF S (b) GEMF I_1 (c) GEMF I_2

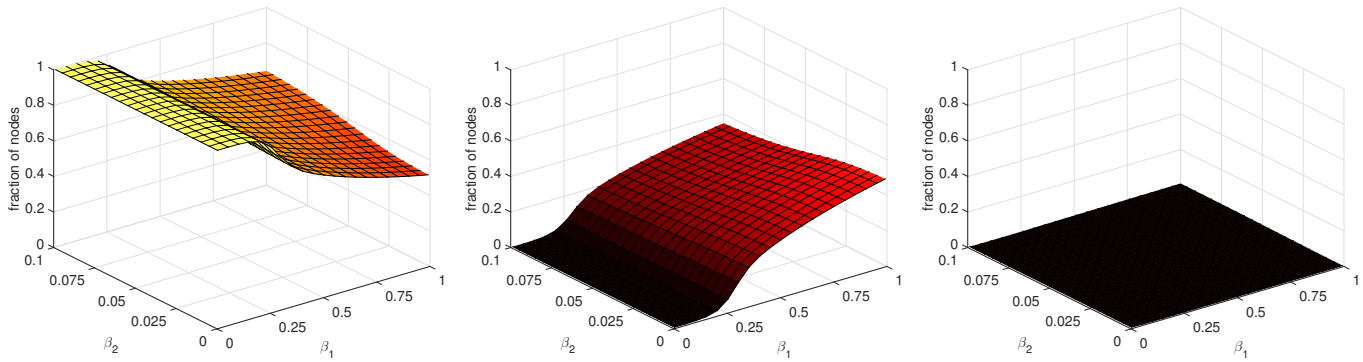


(d) exact S (e) exact I_1 (f) exact I_2

Fig. 9. Expected fraction of nodes in corresponding compartments in the steady state obtained by GEMF (top) and the exact process (bottom) in a complete network of $N = 100$ nodes.



(a) GEMF S (b) GEMF I_1 (c) GEMF I_2



(d) exact S (e) exact I_1 (f) exact I_2

Fig. 10. Expected fraction of nodes in corresponding compartments in the steady state obtained by GEMF (top) and the exact process (bottom) in a star network of $N = 100$ nodes.