J. Parallel Distrib. Comput. 71 (2011) 87–104

Contents lists available at ScienceDirect

J. Parallel Distrib. Comput.

journal homepage: www.elsevier.com/locate/jpdc

An analytical model for multi-epidemic information dissemination

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ARTICLE INFO

Article history:
Received 19 February 2010
Received in revised form
18 May 2010
Accepted 22 August 2010
Available online 16 September 2010

Keywords:
Epidemics
Information dissemination
Stochastic process
Transmutation

ABSTRACT

Contemporary distributed systems usually involve the spreading of information by means of ad-hoc dialogs between nodes (peers). This paradigm resembles the spreading of a virus in the biological perspective (epidemics). Such abstraction allows us to design and implement information dissemination schemes with increased efficiency. In addition, elementary information generated at a certain node can be further processed to obtain more specific, higher-level and more valuable information. Such information carries specific semantic value that can be further interpreted and exploited throughout the network. This is also reflected in the epidemical framework through the idea of virus transmutation which is a key component in our model. We establish an analytical framework for the study of a multi-epidemical information dissemination scheme in which diverse 'transmuted epidemics' are spread. We validate our analytical model through simulations. Key outcomes of this study include the assessment of the efficiency of the proposed scheme and the prediction of the characteristics of the spreading process (multi-epidemical prevalence and decay).

1. Introduction

In this paper we focus on a distributed computing setting with the following characteristics:

- An unstructured network of a large number of nodes.
- Nodes send and receive information.
- Nodes can relay information to other nodes.
- Nodes have specific neighbours either due to communication constraints or some logical organization.
- Exchanged information carries certain semantic value and presents dependencies to other pieces of information.
- Semantic dependencies can be further exploited by nodes to more accurately tune their behaviour.

We propose an analytical model for disseminating pieces of information in various levels of detail. The considered model is probabilistic, so that each node decides with a given probability whether to transmit/relay information or not. We capitalize on the similarity between information dissemination and the epidemic model, mostly in the abstractions of virus spreading, virus severity, virus interdependencies and possible transmutations.

The great advantage of epidemic-inspired communication is that dissemination proceeds on a local basis, without any central coordination [12]. Such communication is highly reliable, efficient, and resilient to sudden failures of communication links and nodes. However, the study on dissemination of dependent pieces of information is a major challenge. For instance, consider the dissemination of fragments of files in a peer-to-peer overlay, news updates and semantic multimedia meta-data updates in a social network, more accurate and up-to-date measurements in a wireless sensor network. Specifically, the dependent pieces of information that have been disseminated can be further processed by receiving nodes to obtain more up-to-date, specific, higher-level and more valuable information. In this paper we adopt the epidemics-based dissemination model, which reliably and efficiently handles the information dissemination issue.

Information dissemination is handled by a simple, message passing protocol to fulfil certain information diffusion requirements, i.e., spatio-temporal constraints. A piece of information should reach potential recipients within a certain time horizon (e.g., in less than 2 min). Moreover, such piece may be considered valid only in the neighbourhood of the node (in specific geographical area or within a certain number of hops). This means that, not all information needs to be propagated everywhere. Through this paper we show that the stochastic nature of epidemic spreading achieves very high coverage (even) for low forwarding probability: probability of a message passing to a neighbour. The similarity of our model to epidemics extends beyond the spreading scheme.

The basic scheme: A disseminated piece of information matches an epidemic in the sense that, a node carrying a piece of information becomes infected; otherwise it is susceptible. An infected node

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doi:10.1016/j.jpdc.2010.08.010

can disseminate information to its neighbours in an opportunistic way. When a node carries information, which has become invalid, e.g., beyond the scope, then it becomes susceptible and can be probably re-infected at a later stage.

The extended scheme: The severity of epidemics is included in our abstraction. We introduce the metaphor of a stronger epidemic than the one, which has previously infected a given node. Stronger epidemics abstract more detailed information, i.e., information which is valued more by the receiving node. To be able to assess the value of the incoming piece of information, each node provides a mechanism which unambiguously specifies interdependencies among pieces of information and evaluates them. In addition, some nodes provide a mechanism to generate/infer information. We can figure out the following scenarios:

- the incoming piece of information, denoted as p, is of *better quality* than the one previously received (e.g., more up-to-date message), or p is *more detailed* than the one previously received by the node (e.g., incoming p refers to more accurate position), and,
- the incoming p can act as a *seed* thus enabling the receiving end to generate/infer additional information. For instance, the incoming p covers the missing elements needed for generating extra information; the combination of location, time and information about the scheduled lectures of a conference could be exploited by a personal conference assistant application. We adopt the term 'information completion' for this case, where some vital pieces of information are missing (until reception of incoming p) in order to generate new information of higher semantic value. Since, in any of these cases, the derived information (i.e., after the reception of new information from the neighbourhood) is deemed more valuable for the receiving node, we abstract them as 'epidemical aggravation'.

The last part of the abstraction is related to the dissemination of derived/inferred information. Imagine that the derived information is not kept locally in the node, but further treated as a virus and propagated to the neighbourhood as new information. A new virus starts spreading and will possibly infect nodes in the proximity. Hence, in a group of nodes multiple pieces of information circulate, thus, leading to the need of studying a multi-epidemic information dissemination scheme. The contribution of this paper is the study of the multi-epidemical information dissemination. Our objective is to develop a mathematical model that deals with:

- the multi-epidemical spreading in the light of information completion and aggravation, and,
- the multi-epidemical thresholds, which are quantitative indicators on the spreading dissemination, e.g., the infection rates that turns the epidemic into a pandemic or causes the epidemic to fade out.

To this purpose,

- we first abstract the problem of multi-epidemics assuming that nodes are capable of deciding whether to replace existing pieces of information with more valuable incoming information or not.
- we use a graph-based representation in order to formulate multi-epidemical propagation through a spatio-temporal random process based on interactions among nodes.
- we make use of a discrete-time model and network topology information to describe the spatial and temporal statistical dependencies among diverse epidemics in arbitrary network topologies. The temporal dependency is modelled as a Markov process.
- we consider conditional probability independence among neighbouring infected nodes; the assumption of such independence introduces an exact joint probability distribution depending on one-node marginal probabilities. The proposed model only allows one event, i.e., Susceptible $\rightarrow$ Infected, or Infected $\rightarrow$ Susceptible, in a single discrete time step focusing on the transient behaviour of multi-epidemical propagation.
- we generalize the Markov model in [25] (w.r.t. mathematical model), which deals with mono-epidemical spreading.
- we apply our model to describe the final size of infection, which corresponds to the equilibrium solution and characterizes the potential distribution of the epidemics over a network.
- the proposed model emphasizes the steady-state solution and the epidemical threshold for each epidemic. The model captures the impact of the underlying topology but is not limited to it. The viral propagation is largely determined by the intrinsic characteristics of the network. We show that, the multi-epidemical threshold is closely related to the eigenvalues of the network adjacency matrix and decays exponentially over time.
- we study the significance of the topology in determining epidemical thresholds and the speed of propagation for each transmuted epidemic.

The article is organized as follows: Section 2 discusses related work and Section 3 introduces the concept of multi-epidemical information dissemination. In Section 4 we analyse the corresponding mathematical model. Section 5 reports analytical and simulation results. Finally, in Section 6 we discuss possible exploitation and adoption of the proposed model with other relevant models, and conclusions and directions for future work are presented in Section 7.

2. Related work

Several approaches have been proposed to model and simulate mono-epidemical spreading in networks. Therefore, considerable research related to reliable epidemic-based information dissemination in ad-hoc networks has been studied in [19]. The work in [11] presented a model which analyses the epidemical spreading in random graphs. The authors in [5] studied epidemical spreading in complex networks and the authors in [22] proposed a spreading model for arbitrary network topologies. In [25] the authors analyse a Markov process-based framework that characterizes the spreading of epidemics through the SIS model and the impact of the underlying topology on propagation. In that work, a mono-epidemical propagation model is introduced based on temporal and spatio-temporal dependency of the infected nodes (an Independent and Markov model, respectively). However, the concept of transmutation of an epidemic is not considered since there is no information processing (e.g., information completion). Additionally, the architecture discussed in [16] proposed an approach for dissemination among groups of nodes. Such architecture takes into account the reliability of information in the dissemination process. Nevertheless, the information spreading adopts a simple flood-based model. The performance handicap of a flood-based approach with respect to epidemical spreading is studied in [1] and in this paper, too. The work in [13] deals with the application of the *Susceptible-Infected* (SI) epidemiological model across ad-hoc networks. Nonetheless, the SI model cannot be considered appropriate for multi-epidemical information dissemination, since infected nodes cannot recover or get reinfected (see Section 3). Moreover, the autonomous gossiping algorithm in [8] refers to the selectivity attribute of the epidemical spreading. However, such an algorithm does not consider any information process, thus, nodes cannot generate new information and, consequently, cannot augment knowledge. The model in [10] refers only to spatially constrained information dissemination. The authors in [22] investigate the threshold of a mono-epidemical propagation exploiting the eigenvalue of the adjacency matrix of the network. Our model generalizes the model in [22] investigating the behaviour of

each epidemic, thus, in case of a mono-epidemical propagation our results coincide with those in [22]. To the best of our knowledge, there is no other approach for multi-epidemiological dissemination. The proposed model goes beyond a simple epidemiological model and introduces the abstraction of stronger and transmuted epidemics. The prior work however has not incorporated the concept of aggravation and transmutation.

3. Multi-epidemical information dissemination model

A node can store a piece of information in order to disseminate it to nearby nodes under certain time-space constraints. The proposed model adopts a simplistic routing scheme where dissemination proceeds on a local basis and does not require central coordination or complex routing schemes. In addition, all nodes are stationary, all pieces of information are transmitted in the form of local (1-hop) broadcast and a node opportunistically forwards information to neighbours.

According to the Kermack and McKendrick model, an individual can be in three states: *I*nfected (i.e., an individual is infected with epidemic), *S*usceptible (i.e., an individual is prone to be infected) and *R*emoved (i.e., an individual is immune, as it has recovered from the disease). This kind of model is usually referred to as *S*usceptible-*I*nfected-*R*ecovered (SIR). A simplified version of that model is the *S*usceptible-*I*nfected-*S*usceptible (SIS) model in which an individual can exist in only susceptible and infected states. This means that an individual never gets immune after its contact with the epidemic. Moreover, SIS assumes that an infected individual cannot be re-infected by a stronger epidemic. In addition, in the *S*usceptible-*I*nfected (SI) model, an individual never turns susceptible if infected once. The SI, SIR and SIS models cannot abstract the proposed multi-epidemic spreading since in our model (i) an individual might return to the susceptible state from the infected state, (ii) might be infected by a stronger epidemic, and (iii) never turns immune from an epidemic. Evidently, we extend SIS at that point, i.e., an infected node with epidemic p can be re-infected with a stronger epidemic q resulting in the aggravation of the node's condition. Then, the node may infect neighbours, for instance, with more detailed information. On the other hand, the cure of a node refers to the improvement of its condition. The abstraction of a cure indicates that, for instance, the information is no longer valid (i.e., beyond the spatial scope or out-of-date). We adopt the acronym SI^KS for the extended SIS model indicating that the proposed model deals with K re-infections of *different* epidemics; for $K = 1$ we obtain the SIS model.

SI^kS deals with numerous dependent pieces of information – epidemics – of different quality levels spread in the network simultaneously. The 'strongest' epidemic (with highest quality level) has the potential to infect a large portion of susceptible nodes, contrary to weaker epidemics, which infect a smaller portion of the group. The multi-epidemic dissemination is totally different from the fact that several independent epidemics co-exist in a network, as shown in the selective dissemination schemes.

The specific relations among epidemics are exploited by the nodes to substitute, complete, or generate new information. Nodes incapable of generating new information can acquire supplementary/additional knowledge once information inference takes place: if a node in a group has the ability to generate/infer information then it can disseminate it into the group, thus, augmenting the current common knowledge. This *reciprocity* guarantees a *modus operandi* for a network of autonomous nodes working under the S^KIS model for collaboratively sharing and inferring information.

Consider the set of pieces of information C. Let us define a (semantic) relation $>$ over C such that $p > q$ denotes that p is more preferable than q (e.g., w.r.t. quality, freshness, refinement). Preferable piece of information could be, for instance, 'more detailed', 'up-to-date', 'specific', 'of better precision' referring to the perspective of interest/interpretation of the node. Each node compares incoming $p \in C$ with existing $q \in C$. If $p > q$ then the node accepts the incoming information and aggravates its condition; otherwise, the node does not replace q with p and does not aggravate its condition. This also denotes the selectivity attribute of the S^KIS model, in which, each node obtains the precise information it requires. Nevertheless, each node can, further, locally refine information independently of other nodes. It is also possible for a node to exploit the accepted piece of information p in order to generate more valuable information ϕ (transmutation from p to ϕ) and disseminate it across the network.

Example. We provide an example that maps the multi-epidemical concepts/abstractions to real contextual information dissemination among location-aware applications. Throughout the paper, we regularly refer to this example to further explain the mapping/analogy of the concepts to the proposed model. At first, in this example, the context type is position information, so the p, q epidemics refer to location information and the semantic relation $p > q$ indicates that location p contains more detailed spatial information in terms of accuracy/precision than that of q. Second, we make use of an ad-hoc, short range, wireless communication scheme between nodes. We also consider attendees in a scientific conference who are equipped with smart-phones. The conference is held in the first two floors of a large convention center. Not all the smart-phones of attendees are equipped with the same set of localization sensors and methods (e.g., terrestrial localization like EOTD, Infrared-beacon, WLAN positioning) or have Wi-Fi network connectivity. Consider that a presentation is given in Room 5/Floor 2 of the conference building with a certain audience. An attendee estimates her position through the EOTD (Enhanced Observed Time Difference) localization method [14]. Hence, her location information is p_1= 'Conference building:EOTD'. That node disseminates p_1 to its local neighbourhood (other attendees) denoting that, her current location is determined with EOTD accuracy along with a time validity indicator (e.g., up to 2 min). At the time being, some attendees can receive the position information p_1 together with temporal and spatial constraints (e.g., inside the convention center valid for 2 min). Another neighbouring attendee estimates with a certain accuracy, her position by the Wi-Fi received signal strength (RSS), thus, the position information p_2= 'Conference building, Floor 2:WLAN'. p_2 is also disseminated to local neighbouring nodes. The attendees on Floor 2 that have been recently infected by p_1 can now be re-infected with p_2 once the latter information describes in more details localization information. In addition, a node infected by p_2 can also infer p_1. Earlier, another neighbouring attendee exploits the incoming location information p_2 and estimates a more accurate position, e.g., the location p_3= 'Conference building, Floor 2, Room 5:Infrared', since an Infrared receiver is mounted on that node [18]. The latter localization estimation (p_3) is far more accurate than p_2 and p_1. The node disseminates p_3 as an improvement on p_2 to its immediate local neighbours for e.g., a certain number of hops (inside the Room 5). The neighbouring attendees of that node can now be supported with more detailed location information, thus, improving the service quality. Finally, in this example it holds true that $p_3 > p_2 > p_1$.

4. Analysis

4.1. Notation

A population of stationary nodes can be defined spatially, i.e., all the nodes found in a certain geographical area. The nodes are capable of stochastically spreading pieces of information among

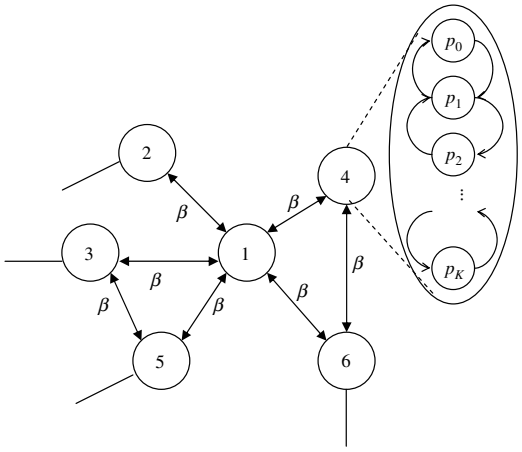

Fig. 1. The multi-epidemical network model.

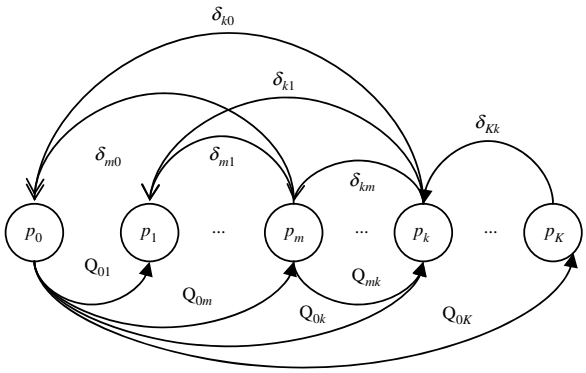

Fig. 2. State transition diagram of a node with transition, partial and full cure rates (not all possible transitions are illustrated for readability reasons).

them through a certain number of hops in order to update and generate new information. The neighbours of a node i are nodes in the communication range of node i. We use a directed graph $\mathcal{G}(V, E)$ to represent a multi-epidemical network, where V is the set of nodes and E is the set of edges. The state of node i at time instant t is denoted by $x_i(t)$. This state assumes $K+1$ values (in our example $K = 3$) which are represented by the $K+1$-dimensional vectors $p_k = [0, \ldots, 0, 1, 0, \ldots, 0]^\top$ where all values are zero except the kth component ($k = 0, 1, \ldots, K$) which takes the value 1. A state of value p_k denotes that the node is in the infected state with epidemic p_k of level k. Therefore, the strongest epidemic that is disseminated across a network is p_K of level K. A node with the most susceptible status is in a state p_0 whereas a node with the most infectious status is in a state p_K. The neighbourhood of node i, denoted by V_i, is a subset of V where every node j in this subset has an edge connecting it to node i, i.e., $V_i = \{j|(j, i) \in E\}$. Each edge (j, i) in E is associated with $\beta_{ji} = \beta$, which is the infection rate with which an infected node j can infect a neighbouring node i. Fig. 1 depicts a representative multi-epidemical network where the neighbourhood of node 1 is $V_1 = \{2, 3, 4, 5, 6\}$. For the values of the state of a node we assume an ordering $p_l > p_k$ if $l > k$. A node infected by p_k is likely to be re-infected by p_l as this stronger epidemic spreads across the network. In addition, $n_k(t)$ is the expected number of infected nodes for all epidemics p_k, $k = 1, \ldots, K$. A node i of infection level p_l, can only infect a node j in its neighbourhood, which is in state p_k, if and only if $p_l > p_k$, that is either node j is susceptible (p_0) or it is infected at a lower level than l.

A node i of infection level p_k can be cured in two ways. In the full cure case, node i transits in one step to the susceptible state p_0 with rate δ_{k0} (see Fig. 2). In the partial cure case, node i, being at state p_l, transits in a lower state p_k, $1 \le k < l$, with rate δ_{lk}. Partial cure means that the node is still infected since it transits to a state corresponding to a weaker epidemic than the previous one. Nevertheless, a partially cured node can be re-infected with a stronger epidemic. Fig. 2 depicts the state transition diagram of a node. It should be noted that the transition rates Q_{kl} depend on the number of neighbouring infected nodes, their infection level and the infection rate β. A discussion on the values of Q_{kl} follows in the next subsection. Surely, it is possible for a node to jump directly from the most infectious state p_K to the susceptible state p_0.

4.2. The spatio-temporal $\mathrm{SI}^K S$ model

As node i can be infected only by its neighbours, the state $x_i(t)$ at time t is statistically dependent on the status of its neighbours and on the the previous state $x_i(t-1)$ at time $t-1$. Since the status of a neighbour also depends on its own neighbours, the status of all nodes is statistically dependent in space and time. Let the vector $\mathbf{x}(t)$ denote the status of all nodes at time t, that is

$$\mathbf{x}(t) = [x_1(t), x_2(t), \ldots, x_M(t)]^\top \tag{1}$$

where M is the number of nodes in the network. It is clear that $\mathbf{x}(t)$ is a spatiotemporal process.

The infection and recovery rates β and δ_{lk}, respectively, are very important. The infection rate β denotes the birth rate of an epidemic from a node $j \in V_i$. Given the status of the neighbours of node i at time t and the fact that node i may be infected at level k, at the next time instant $t+1$ node i will be infected at a higher level l with probability

$$Q_{kl} = \left(1 - \sum_{m<k} \delta_{km}\right) \cdot \left(1 - \prod_{j \in V_i} (1-\beta)^{x_j^T(t) \cdot p_l}\right) \times \prod_{j \in V_i} (1-\beta)^{x_j^T(t) \cdot \sum_{m>l} p_m}. \tag{2}$$

The expression $(1 - \sum_{m<k} \delta_{km}) \cdot \left(1 - \prod_{j \in V_i} (1-\beta)^{x_j^T(t) \cdot p_l}\right)$ in (2) is the probability that one or more nodes will infect node i at infection level l and node i will not recover. The second product in (2), i.e., $\prod_{j \in V_i} (1-\beta)^{x_j^T(t) \cdot \sum_{m>l} p_m}$, expresses the probability that all the nodes $j \in V_i$ with an infection level greater than l will not infect node i. According to our example, let Q_{12} denote the transition probability in which a node i receives information p_2 while it has recently received information p_1. This will happen once (a) some neighbouring node $j \in V_i$ relays p_2 to node i with infection rate β, (b) node i will not discard p_1 and, (c) some neighbouring node, which has the p_3 piece of information, will not relay it to node i with probability $(1-\beta)$.

If we consider a Markov chain of unit time transition periods, the transition probabilities (see Fig. 2) that express the temporal dependence of states of a node i are:

Full cure case:

$$P\{x_i(t+1) = p_0 | x_i(t) = p_k\} = \delta_{k0} \tag{3}$$

that is the probability for a node i to automatically get to the susceptible state from the k-level epidemic.

Partial cure - condition improvement case:

$$P\{x_i(t+1) = p_k | x_i(t) = p_l\} = \delta_{lk}, \quad 1 \le k < l \tag{4}$$

that is the probability for a node i to automatically get to the k-level state from the higher l-level epidemic.

Infection - condition aggravation at a higher level $k < l$:

$$P\{x_i(t+1) = p_l | X_{V_i}(t) = \mathbf{x}_{V_i}(t), x_i(t) = p_k\} = Q_{kl} \tag{5}$$

where, the random vector $X_{V_i}(t)$ denotes the status of all neighbours of node i, i.e., $X_{V_i}(t) = [x_j(t), j \in V_i]$ and $\mathbf{x}_{V_i}(t)$ is a realization of $X_{V_i}(t)$. The transition probability in Eq. (5) is stimulated by the neighbouring nodes of the node i. Given the conditional probabilities in Eqs. (3)–(5) we can calculate the probability that a node i is in state p_k at time instant $t+1$, that is

$$\begin{aligned}
&P\{x_i(t+1) = p_k\} \\
&= \sum_{m=0}^{K} P\{x_i(t+1) = p_k, x_i(t) = p_m\} \\
&= \sum_{m=0}^{K} P\{x_i(t+1) = p_k | x_i(t) = p_m\} P\{x_i(t) = p_m\} \\
&= \sum_{m=0}^{K} \sum_{\mathbf{x}_{V_i}(t)} P\{x_i(t+1) = p_k, X_{V_i}(t) = \mathbf{x}_{V_i}(t) | x_i(t) = p_m\} \\
&\quad \times P\{x_i(t) = p_m\}
\end{aligned}$$

and finally

$$\begin{aligned}
&P\{x_i(t+1) = p_k\} \\
&= \sum_{m=0}^{K} \sum_{\mathbf{x}_{V_i}(t)} P\{x_i(t+1) = p_k | X_{V_i}(t) = \mathbf{x}_{V_i}(t), x_i(t) = p_m\} \\
&\quad \times P\{X_{V_i}(t) = \mathbf{x}_{V_i}(t) | x_i(t) = p_m\} P\{x_i(t) = p_m\}. \qquad (6)
\end{aligned}$$

Eq. (6) provides a recursive relationship between $x_i(t+1)$ and $x_i(t), x_j(t)$ for $j \in V_i$, and gives a formal stochastic model. Eq. (6) can be used to study the behaviour of epidemical spreading. Specifically, the conditional probability

$$P_m(t) = P\{X_{V_i}(t) = \mathbf{x}_{V_i}(t) | x_i(t) = p_m\} \qquad (7)$$

explicitly characterizes the statistical dependencies due to the network topology (local knowledge on the neighbours of a node i) and the interactions among neighbouring nodes. The computational cost for $P_m(t)$ is $O((K+1)^{|V_i|})$, since there are $(K+1)$ states (including the susceptible state) for each neighbour $j \in V_i$ of a node i. Therefore, the simplest approximation is to assume conditional probability independence. With the conditional independence assumption, the conditional probability in Eq. (7) can be factorized into a form that only depends on one-node marginal probabilities. That is, given that a node i is in state m (Eq. (7)) at some time t, the status of its neighbouring nodes $j \in V_i$ does not depend on the current status p_m of node i at time t. Hence,

$$\begin{aligned}
P_m(t) &= P\{X_{V_i}(t) = \mathbf{x}_{V_i}(t) | x_i(t) = p_m\} \\
&= P\{X_{V_i}(t) = \mathbf{x}_{V_i}(t)\} = \prod_{j \in V_i} P\{x_j(t)\}. \qquad (8)
\end{aligned}$$

The probability in Eq. (8) certainly maintains temporal dependency and detailed (neighbour) topology information. Moreover, since a node i has $|V_i|$ neighbours, the total number of states needed to describe $P_m(t)$ is reduced from $O((K+1)^{|V_i|})$ to $O(|V_i|)$. This means that, the computation cost for calculating $P_m(t)$ with the conditional independence assumption is independent on the number of epidemics K and linear to the number of neighbours $|V_i|$. It should be noted that the conditional independence assumption has been also adopted in [22]. The model given here, however, differs from the model in [22] in the following aspects: our model is derived from the accurate spatial-temporal process and only allows one event (i.e., transition among susceptible, infection, or aggravation) in a single time step, while the model in [22] allows infection and recovery to occur simultaneously.

We now focus on the calculation of the probability

$$Q_{mk} = P\{x_i(t+1) = p_k | X_{V_i}(t) = \mathbf{x}_{V_i}(t), x_i(t) = p_m\}$$

in Eq. (6). Q_{mk} expresses the probability of a node i to be infected by an epidemic of level k at time $t+1$ given that the node is infected with epidemic of level m and the neighbouring nodes have specific information value at time t. Several cases can be considered depending on the values of m and k.

Node i remains susceptible.

In this case $m = k = 0$, which means that node i is not infected by any epidemic from neighbouring infectious nodes. The probability of this event is

$$Q_{00} = \prod_{j \in V_i} (1-\beta)^{\sum_{k>0} x_j^T(t) \cdot p_k}. \qquad (9)$$

According to our example, Q_{00} denotes the probability that node i does not receive any piece of position information (neither p_1 nor p_2 nor p_3) from any neighbouring node $j \in V_i$. That is, every neighbouring node j does not relay its possible piece of information of level $0 < k \leq 3$ with rate $(1-\beta)$.

A node i is infected with a stronger epidemic p_k, $m < k$.

The probability of this event is given in Eq. (2) which is repeated here for clarity

$$\begin{aligned}
Q_{mk} &= \left(1 - \sum_{l<m} \delta_{ml}\right) \cdot \left(1 - \prod_{j \in V_i} (1-\beta)^{x_j^T(t) \cdot p_k}\right) \\
&\quad \times \prod_{j \in V_i} (1-\beta)^{x_j^T(t) \cdot \sum_{l>k} p_l}. \qquad (10)
\end{aligned}$$

Mapping the probability Q_{km} of our example to Eq. (10), node i receives a piece of position information of level p_k once: at least some neighbouring node $j \in V_i$ relays p_m to node i with infection rate β, and information p_k is valid for node i (i.e., node i does not discard p_k with cure rate $\sum_{l<m} \delta_{ml}$) and, finally, none of the neighbouring nodes, which have information of level $l > k$, relays it to node i.

Node i remains infected at the same level p_k.

The probability of this event is

$$Q_{kk} = \left(1 - \sum_{l<k} \delta_{kl}\right) \prod_{j \in V_i} (1-\beta)^{x_j^T(t) \cdot \sum_{l>k} p_l}. \qquad (11)$$

That is, the node's condition is not aggravated due to infection by a stronger epidemic and is not improved (partial or full cure). Based on our example, node i maintains its position information of level p_k, $1 \leq k \leq 3$, once together holds true that p_k is still valid for node i (i.e., node i does not discard p_k with cure rate $\sum_{l<m} \delta_{ml}$) and none of the neighbouring nodes $j \in V_i$, which have information of level $l > k$, relays it to node i.

Node i is partially or fully cured.

The probability of this event is

$$Q_{mk} = \delta_{mk}, \quad m > k. \qquad (12)$$

If $k = 0$ the node is fully cured, otherwise it is only partially cured. In our example, we can consider that a node i either discards a piece of position information p_m at all once it is invalid (e.g., not valid for a certain time horizon) corresponding to the cure rate δ_{m0} or it holds true a piece of position information of lower accuracy level k corresponding to the cure rate δ_{mk}, $k > 0$. For instance, if p_3 does not represent the position of node/attendee i at some time, then possibly its position can be either represented by p_1 (cure rate δ_{31}) or p_2 (cure rate δ_{32}), or even unknown — p_0 (cure rate δ_{30}).

Node i remains in the highest level of infection.

The probability of this event is

$$Q_{KK} = 1 - \sum_{l<K} \delta_{Kl}. \qquad (13)$$

4.3. An eigenvalue approach for multi-epidemical spreading

In this section we analyse the epidemical spreading and determine specific performance thresholds through an eigenvalue approach. This is a generalization of the work in [22], while the conclusions of such generalization are reported at the end of this section. Specifically, the epidemical threshold for an epidemic p_k or its transmutation denotes whether an outbreak of p_k occurs (pandemic), i.e., full coverage of the entire network, or not. We consider the Markov chain consisting of $M \times (K+1)$ states, where M is the number of network nodes and K is the number of different epidemics. The states of this Markov chain are denoted by $S_{i,k}$, which means that node i is infected by epidemic p_k. To simplify our notation we use $P_{i,t}^k$ to denote the probability that node i is infected by p_k at time t, that is

$$P_{i,t}^k = P\{x_i(t) = p_k\}.$$

Therefore, $P_{i,t}^k$ is the probability that the Markov chain is in state $S_{i,k}$ at time instant t. Writing the balance equations for states $S_{i,k}$, $1 \le k \le K$, we obtain

$$P_{i,t+1}^k = \sum_{m<k} \sum_{\mathbf{x}_{V_i}(t)} Q_{mk} \prod_{j \in V_i} P\{x_j(t)\} \cdot P_{i,t}^m + \sum_{\mathbf{x}_{V_i}(t)} Q_{kk} \prod_{j \in V_i} P\{x_j(t)\} \cdot P_{i,t}^k + \sum_{m>k} \delta_{mk} P_{i,t}^m. \tag{14}$$

The double sum in (14) expresses the transitions into state $S_{i,k}$ from the neighbouring infected nodes whose infection level is lower than k. More specifically, this quantity expresses the probability that node i transits from an epidemic of level $m < k$ to the epidemic of level k due to infection of some neighbouring node, which at time t is infected with p_m. The second sum in (14) expresses the transition from state $S_{i,k}$ to itself. In this case, no cure or infection at a higher level should occur. Specifically, this quantity expresses the probability that node i will be still infected with epidemic of level k since neither infections are possible by neighbouring nodes nor node i automatically transits to epidemic of lower level. The final sum in (14) expresses partial cure transitions from a state of node i, $S_{i,m}$, of a higher infection level ($m > k$). More specifically, this quantity expresses the probability that node i transits from a higher level of epidemic $m > k$ to epidemics of level k through a partial cure rate of value δ_{mk}. We treat each case independently.

Given the definition of Q_{mk} in (10), we calculate the probability that node i transits from some epidemic of level $m < k$ to the epidemic of level k at time $t+1$ due to the infection of some neighbouring node $j \in V_i$ given that neighbouring node j is infected with p_m at time t. Hence,

$$\begin{aligned}
&\sum_{m<k} \sum_{\mathbf{x}_{V_i}(t)} Q_{mk} \prod_{j \in V_i} P\{x_j(t)\} P_{i,t}^m \\
&= P_{i,t}^m \left[\sum_{m<k} \sum_{\mathbf{x}_{V_i}(t)} \left(1 - \sum_{l<m} \delta_{ml}\right) \prod_{j \in V_i} (1-\beta)^{x_j(t)^T \cdot \sum_{l>k} p_l} P\{x_j(t)\} \right. \\
&\quad \left. - \sum_{m<k} \sum_{\mathbf{x}_{V_i}(t)} \left(1 - \sum_{l<m} \delta_{ml}\right) \prod_{j \in V_i} (1-\beta)^{x_j(t)^T \cdot \sum_{l \ge k} p_l} P\{x_j(t)\} \right].
\end{aligned} \tag{15}$$

If, for a neighbouring node $j \in V_i$, we set $f(x_j(t)) = P\{x_j(t)\} \cdot (1-\beta)^{x_j(t)^\top \cdot \sum_{l>k} p_l}$, $j = 1, \ldots, |V_i|$ then,

$$\sum_{\mathbf{x}_{V_i}(t)} \prod_{j \in V_i} f(x_j(t)) = \prod_{j \in V_i} \sum_{x_j(t)} f(x_j(t)).$$

The proof for the above equality is provided in Appendix B.1.

Hence,

$$\begin{aligned}
&\sum_{\mathbf{x}_{V_i}(t)} \prod_{j \in V_i} (1-\beta)^{x_j(t)^T \cdot \sum_{l>k} p_l} P\{x_j(t)\} \\
&= \prod_{j \in V_i} \sum_{x_j(t)} (1-\beta)^{x_j(t)^T \cdot \sum_{l>k} p_l} P\{x_j(t)\} \\
&= \prod_{j \in V_i} \left(1 - \beta P_{j,t}^{k+1} - \cdots - \beta P_{j,t}^K\right).
\end{aligned} \tag{16}$$

Using the approximation $(1-a)(1-b) \simeq 1-a-b$, when a and b are small, Eq. (16) can be further simplified into

$$\begin{aligned}
&\sum_{\mathbf{x}_{V_i}(t)} \prod_{j \in V_i} (1-\beta)^{x_j(t)^T \cdot \sum_{l>k} p_l} P\{x_j(t)\} \\
&= 1 - \beta \sum_{j \in V_i} \left(P_{j,t}^{k+1} + \cdots + P_{j,t}^K\right).
\end{aligned} \tag{17}$$

These findings simplify the calculation of Eq. (15). Similarly, we obtain

$$\begin{aligned}
&\sum_{\mathbf{x}_{V_i}(t)} \prod_{j \in V_i} (1-\beta)^{x_j(t)^T \cdot \sum_{l \ge k} p_l} P\{x_j(t)\} \\
&= 1 - \beta \sum_{j \in V_i} \left(P_{j,t}^{k} + P_{j,t}^{k+1} + \cdots + P_{j,t}^K\right)
\end{aligned} \tag{18}$$

and, therefore, Eq. (15) simplifies to

$$\begin{aligned}
&\sum_{m<k} \sum_{\mathbf{x}_{V_i}(t)} Q_{mk} \prod_{j \in V_i} P\{x_j(t)\} P_{i,t}^m \\
&= \sum_{m<k} P_{i,t}^m \beta \left(1 - \sum_{l<m} \delta_{ml}\right) \sum_{j \in V_i} P_{j,t}^k.
\end{aligned} \tag{19}$$

Now, we have to calculate the probability that node i will be still infected with epidemic of level k since neither infections are possible by neighbouring nodes nor does node i automatically transit to an epidemic of a lower level. Working in a similar fashion we can find

$$\begin{aligned}
&\sum_{\mathbf{x}_{V_i}(t)} Q_{kk} \prod_{j \in V_i} P\{x_j(t)\} \cdot P_{i,t}^k \\
&= \left(1 - \sum_{l<k} \delta_{kl}\right) \sum_{\mathbf{x}_{V_i}(t)} \prod_{j \in V_i} (1-\beta)^{x_j(t)^T \cdot \sum_{l>k} p_l} P\{x_j(t)\} \cdot P_{i,t}^k \\
&= \left(1 - \sum_{l<k} \delta_{kl}\right) \left[1 - \beta \sum_{j \in V_i} \left(P_{j,t}^{k+1} + \cdots + \beta P_{j,t}^K\right)\right] P_{i,t}^k.
\end{aligned} \tag{20}$$

By substituting Eqs. (19) and (20) in (14), we obtain

$$\begin{aligned}
P_{i,t+1}^k = {} & P_{i,t}^0 \cdot \beta \cdot \sum_{j \in V_i} P_{j,t}^k + \cdots \\
& + P_{i,t}^{k-1} \cdot \beta \cdot \left(1 - \sum_{l<k-1} \delta_{k-1,l}\right) \sum_{j \in V_i} P_{j,t}^k \\
& + \left(1 - \sum_{l<k} \delta_{kl}\right) \cdot \left[1 - \beta \sum_{j \in V_i} \left(P_{j,t}^{k+1} + \cdots + \beta P_{j,t}^K\right)\right] P_{i,t}^k \\
& + \sum_{m>k} \delta_{mk} P_{i,t}^m.
\end{aligned} \tag{21}$$

Eq. (21) expresses the probability of a node i being at state $S_{i,k}$ at time instant $t+1$, in terms of the probabilities of states $S_{j,m}$, ($j = 1, \ldots, M$, $m = 1, \ldots, K$) at time instant t. This interesting equation indicates the temporal and spatial (through neighbouring topology information) dependencies of the statuses of the

neighbouring nodes $j \in V_i$ to the calculation of the probability that the node i be infected with a k-level epidemic at the next time. The equation is non-linear and, therefore, it is difficult to obtain the steady state probabilities. Therefore, we substitute $P^0_{i,t} = 1 - \sum_{k=1}^{K} P^k_{i,t}$ in (21) and omit all terms that involve products of probabilities. Using this approximation we obtain the recursive equation:

$$P^k_{i,t+1} = \beta \sum_{j \in V_i} P^k_{j,t} + \left(1 - \sum_{l<k} \delta_{kl}\right) P^k_{i,t} + \sum_{m>k} \delta_{mk} P^m_{i,t}. \tag{22}$$

The probability in Eq. (22) indicates that the status of the node i at time $t+1$ depends linearly to the probability of its status at time t, to the fully and partial cure rates of node i and to the the probabilities of the statuses of each neighbouring node $j \in V_i$ at time t. Hence, we can recursively calculate the probability of node i to be infected with epidemic of some level $1 \le k \le K$, $P^k_{i,t+1}$ at some time t by starting from an initial status of the node i at time $t = 0$, $P^k_{i,0}$. In our example, all attendees at time $t = 0$ are not infected with position information (i.e., $P^k_{i,0} = 0, \forall k > 0$) except for some attendees, who are able to estimate their position (i.e., for instance $P^k_{i,0} = 1$, for some $1 \le k \le 3$), and thus disseminate such pieces of information throughout the neighbouring attendees.

We now define the following vector for all the network nodes:

$$\mathbf{P}^k_t = \begin{bmatrix} P^k_{1,t} & P^k_{2,t} & \cdots & P^k_{M,t} \end{bmatrix}^T.$$

Using this definition then Eq. (22), for $i = 1, \ldots, M$, is written in compact form as

$$\begin{aligned} \mathbf{P}^k_{t+1} &= \beta A \mathbf{P}^k_t + \left(1 - \sum_{l<k} \delta_{kl}\right) I \mathbf{P}^k_t + \sum_{m>k} \delta_{mk} \mathbf{P}^m_t \\ &= \left(\left(1 - \sum_{l<k} \delta_{kl}\right) I + \beta A\right) \mathbf{P}^k_t + \sum_{m>k} \delta_{mk} \mathbf{P}^m_t \end{aligned} \tag{23}$$

where A is the $M \times M$ adjacency matrix that depends on network topology and I is the identity matrix. Stacking the vectors $\mathbf{P}^k_{t+1}$ on top of each other for $k = 1, \ldots, K$, we obtain

$$\begin{pmatrix} \mathbf{P}^1_{t+1} \\ \mathbf{P}^2_{t+1} \\ \vdots \\ \mathbf{P}^K_{t+1} \end{pmatrix} = \begin{pmatrix} \mathbf{A}_1 & \delta_{21} I & \cdots & \delta_{K1} I \\ \mathbf{0} & \mathbf{A}_2 & \cdots & \delta_{K2} I \\ \vdots & \vdots & \ddots & \vdots \\ \mathbf{0} & \mathbf{0} & \cdots & \mathbf{A}_K \end{pmatrix} \begin{pmatrix} \mathbf{P}^1_t \\ \mathbf{P}^2_t \\ \vdots \\ \mathbf{P}^K_t \end{pmatrix} \tag{24}$$

where

$$\mathbf{A}_k = \left(1 - \sum_{l<k} \delta_{kl}\right) I + \beta A.$$

From now on we refer to the matrix $\mathbf{A}_k$ as the *matrix of infection level k*. The $KM \times KM$ matrix in Eq. (24), is an upper triangular block matrix and, therefore, its eigenvalues are the same as the eigenvalues of the matrices $\mathbf{A}_k$ along its diagonal. On the other hand, the matrices $\mathbf{A}_k$ and A have the same eigenvectors and their eigenvalues $\epsilon_{i,\mathbf{A}_k}$ and $\epsilon_{i,A}$, $i = 1, \ldots, M$, respectively, are related as follows:

$$\epsilon_{i,\mathbf{A}_k} = 1 - \sum_{l<k} \delta_{kl} + \beta \epsilon_{i,A}, \quad i = 1, \ldots, M.$$

(see Lemma 1 in Appendix A). For an infection of level k to fade out and not become a pandemic, the vector $\mathbf{P}^k_t$ should tend to zero (component-wise) for large t. This is possible if the largest eigenvalue of $\mathbf{A}_k$ is less than one; see Lemma 2 in Appendix A. By ordering the eigenvalues of matrix $\mathbf{A}_k$ in descending order, such that $\epsilon_{1,\mathbf{A}_k} \ge \epsilon_{2,\mathbf{A}_k} \ge \cdots \ge \epsilon_{M,\mathbf{A}_k}$, the condition for the infection level k to gradually extinct is $\epsilon_{1,\mathbf{A}_k} < 1$ that is,

$$1 - \sum_{l<k} \delta_{kl} + \beta \epsilon_{1,A} < 1 \Leftrightarrow \frac{1}{\epsilon_{1,A}} > \frac{\beta}{\sum_{l<k} \delta_{kl}}. \tag{25}$$

Hence, the value

$$\theta_k = \frac{\beta}{\sum_{l<k} \delta_{kl}}$$

denotes the multi-epidemical threshold of p_k (Lemma 2; Appendix A). Specifically, p_k diminishes with an inverse rate of the partial or full cure rates $\delta_{k0}, \ldots, \delta_{k,k-1}$. This generalizes the epidemic threshold in mono-epidemical spreading, i.e., $K = 1$. Actually, from Eq. (25) we obtain the classical epidemical threshold for only one epidemic p_1, which is,

$$\theta_1 = \frac{\beta}{\delta}.$$

The mono-epidemic threshold θ_1 is also discussed in [22].

The physical interpretation of the epidemical threshold in information dissemination is as follows: in case of the mono-epidemic threshold, once a node is being infected (receives information) with a rate β lower than the rate it turns susceptible (δ) then the node cannot disseminate the received information across the network. That is because the rate at which a node transits to the probability of being susceptible ($P\{x_i(t) = p_0\}$) is higher than being at the infected state ($P\{x_i(t) = p_1\}$). Thus, for large t, the steady state probability $P\{x_i(t) = p_1\} \to 0$. According to our example, we can consider that the rate δ is the staleness rate of the piece of information p_1, which is injected into the network. Staleness rate indicates that rate at which a piece of information gets obsolete or invalid (e.g., $p_1 \neq$ 'Conference building: TDOA'). Then, p_1 is going to flood the entire network if and only if each node has at least to relay p_1 (i.e, to infect with probability β) with a rate higher than that of p_1 being stale. In case of the multi-epidemical threshold, an interesting interpretation is observed. Specifically, an epidemic of level k, e.g., the p_3 position information of our example, diminishes in a network if and only if all the (logical) conditions that satisfy the existence of p_k do not hold true. That is, p_k will flood the network once all epidemics p_l, $l < k$ of lower level remain valid until the prevalence of p_k. This implies that the rate at which p_k is disseminated through the network (e.g., the nodes relay p_3 with probability β) has to be at least higher than all the rates at which epidemics of level p_l, $l < k$ either die off (δ_{l0}) or transit to even lower level (δ_{lm}, $m < l < k$). In our example, p_3 holds true once the attendees are located in the Room 5. This implicitly means that for all attendees together the p_2 and p_1 position information holds true. Hence, p_3 can be relayed throughout the attendees of Room 5 once the probability of forwarding p_3 is higher than the rate of p_2 and p_1 epidemics turn obsolete. On the other hand, the opposite implication does not hold true. That is, once p_3 does not hold true (e.g., the attendees are no longer located in Room 5), this does not imply that p_2 does not hold true too (i.e., the attendees are not located in Floor 2). Such remarkable conclusions are discussed as follows, which indicate the need for the generalization of the epidemical threshold through the eigenvalue approach.

Several conclusions can be drawn from Eq. (25). Specifically,

- Conclusion 1: If the cure rates δ_{km} depend on the state m, i.e., $\delta_{km} = \delta_m$, or if the cure rates δ_{km} depend on the transmutation level $k - m$, i.e., $\delta_{km} = \delta_{k-m}$ then epidemic p_k dies off if p_m dies off. Hence, we can state this conclusion as a corollary:

  Corollary 1. *Let p_m be an epidemic and p_k be its transmutation, that is, $p_k \succ p_m$. Let the cure rates δ_{kl} be such that either $\delta_{kl} = \delta_l$ or $\delta_{kl} = \delta_{k-l}$. Then, epidemic p_k dies off if p_m dies off.*

  See Appendix A for proof of Corollary 1.

- Conclusion 2: For arbitrary values of δ_{kl} some epidemics may die off, i.e., these for which the matrix of infection level k has its maximum eigenvalue less than one ($\epsilon_{1,\mathbf{A}_k} < 1$), and others may become a pandemic, i.e., these for which the matrix of infection level k has at least one eigenvalue greater than one ($\exists i : \epsilon_{i,\mathbf{A}_k} > 1$). This indicates that in a multi-epidemical network there can be some epidemics that do not decay over time, but some other epidemics die off. In our example, we can have dissemination of the p_2 position information but not of the p_3, but the opposite does not hold true.
- Conclusion 3: It should be noted that Corollary 1 does not simply state that once p_k dies off then p_m dies off too, having $p_k > p_m$. Instead, for certain cure rates, p_k dies off as long as p_m decays continuously over time. For instance, if p_k denotes a piece of information that is concluded/inferred by p_m then, p_k holds true as long as p_m also holds true. Once p_m is not valid then, its corresponding implicit conclusion p_k is not valid too. In our example, recall that the (disseminated) p_3 location of an attendee is the Room 5 of the Floor 2 of the Conference building. Hence, if p_3 dies off due to e.g., the event the presentation is finished, or there is a break, or p_3 is not temporarily valid, then it does not necessarily hold true that the attendee is not located on Floor 2 (p_2) or has left the building (p_1). But, once p_1 does not hold true, then neither p_2 nor p_3 holds true.
- Conclusion 4: The threshold conditions in Eq. (25) are general and hold for any arbitrary adjacency matrix A reflecting the network topology. Therefore, the epidemical threshold for an epidemic of infection level 1 in a homogeneous network or a random Erdös–Rényi (ER) network graph [17] is $\theta_1 = \frac{\beta}{\delta} = \frac{1}{\langle d \rangle}$, where $\langle d \rangle$ is the average connectivity of nodes. Hence, in a homogeneous or random network the largest eigenvalue of the adjacency matrix A is $\langle d \rangle$. We can conclude that an epidemic p_k of infection level $k > 1$ becomes pandemic if $\theta_k > \frac{1}{\langle d \rangle}$; otherwise it fades out.

5. Analytical and simulation results

We assess the behaviour of the proposed multi-epidemical dissemination model using simulations on different networks. We examine the process of the SI^{K}S model and the application of the epidemical thresholds studied through the proposed eigenvalue approach.

As a reference of a homogeneous network, we consider a regular two-dimensional (2D) lattice. A node in a 2D lattice is represented by its coordinates (x, y), where x, y, are integers and $1 \leq x, y \leq \sqrt{M}$, M is the number of nodes. Note that a node in (x, y) has four neighbours, thus, the average node degree $\langle d \rangle$ for a 2D-lattice for inner nodes is $\langle d \rangle = 4$. We experiment with the small world network, which is generated by randomly replacing a fraction of the links of a lattice with new random links with a given *rewiring* probability f. Such a network is a hybrid form between the two limiting cases of a regular lattice ($f = 0$) and a random graph ($f = 1$) [7]. The small world network is characterized by the property of the local neighbourhood – similar to regular lattices – and by the diameter of the network, which increases logarithmically with the number of nodes — similar in random graphs. The latter property indicates that, in a small world network, it is possible to reach any two nodes through just a few hops. We apply the eigenvalue-based analysis in order to examine the multi-epidemical thresholds of the disseminated pieces of information for various network topologies.

A node i, which can generate epidemic q, disseminates q to its neighbourhood. However, it is possible for a neighbouring node $j \in V_i$ to generate p from the incoming q ($p > q$). In a similar fashion, node j can circulate p as a transmutation of q to p and so on. The prevalence of the strongest epidemic denotes the equilibrium state of the multi-epidemical network. For the analytical and simulation results we assume the diffusion of $K = 3$ epidemics with $p_3 > p_2 > p_1 > p_0$ (consider our example; p_1 for terrestrial localization, p_2 for WLAN localization, p_3 for Infrared localization). A node i transits between four states. We require that the entire network be 'infected' with p_K assuming a low number of contacts (average number of messages each node sends to its neighbors in order to propagate the epidemic).

Table 1
Simulation parameters.

Number of nodes	M
Average number of infected nodes of level p_k	$n_k(t)$
Number of epidemics	K
Initial probabilities of infection	$[P\{x_i(0) = p_k\}]_{k=0:K, i=1:M}^{\top}$
Infection rate, full cure rates, partially cure rates	β, δ_{k0}, δ_{kl}, $0 < l < k \leq K$
Rewiring probability	f
Efficiency, K-level efficiency	e, e_K

It is worth noting that with the conditional independent assumption, the number of states needed to describe $P_m(t)$ provided in Eq. (8) for a node i is proportional to the number of neighbouring nodes $|V_i|$. Since a node in a homogeneous network has $\langle d \rangle = 4$ neighbouring nodes then we need four states to calculate $P_m(t)$ independently of the number of epidemics $K = 3$. In case of the conditional dependency assumption we should need $(K+1)^{\langle d \rangle}$ states to calculate $P_m(t)$ (provided in Eq. (7)), that is 256 states for $\langle d \rangle = 4$ neighbours and $K = 3$.

We used MATLAB for all simulations (over 10000 runs) and managed to:

- compare the performance of the analytical model with the simulation results w.r.t. epidemical prevalence and decay for all epidemical levels,
- predict the coverage of a network with information p_K of the highest quality, and,
- measure the efficiency and K-level efficiency (see in the following section) of the proposed model along with the corresponding Flooding scheme.
- compare the performance of SI^{K}S model with the (local) uniform Gossip and Flooding dissemination algorithms w.r.t. a number of metrics in diverse network types (e.g., homogeneous, small-world and scale-free network models).

The simulation parameters are also listed in Table 1 for better reading.

5.1. Multi-epidemical prevalence

We, now, focus on the calculation of the probabilities $P\{x_i(t) = p_0\}$, $P\{x_i(t) = p_1\}$, $P\{x_i(t) = p_2\}$ and $P\{x_i(t) = p_3\}$ at each time t derived from the recursive equations (6)–(13). The exact analytical equations for such probabilities are provided in Appendix B.2. In our simulations, all disseminated pieces of information are considered valuable by nodes. Our aim is the study of the behaviour of the multi-epidemical spreading due to epidemical transmutation. All simulations were executed several times in order to derive average values. Fig. 3 compares the performance of the proposed model with the simulation results on the evolution of the average portion of infected nodes $n_1(t)$, $n_2(t)$ and $n_3(t)$ for the three epidemics p_1, p_2 and p_3. We use a 2D lattice network with $M = 1000$ nodes, infection rate $\beta = 0.1$, full/partial cure rates $\delta_{lk} = 0.01$, $0 \leq k < l \leq K$. The initially infected nodes are randomly chosen with probabilities [0.9, 0.06, 0.03, 0.01] for p_0, p_1, p_2 and p_3, respectively.

One can observe how accurately our model can predict the epidemical prevalence in a multi-epidemical network. Moreover, the relationship among epidemics demonstrates an interesting behaviour. The strongest epidemic (p_3) actually infects the largest portion of the network. At the beginning of the propagation process

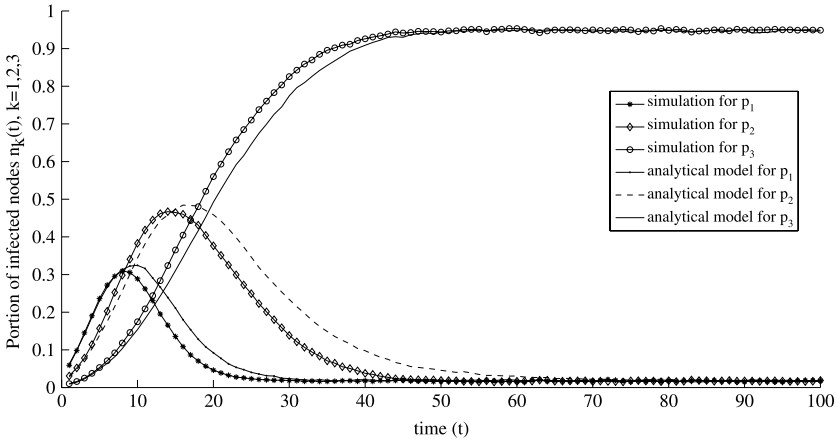

Fig. 3. Simulated and analytical epidemical prevalence of the p_1, p_2 and p_3 epidemics for $\beta = 0.1$.

it is observed that, $n_3(t) < n_2(t) < n_1(t)$. This is due to the fact that, nodes first circulate p_1, which can be further transmuted to a stronger epidemic. Evidently, in the long run, more information is accumulated in the collaborating nodes, thus, $n_2(t)$ dominates $n_1(t)$. This is attributed to the fact that nodes are reinfected with the current strongest epidemic. In the long run, the strongest epidemic p_3 infects the majority of nodes, thus, signalling the equilibrium state of the network. It should be noted that at the aggravation phase a node replaces p_k with p_m once $p_m > p_k$. For that reason $n_1(t)$ and $n_2(t)$ assume lower values than $n_3(t)$ after the pandemic propagation of p_3. The final number of nodes, which are infected with the strongest epidemic, is observed at time instance $t \geq 50$. The whole network is infected with the strongest epidemic quite rapidly with quite a small infection rate $\beta = 0.1$. This increased efficiency, which also impacts the energy condition of the nodes, justifies the selection of epidemical propagation instead of a simple flooding scheme (see the following section for a relevant discussion). Evidently, higher values of β result in faster epidemical prevalence. In addition, it is worth noting that the minor discrepancy between the analytical and simulation curves is obtained due to the assumption of the conditional independence assumption on the Eq. (7). Nevertheless, such discrepancy is not remarkable neither in the multi-epidemical prevalence nor in the multi-epidemical decay (see next section) also having a linear complexity of calculating the probability in Eq. (7) w.r.t. the number of the neighbouring nodes.

We now examine the evolution of the epidemical spreading for low values of infection rate and observe the case in which the epidemical prevalence on the network cannot be achieved. In Fig. 4 we assess the performance of the analytical model with simulations when β assumes low values ($\beta = 0.05, \beta = 0.03$) regarding the prevalence for p_1 and p_2 (we leave out p_3 for readability reasons) with initial probabilities ([0.5, 0.25, 0.25]) and $\delta_{lk} = 0.01, 0 \leq k < l \leq K$. Evidently, for low β values w.r.t. partial and full cure rates, the SI^{K}S model predicts the evolution of the epidemical spreading. It is worth noting that the epidemics cannot predominately infect the whole network for very low values of β ($\beta = 0.03$). This is very interesting especially when choosing the infection and cure rates. Specifically, the value for the infection rate highly depends on the values of partial and fully cure rates. Hence, it is of high importance to estimate such dependency in order either to study the multi-epidemical prevalence for each level of epidemic or the multi-epidemic decay of some epidemics. In the next section we study the multi-epidemical decay and, more interestingly, we propose an eigenvalue approach for estimating the epidemical thresholds and predicting the multi-epidemical decay. As it will be shown, the dependency between the infection rate β with the cure rates relates explicitly with the maximum eigenvalue of the adjacency network matrix, that is, the network topology. We also show that the dynamics of the multi-epidemical information dissemination depend highly on the topology of the network.

5.2. Multi-epidemical decay

In this section, we compare the performance of the SI^{K}S model with the simulation results on the decay of the epidemical propagation over homogeneous and small-world networks based on the eigenvalue approach. The multi-epidemical decay denotes the situation in which the epidemics cannot predominately infect the whole network and, consequently, die off. First, we consider a 2D lattice with $M = 1000$ nodes, infection rate $\beta = 0.02$, and full/partial cure rates $\delta_{ij} = 0.1$ for $i = 1, \ldots, 3, j = 0, \ldots, 2$ and $j < i$. Therefore, the largest eigenvalue of the adjacency matrix $\mathbf{A}$ of a homogeneous network is $\epsilon_{1,\mathbf{A}} = \langle d \rangle = 4$. Hence, the epidemical threshold for an epidemic p_k is $\theta_k = \frac{1}{\langle d \rangle} = 0.25, k = 1, 2, 3$, in order for p_k to be disseminated across the 2D lattice. That is, $\theta_k = \frac{0.02}{k0.1}, k = 1, 2, 3$. Having $\theta_3 < \theta_2 < \theta_1 < 0.25$ we obtain that all the three epidemics fade out. It is expected, from Corollary 1: p_3 fades out faster than p_2, and p_2 fades out faster than p_1. This is attributed to the fact that, $n_k(t)$ decays exponentially with a factor of $(\epsilon_{1,\mathbf{A}_k})^t, k = 1, 2, 3$, and $\epsilon_{1,\mathbf{A}_3} < \epsilon_{1,\mathbf{A}_2} < \epsilon_{1,\mathbf{A}_1}$. Fig. 5 shows the performance of the proposed model and the simulation for the decay of the multi-epidemical propagation. It is observed that, the curves for both the model and the simulation are much alike, and, the stronger the epidemic is the faster it fades out for the same initial probabilities $[0.3, 0.3, 0.3]^\top$ for p_1, p_2, p_3, respectively. Evidently, based on the predicted thresholds θ_k, the smaller the infection rate becomes the faster all the epidemics die off. According to our example, once the position information p_1 of a lower accuracy level does not hold true, i.e., it fades out, then all concluded information based on p_1 fades out too. Especially, the most detailed position information p_3 fades out faster than p_2 and p_1 information. Another interesting observation is that the decay curve of each p_k is inversely proportional to the (semantic) information relation $>$, i.e., $n_3(t) < n_2(t) < n_1(t) \Rightarrow p_3 > p_2 > p_1$ (see Conclusion 3 and Fig. 5).

We now examine the multi-epidemical decay in different network topologies. We consider the Watts–Strogatz (WS) network model [23] of $M = 1000$ nodes generated by a 2D lattice with rewiring probability $f \in [0, 1]$. According to our approach (see Lemma 2), in order for an epidemic $p_k, k = 1, 2, 3$ to die off in that network it must hold that:

$$\left(1 - \sum_{l<k} \delta_{kl} + \beta \epsilon_{1,\mathbf{A}}\right)^t \to 0$$

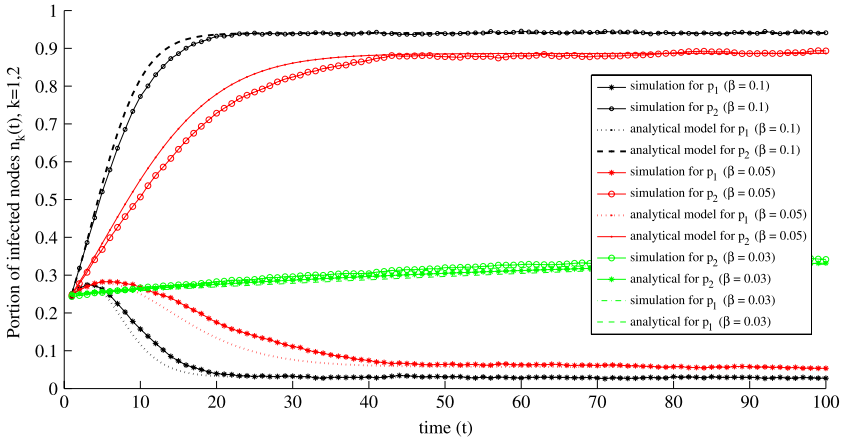

Fig. 4. Simulated and analytical epidemical evolution of the p_1 and p_2 epidemics for low values of β.

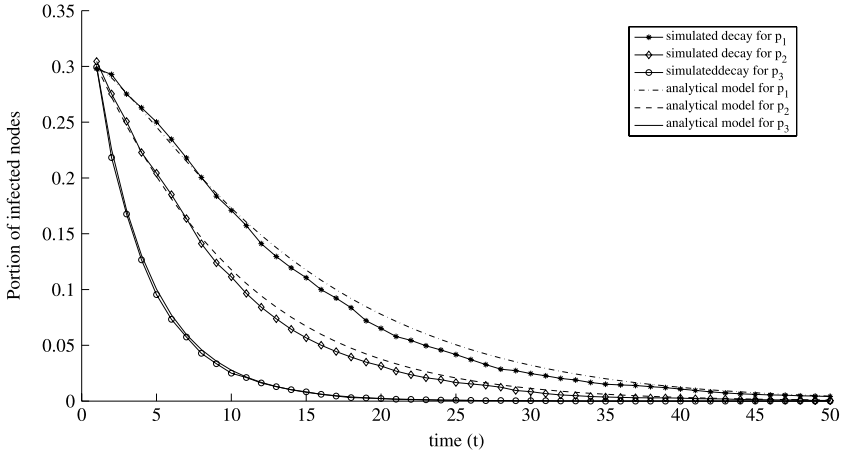

Fig. 5. Simulated and analytical epidemical decay of the p_1, p_2 and p_3 epidemics. Observe that, the stronger the epidemic is the faster it fades out.

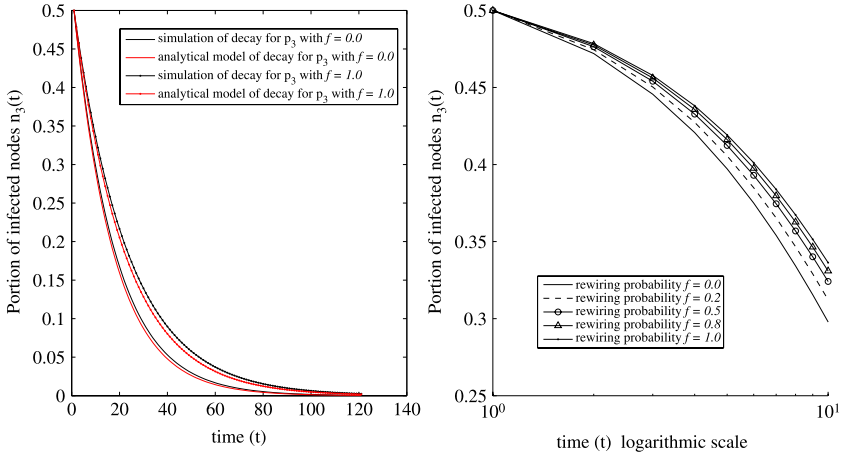

Fig. 6. (Left) Analytical and simulated epidemical decay for p_3 based on the eigenvalue approach for networks with rewiring probability $f = 0.0$ (regular lattice), and $f = 1.0$ (random network). (Right) Analytical decay for p_3 with $f = (0.0, 0.2, 0.5, 0.8, 1)$.

for large t. The maximum eigenvalue (after 10,000 iterations) of the WS adjacency matrix **A** with values of $f = (0.0, 0.2, 0.5, 0.8, 1)$ is estimated at $\epsilon_{1,\mathbf{A}}(f) = (3.8380, 4.1132, 4.3322, 4.4543, 4.5505)$, respectively, assuming epidemical thresholds for $p_3(0.2606, 0.2431, 0.2308, 0.2198)$, respectively. In a WS network, in which each node links to d neighbours, we have that $\langle d \rangle = d$. Fig. 6(left) shows for $f = 0.0$ and $f = 1.0$ the decay of the p_3 epidemic as predicted by our eigenvalue approach and as derived from the corresponding simulations (similar curves correspond to p_1, p_2). The simulation setting is: infection rate $\beta = 0.02$ and full/partial cure rates $\delta_{ij} = 0.1$ for $i = 1, \ldots, 3, j = 0, \ldots, 2; j < i$. A low rewiring probability f gives a network that approaches a regular lattice network and all epidemics die off faster in that WS network comparing to high values of f (random network). Specifically, in a WS network with low values of f, the decay rate (mean value) for the highest K-level epidemic is 46% higher than that of a WS network with high values of f. This is also illustrated in Fig. 6(right) for $f = (0.0, 0.2, 0.5, 0.8, 1)$. The proposed eigenvalue approach

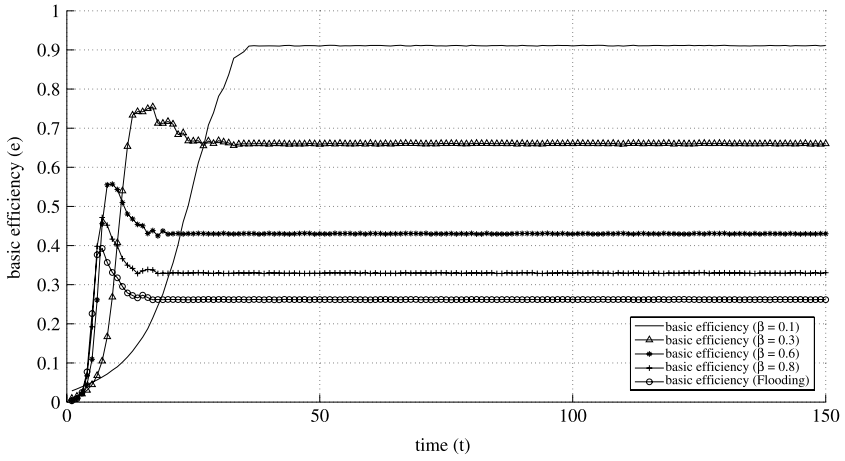

Fig. 7. Basic efficiency (e) for SI^{K}S and Flooding scheme for various values of β.

for predicting the epidemical decay is considered appropriate for any arbitrary adjacency matrix (network topology) − see Conclusion 4.

5.3. Model efficiency

We now examine the information dissemination efficiency of the proposed model, that is, we focus on the percentage of nodes that were infected but with the lowest network message overhead. We define the basic efficiency $e \in [0, 1]$ as the portion of nodes $n_k(T)$ infected with some $p_k, 0 < k \leq K$ (see Section 5.1) out of the total number of contacts (for message passing) $c_{0k}(T)$ of those nodes to transit from p_0 to all p_k at the end of the simulation time horizon T. Hence,

$$e = \frac{1}{K}\sum_{k=1}^{K}\frac{n_k(T)}{c_{0k}(T)}. \tag{26}$$

Efficiency e should be close to unity. Since the infected nodes may be re-infected by stronger epidemics, we define the l-level efficiency $e_l \in [0, 1]$ as the portion of the infected nodes $n_{lk}(T)$ transiting from p_l to all $p_k, 0 < k \leq K, 0 < l < k$ out of the number of total contacts $c_{lk}(T)$, that is,

$$e_l = \frac{1}{K-l}\sum_{k=l+1}^{K}\frac{n_{lk}(T)}{c_{lk}(T)}.$$

The K-level efficiency is defined as the sum of all e_l for $0 < l \leq K$, that is,

$$e_K = \frac{1}{K}\sum_{l=1}^{K} e_l. \tag{27}$$

Similarly, e_K should also assume a high value indicating that nodes efficiently transit between middle-level epidemics in order to be infected with p_K. The e_K metric denotes the efficiency of a multi-epidemic algorithm in disseminating information once the nodes are, at least, infected with some epidemic. Note that, if we set $l = 0$ in e_l, we obtain that $e_0 = e$, and for $K = 1$, we obtain 1-level efficiency (the known efficiency metric in information dissemination).

Based on the e and e_K metrics we examine the efficiency of the SI^{K}S and Flooding schemes. All nodes convey and spread information across the network with infection rate $0 < \beta < 1$. We get the Flooding scheme when $\beta = 1$, that is, a node always disseminates information, if it exists, upon contact with a neighbouring node. We measure the basic efficiency e for SI^{K}S and Flooding for various values of infection rates having the cure rates constant $\delta_{ij} = 0.01$ for $i = 1, \ldots, 3, j = 0, \ldots, 2, j < i$. Specifically, for SI^{K}S we set values for $\beta = (0.1, 0.3, 0.6, 0.8)$ and in Flooding we set $\beta = 1$. Fig. 7 plots the efficiency metric e for different values of β until the model converges. One can observe that the epidemical spreading assumes high values of e which converge at 0.8820 for $\beta = 0.1$, while, in flooding, the efficiency is $e = 0.2628$. This denotes that even with low infection rates, the epidemical spreading is quite efficient for disseminating information across the network.

In addition, Fig. 8 depicts the K-level efficiency e_K for various values of β. e_K assumes higher value in SI^{K}S than in Flooding, which is more efficient in disseminating information to nodes that have already been infected with some epidemic. Specifically, in SI^{K}S we obtain convergence values of $e_K = (0.4660, 0.3393, 0.2182, 0.1651)$ for infection rates $\beta = (0.1, 0.3, 0.6, 0.8)$, respectively, and in Flooding $e_K = 0.1353$.

In Fig. 9 we depict both efficiency metrics for various values of $\beta = 0.1..1$ after 10,000 simulations in order to compare them. Observe that the e_K always assumes lower values than e since the SI^{K}S model attempts to propagate and infect the whole network with the highest quality of information. This induces an additional load once infected nodes are willing to be infected with more valuable information, if it exists. By adopting the flooding scheme, both efficiency metrics assume the lowest values.

5.4. Comparison with other models

We experiment with the Gossip algorithm which falls in the same broader category of information dissemination algorithms. The Gossip algorithm is characterized by its distributed nature and robustness to network dynamics. In a Gossip algorithm each node picks, according to some underlying deterministic or random rule, another neighbouring node and exchanges information (a.k.a. rumour) with it. Two basic schemes are discussed in the literature for neighbour selection: the uniform gossip, in which each node chooses to communicate with a randomly chosen node at each step [9], and the standard gossip, in which a node picks, according to a probabilistic distribution, one of its immediate neighbours [6,24]. In addition, the authors in [10] propose the spatial gossip algorithm, where node selection is based on a probability inversely proportional to the distance between nodes.

In the multi-epidemic dissemination, the exact knowledge on the entire network's structure is not individually known to each node. Instead, each node i can only locally communicate with its neighbouring nodes (V_i) and exchange information with them. We compare the SI^{K}S model with the local (uniform) Gossip model and the Flooding model. Specifically, in the local gossip model, a node i uniformly chooses to communicate with

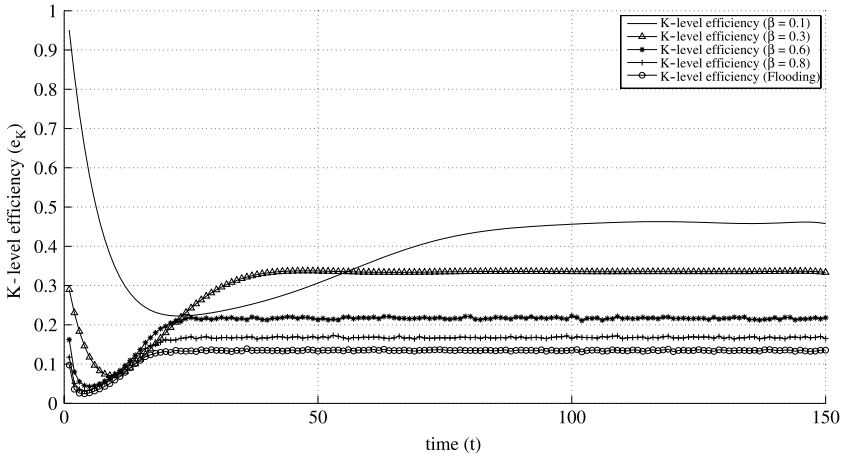

Fig. 8. K-level efficiency (e_K) for SI^{K}S and Flooding scheme for various values of β.

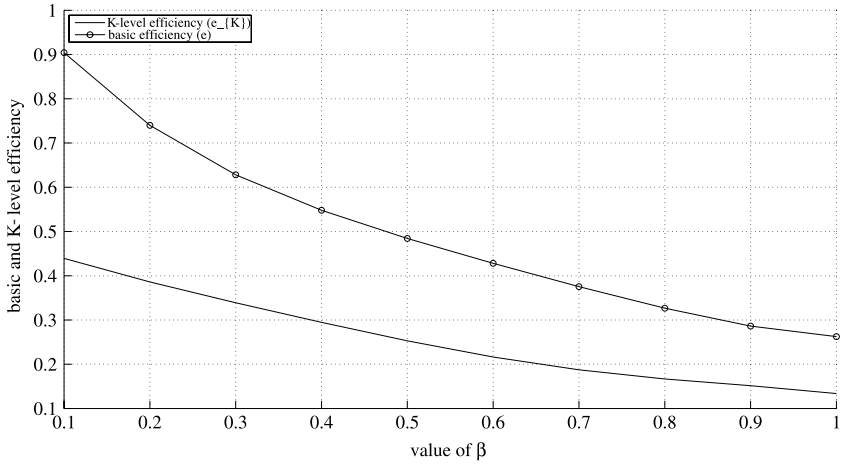

Fig. 9. The efficiency metrics e_K and e for the SI^{K}S and Flooding schemes ($0.1 \leq \beta \leq 1$).

one neighbouring node $j \in V_i$; in the Flooding model, a node i communicates deterministically with all neighbouring nodes. In the multi-epidemic dissemination the nodes have to efficiently exchange information of intermediate levels in order for the K-level information to cover the entire network as soon as possible. Besides the K-level efficiency and the basic efficiency metrics for comparative performance assessment, additional performance criteria that we adopt for assessing the various schemes are: *time average of global infection status* with epidemic of any level $k > 0$ and *efficient information delivery* in terms of information content transmission. The time average of global infection status metric $\gamma_t \in [0, 1]$ denotes the time average percentage of the number of nodes $n_k(t), 1 \leq k \leq K$ that have been infected by k-level epidemic up to time t. Since, $n_0(t) = 1 - \sum_{k=1}^{K} n_k(t)$ we obtain,

$$\gamma_T(t) = 1 - \frac{1}{t}\sum_{\tau=0}^{t}(n_0(\tau))$$

where T is the total simulation time. For each model we assess the capability of infecting a large number of nodes with epidemic(s) in a given time horizon T. More specifically, the $\gamma_T(t)$ metric for a dissemination model expresses the rate of infection up to time t; high (low) rate is obtained once γ_t is close to unity (zero).

In order to define the efficient information delivery metric, we have to determine whenever a message is meaningful for a recipient node or not. Consider an infected node i by an epidemic of level $l > 0$, which at time t sends an l-level virus (message) to its neighbouring node $j \in V_i$. The message is considered 'meaningful' for the node j if the node j is either susceptible or infected by epidemic of level $0 < u < l$ at time t. Otherwise, the message is 'meaningless' for the node j. Let $m_{ul}(T)$ be the total mean number of the meaningful messages over T that are sent for transmuting epidemic of level u to level l in time horizon T. Hence, efficient information exchange involves the dissemination of as many meaningful messages as possible. We define the efficient information delivery metric $\gamma_K \in [0, 1]$ for delivering an epidemic of infection level K through intermediate multiple infections of lower epidemic levels as

$$\gamma_K = \frac{1}{K}\sum_{l=1}^{K}\left(\sum_{u=0}^{l}\frac{m_{ul}(T)}{c_{ul}(T)}\right)$$

where $c_{ul}(T)$ is the total number of contacts (transmission of meaningful and meaningless messages) that are used for transiting nodes from p_u to $p_l, 0 < u \leq K, 0 < l < u$ epidemic within time horizon T (see also Section 5.3). The ultimate difference of the efficiency metrics (K-level efficiency e_K and basic efficiency e) with the $\gamma_T(t)$ and γ_K metrics is that the latter metrics examine the capability of a model in propagating as much meaningful information content as possible within a given time horizon. On the other hand, the efficiency metric generally counts the portion of the infected nodes out of the required contacts irrespectively of the absolute coverage of infected nodes ($\sum_{k=1}^{K} n_k(t)$ out of M) and the number of meaningless information delivery messages ($c_{kl}(t) - m_{kl}(t)$). Therefore, we have to consider all the metrics in order to objectively compare the dissemination models. We, then,

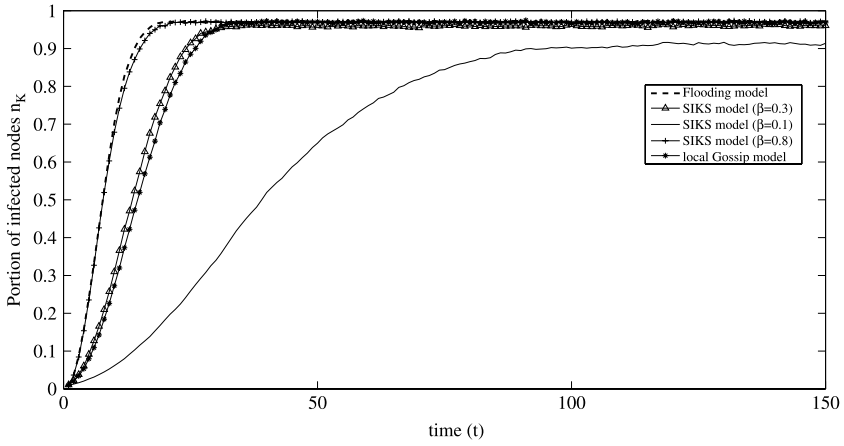

Fig. 10. Infection rate of the K-level epidemic for the SI^{K}S, local Gossip and Flooding models.

define a holistic metric $\gamma \in [0, 1]$ for a dissemination model, which indicates time average coverage of infected nodes along with efficient information content transmission with in time horizon T, thus,

$$\gamma = \omega\gamma_T(T) + (1 - \omega)\gamma_K. \tag{28}$$

The $\omega \in [0, 1]$ weight balances the importance between $\gamma_T(T)$ and γ_K. A value of γ close to unity denotes that the dissemination model can infect the entire network with an epidemic of the highest level in an efficient manner early in time. We compare the models SI^{K}S, local Gossip and Flooding w.r.t. the γ ($\omega = 0.5$) and efficiency metrics (K-level efficiency and basic efficiency) within homogeneous 2D lattice networks, small-world (WS) networks and scale-free networks (see Section 5.4.3).

At first we examine the behaviour of the portion n_K for the three models concerning the epidemic of K level. Fig. 10 depicts the evolution of n_K for $M = 1000$ nodes, time horizon T, and $\delta_{lk} = 0.01, 0 \leq k < l \leq K = 3$ (the epidemics of level lower than K are not illustrated for readability reasons). We can observe that the rate of infection in the local Gossip is between the rate of infection in the SI^{K}S model (for diverse values of β) and that of the Flooding model ($\beta = 1$). Especially, the local Gossip model infects the entire network earlier than the SI^{K}S model with $\beta <$ 0.3.This behaviour is also reflected in the evaluation of the K-level efficiency, basic efficiency and γ metric, as discussed later.

5.4.1. Homogeneous network type

We assess the performance of the γ metric in 2D lattice networks (10,000 simulations) for the three models. Fig. 11 depicts the γ metric for diverse values of β. The γ metric for the local Gossip model is also illustrated for comparison reasons. One can observe that for a low value of β ($\beta = 0.1$) both SI^{K}S and local Gossip assumes quite similar values w.r.t. γ. The Flooding model assumes a very high γ value ($\beta = 1$). That is because of the extremely high rate of infection of the entire network with epidemic(s). However, we have to examine the K-level efficiency and the basic efficiency for all models, as reported in Table 2. Table 2 reports the efficiency metrics for the models in 2D lattice networks. We can observe that for $\beta < 0.3$ the SI^{K}S model appears more efficient than the local Gossip model w.r.t. basic efficiency. For $\beta > 0.3$ the local Gossip model efficiently disseminates epidemics compared to SI^{K}S and Flooding. The Flooding model exhibits the worst performance w.r.t. the basic efficiency metric. Similar conclusions for the SI^{K}S and local Gossip models can be drawn for the K-level efficiency (see Table 2).

Table 2
Efficiency e and e_K metrics for 2D network model.

Model		e	e_K
SI^{K}S model	β		
	0.1	0.90	0.44
	0.2	0.74	0.38
	0.3	0.63	0.33
	0.4	0.53	0.29
	0.5	0.49	0.25
	0.6	0.42	0.20
	0.7	0.37	0.19
	0.8	0.33	0.16
	0.9	0.28	0.14
Flooding model	1	0.26	0.13
Local Gossip model		0.65	0.32

5.4.2. Small-world network type

We also experiment with WS networks models in which the rewiring probability ranges from $f = 0$ (homogeneous network) to $f = 1$ (totally random network). Fig. 12 depicts the γ metric for the three models vs. f for diverse β values. We can observe that the γ value difference between the local Gossip and SI^{K}S model is greater than that in a 2D lattice network. Specifically, for $0.1 < f \leq 1$ (less regular network models) the γ metric for the local Gossip is 10.14% to 24.08% lower than that of the SI^{K}S model (mean value for all β). In addition, as the f value increases the SI^{K}S model tends to the same γ value as that of the Flooding model ($\gamma = 0.78$), while the local Gossip model tends to assume a constant γ value ($\gamma = 0.61$). One can conclude that in small-world networks the SI^{K}S model exhibits better performance in γ metric than the local Gossip model for all β values. We have also to consider the efficiency metrics for WS network models as illustrated in Fig. 13. Fig. 13 depicts the efficiency metrics for each model vs. the rewiring probability f. We can observe that the e_K (Fig. 13(a)) and e (Fig. 13(b)) metric remains constant with f for all models. In addition, the basic efficiency e for the local Gossip model assumes a higher value than that of the SI^{K}S model when $\beta > 0.3$ and Flooding. For $\beta < 0.3$ the SI^{K}S model appears the more efficient model, while the Flooding model assumes the lowest value in e and e_K. Similar conclusions on the SI^{K}S and local Gossip models hold for the K-level efficiency.

5.4.3. Scale-free network type

Many networks are classified as scale-free networks, meaning that they have power-law node degree distributions, while random network models such as the Erdös-Renyi (ER) model and the Watts–Strogatz (WS) model do not exhibit power laws. The Barabasi–Albert (BA) [2] network algorithm generates a scale-free

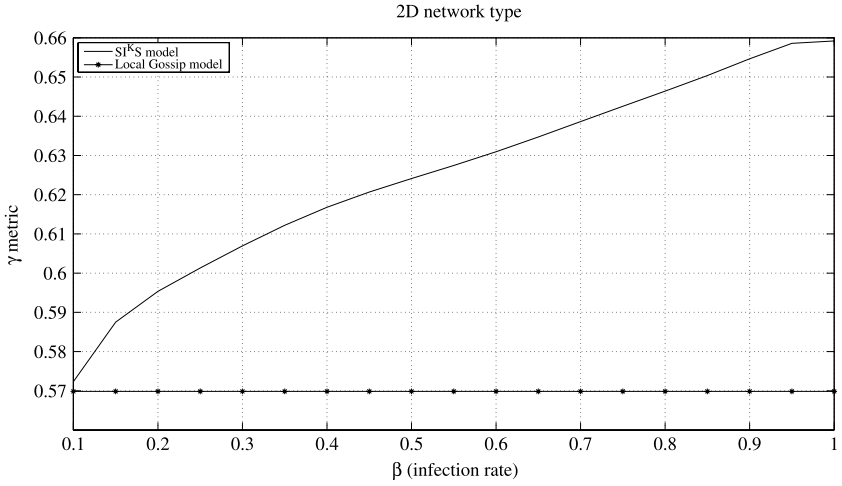

Fig. 11. The γ metric vs. infection rate β in 2D network model.

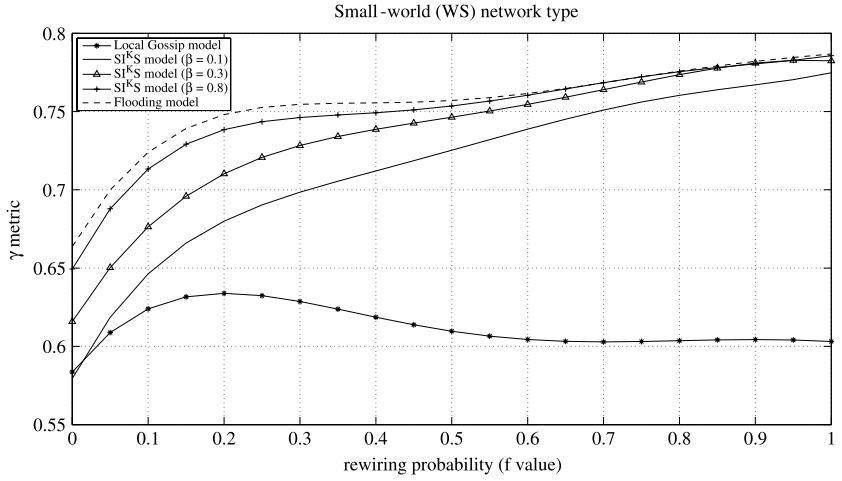

Fig. 12. The γ metric vs. rewiring probability f in WS network model.

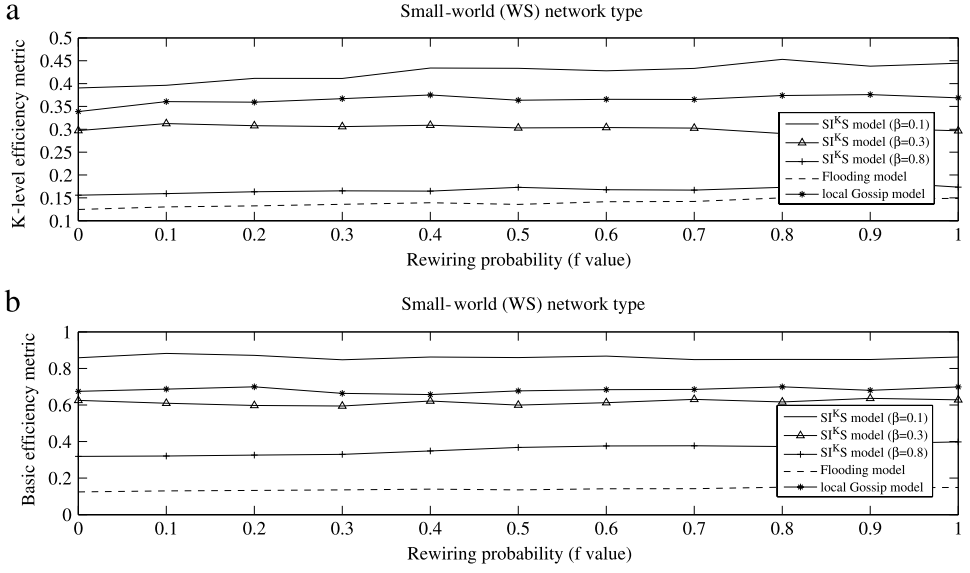

Fig. 13. The e and e_K metrics vs. rewiring probability f in WS network model.

network based on the most important characteristic 'preferential attachment'. The 'preferential attachment' characteristic denotes that the more connected a node is, the more likely it is to receive new contacts. Nodes with a higher degree $\langle d \rangle$ have a stronger ability to *grab* contacts added to the network. The BA algorithm is as follows: The network begins with an initial network of M_0 nodes.

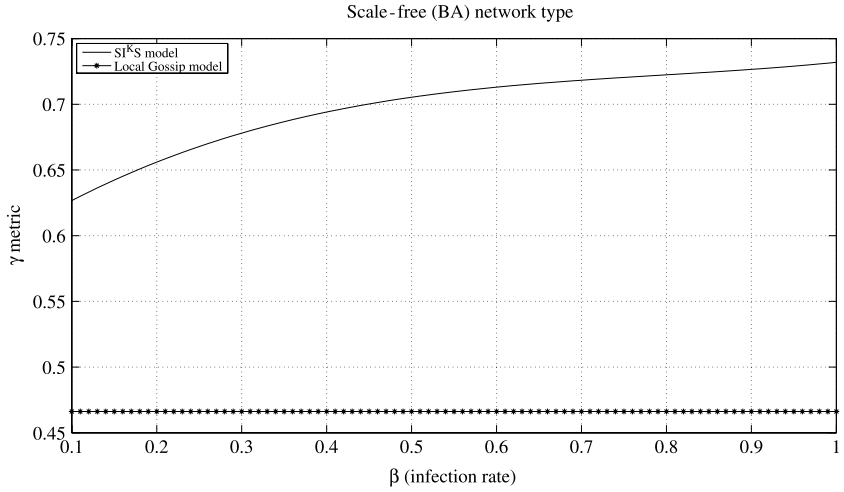

Fig. 14. The γ metric vs. infection rate β in BA network model.

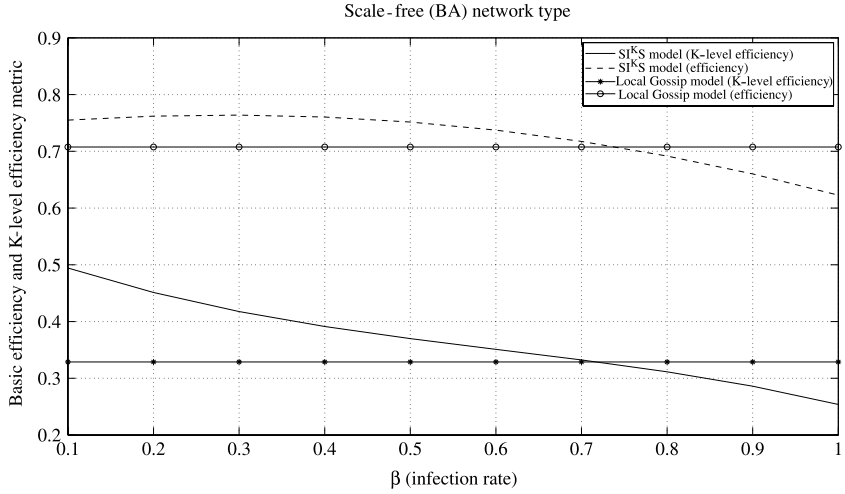

Fig. 15. The e and e_K metrics vs. infection rate β in BA network model.

New nodes are added to the network one at a time. Each new node is connected to some existing node with a probability proportional to the number of links that the existing node already has. That is, the probability $P(i)$ that the new node is connected to an existing node i is $P(i) = \frac{\langle d\rangle_i}{\sum_j \langle d\rangle_j}$, where $\langle d\rangle_i$ is the degree of node i. It is worth noting that heavily linked nodes tend to quickly accumulate even more links, while nodes with only a few links are unlikely to be chosen as the destination for a new link; the new nodes have a 'preference' to attach themselves to the already heavily linked nodes. The $P(\langle d\rangle)$ distribution resulting from the BA model is of the form $P(\langle d\rangle) = \langle d\rangle^{-\lambda}$ with exponent λ usually between 2 and 3. We assess the performance of the SI^{K}S, Flooding and local Uniform Gossip w.r.t. γ and efficiency metrics in diverse BA scale-free networks, starting with $M_0 = 10$ nodes (in initialization of the BA network model) until $M = 1000$ nodes.

Fig. 14 depicts the γ metric for the three models vs. β. We can observe that the γ value of the SI^{K}S model is 23.8% to 54.6% greater than that of the local Gossip as β increases. This denotes that in scale-free networks the epidemic-based information dissemination scheme achieves a high diffusion speed along with a high number of meaningful messages over the transactions compared with the local Gossip-based scheme. In addition, the Flooding scheme assumes the highest γ value ($\gamma = 0.73$). Moreover, we can observe from Fig. 14, that for a low β value, the SI^{K}S model assumes very higher γ value than local Gossip, which such difference is not so remarkable considering the WS and ER network models (see previous section).

Finally, we examine the efficiency metrics for BA network models as illustrated in Fig. 15. Fig. 15 depicts the efficiency metrics for each model vs. the infection rate β. We can interestingly observe that the e_K value is at most 12% higher for the SI^{K}S model compared with the local Gossip model for $\beta < 0.74$, approximately. For $\beta > 0.74$ the local Gossip model assumes a better performance on the efficiency metric especially when compared with the Flooding scheme (15% higher). We can observe a similar behaviour on the K-level efficiency. Specifically, for $\beta < 0.73$, approximately, the SI^{K}S model appears more efficient in disseminating intermediate epidemics in order for the strongest epidemic to prevail in a BS network than the local Gossip model. On the other hand, for $\beta > 0.73$, the local Gossip model assumes a slightly better performance on e_K compared with the SI^{K}S model. In addition, the Flooding model assumes the lowest K-level efficiency in BA networks.

Overall, we can conclude that, concerning the γ metric, the SI^{K}S model can efficiently disseminate epidemics through meaningful messages in a 2D lattice, WS and BA network models, when β assumes high values. On the other hand, concerning the e and e_K metrics, the local Gossip model appears more efficient than the SI^{K}S model with infection rate $\beta > 0.3$ for 2D lattice and WS network models. In case of BA networks, the local Gossip appears sightly more efficient w.r.t. SI^{K}S only for relatively high β

values ($\beta > 0.7$) but with no remarkable difference. Hence, the SI^{K}S model with $\beta < 0.3$ appears the best performance both in γ and efficiency metrics for 2D lattice and WS network models. More interestingly, for $\beta < 0.7$, the SI^{K}S model assumes the best performance w.r.t. γ, e, e_K in BA networks.

6. Discussion

In this section we discuss possible implementations of the proposed multi-epidemical model in information dissemination schemes.

The SI^{K}S model could be adopted by applications which collaboratively share pieces of information in a network. The authors in [15] adopted the SIR model for sharing parts (chunks) of files in a peer-to-peer (P2P) file sharing overlay. In such model the nodes (peers) could be either idle, downloading or sharing. A downloading peer is in the infected state (I), an idle peer is in the susceptible state (S) and the sharing peer is in the removed state (R). The authors in [15] report that in a P2P network a file is divided into K chunks and, thus, a downloading peer transits from diverse sub-states I_k of the infected state I. That is, each sub-state I_k denotes that the peer has partially downloaded the file.This involves a transition from I_k to I_{k+1} state with some rate upon the reception of the $k+1$ chunk. This resembles to the partially infected states in our model, meaning that a peer that has downloaded the kth chunk of a file is infected with the p_k epidemic. Once a peer downloads the last chunk of the file (i.e., infected with p_K) then it becomes the most infectious peer. This means that the peer contains the whole file and can make it available to the network. However, such node can also transit to the idle state (leaves the system — becomes a leecher);for reasons of simplicity, the model in [15] assumes that all peers remain active after a completed download. Our model can be adopted to predict the corresponding dynamics of the chunks dissemination process, i.e., steady-state probabilities, the percentage of idle, downloading and sharing peers, assuming that the sharing node does not leave the network. In addition, our model covers the transitions of the downloading peers among partially infected states. This is achieved by exploiting the infection rate from p_k to p_m depending on the states of the neighbouring peers, which is not included in [15]. Finally, the SI^{K}S model can also cover the behaviour of multi-epidemical decay in a P2P network of arbitrary topology based on the partial cure rates (e.g., due to the peer's malfunction), which is not considered in [15].

The NewsNet application is based on the proposed architecture in [20]. NewsNet depends on the user having had a connection to a fixed network. If a user meets another NewsNet user, then the one with more up-to-date or complementary piece of news sends it to the other one. In this way a user is able to receive a news update without actually connecting to the network. NewsNet exploits social groupings of people in which users share similar interests and preferences. We can apply our model by assuming that the p_k piece of news can be either upgraded, replaced or extended by another exchanged up-to-date or complementary p_m piece. Hence, we can study the corresponding dynamics (prevalence and decay) for the news dissemination process. A similar application of our model is discussed in [4], where the authors adopt the SI^{2}S model for collaborative multimedia dissemination. The disseminated information covers software/multimedia content meta-data, and semantic content specification files (e.g., Composite Capabilities/Preference Profiles (CC/PP), User Agent Profile (UAProf)) for wireless multimedia devices and mpeg7 files. Based on such model, the nodes exploit multimedia semantics and, thus, share semantically enhanced multimedia content among them.

If we consider contextual information (context) dissemination as a specific information dissemination issue, one could adopt and extend our model for dealing with spreading of multiple types of context. Of course, temporal, spatial and other specific constraints regarding context dissemination can be taken into account, which are not in the scope of our analytical model. However, in [3] we carried out simulations of the SI^{3}S model for context dissemination regarding the mobility behaviour of the nodes, which is not covered in the current analytical model. Such simulations concern context-aware applications that exchange the context captured by their sensors. The simulated SI^{3}S model relies on semantic dependencies among pieces of context (an hierarchical representation scheme is assumed), the temporal validity of the exchanged context, the operation status of the sensors and the mobility of nodes. In addition, the authors in [21] proposed a model for collaborative exchange of context in a network. However, the nodes may not always have the capability to sense their environment and infer their exact context. Thus, the applications carried by such nodes may not be able to obtain the needed input for execution. Significant economies of scale can be achieved through such model. i.e., not all nodes need to carry the same, overlapping set of sensors or expensive, fault resistant components. Missing or erroneous context can be substituted by other nodes, thus, leading to truly dependable applications. Our model studies the dissemination of diverse dependent pieces of context, thus, it can be adopted in [21] since the concept of information completion and inference is introduced in the multi-epidemical information dissemination.

7. Conclusions

In this work, we introduced an analytical information dissemination model. We exploit the analogy between information and epidemic and extend the epidemiological model SIS to the SI^{K}S, in the sense that: an infected node can be re-infected by stronger epidemic or generate new information, thus, aggravating its condition and can disseminate such epidemic by introducing the concept of transmutation. A stronger epidemic matches with preferable piece of information, denoting that each node can be re-infected with more valuable (e.g., up-to-date, of better quality) information than that it has been recently infected. Hence, multiple epidemics can circulate across the network. As a result, a node is able to autonomously decide whether to augment its local knowledge or not, thus, behave more intelligently.

We have presented a spatio-temporal model to study the dynamics of a multi-epidemical spreading. We model the SI^{K}S epidemical prevalence through a Markov process. In the case of mono-epidemical spreading (i.e., no transmutation is permitted) we obtain the exact mathematical model studied in [25]. In our generalized model, each epidemic assumes a different spreading behaviour and we show that such behaviour may affect the spreading pattern of its transmutations. We also investigate the epidemical decay through an eigenvalue-based approach. We found the relations among the epidemical thresholds of the transmuted epidemics and observed that an epidemic p_k dies off once the largest eigenvalue of the corresponding matrix of infection level k is less than one. Such an approach can be applied on arbitrary network graphs. In the case of a mono-epidemical propagation, the resulting threshold derived from our model is the classical epidemical threshold studied in [22]. Moreover, the behaviour of the proposed multi-epidemical model is assessed through analysis and extensive simulations on regular lattices, small-world networks and scale-free networks. Finally, we examine the efficiency and the introduced K-level efficiency of the proposed model. We show that our model is more efficient in disseminating pieces of information to nodes, which have been already infected with some epidemic, than the Flooding scheme and, in some cases, the uniform Gossip. Moreover, we experiment with certain metrics and show the applicability of our

model in information dissemination w.r.t. the uniform Gossip and Flooding schemes. We also discuss possible implementations of the proposed multi-epidemical model in information dissemination schemes. Our ongoing work is an analytical model, which takes into consideration the mobility behaviour of nodes, through which we can study the multi-epidemical prevalence and decay. Moreover, the scheme of epidemical transmutation will be studied in the information theoretic framework. The entropy analysis of such a mechanism is expected to provide important results for the operation of the network as a whole.

Appendix A

Lemma 1. *The matrices $\mathbf{A}_k$ and A have the same eigenvectors, and their eigenvalues $\epsilon_{i,\mathbf{A}_k}$ and $\epsilon_{i,A}$, $i = 1, \ldots, M$, respectively, are related as follows:*

$$\epsilon_{i,\mathbf{A}_k} = 1 - \sum_{l<k} \delta_{kl} + \beta\epsilon_{i,A}, \quad i = 1, \ldots, M.$$

Proof of Lemma 1. Let $\mathbf{r}_{i,A}$ be the eigenvector of A which corresponds to the eigenvalue $\epsilon_{i,A}$. By definition we have that $A\mathbf{r}_{i,A} = \epsilon_{i,A}\mathbf{r}_{i,A}$; note that the A is a symmetric adjacency matrix. Now,

$$\begin{aligned}\mathbf{A}_k\mathbf{r}_{i,A} &= \left(\left(1 - \sum_{l<k}\delta_{kl}\right)I + \beta A\right)\mathbf{r}_{i,A}\\ &= \left(1 - \sum_{l<k}\delta_{kl}\right)\mathbf{r}_{i,A} + \beta\epsilon_{i,A}\mathbf{r}_{i,A}\\ &= \left(1 - \sum_{l<k}\delta_{kl} + \beta\epsilon_{i,A}\right)\mathbf{r}_{i,A}.\end{aligned}$$

Hence, $\mathbf{r}_{i,A}$ is also an eigenvector of $\mathbf{A}_k$, which corresponds to the eigenvalue $1 - \sum_{l<k}\delta_{kl} + \beta\epsilon_{i,A}$, $i = 1, \ldots, M$. □

Lemma 2. *The epidemical threshold for the epidemic p_k in a multi-epidemical network of K levels is $\theta_k = \frac{\beta}{\sum_{l<k}\delta_{kl}}$, $k = 1, \ldots, K$.*

Proof of Lemma 2. Consider the $KM \times KM$ block system matrix in Eq. (24), $\mathbf{U}_K$, which is an upper triangular block matrix. Hence, the eigenvalues of $\mathbf{U}_K$ are the same as the eigenvalues of the matrices of infection level $k = 1, \ldots, K$, $\mathbf{A}_k$, along the diagonal of $\mathbf{U}_K$. If we consider the column vector $\mathbf{R}_{t+1} = (\mathbf{P}^1_{t+1}, \ldots, \mathbf{P}^K_{t+1})$, then we can have the recursive multi-epidemic system equation:

$$\mathbf{R}_{t+1} = \mathbf{U}_K\mathbf{R}_t = \mathbf{U}^t_K\mathbf{R}_0. \quad \square$$

By using the spectrum decomposition, we obtain:

$$\mathbf{U}_K = \sum_{j=1}^{KM} \epsilon_{j,\mathbf{U}_K}\mathbf{u}_{j,\mathbf{U}_K}\mathbf{u}^\top_{j,\mathbf{U}_K}$$

and

$$\mathbf{U}^t_K = \sum_{j=1}^{KM} \epsilon^t_{j,\mathbf{U}_K}\mathbf{u}_{j,\mathbf{U}_K}\mathbf{u}^\top_{j,\mathbf{U}_K}$$

where $\mathbf{u}_{j,\mathbf{U}_K}$ and $\epsilon_{j,\mathbf{U}_K}$ are the eigenvectors and eigenvalues of the $\mathbf{U}_K$ matrix, respectively. The eigenvalues of $\mathbf{U}^t_K$, for a time t, are equal to $\epsilon^t_{j,\mathbf{U}_K}$. From this it follows that $\lim_{t\to\infty}\mathbf{U}^t_K = \mathbf{0}$ if and only if all eigenvalues of $\mathbf{U}_K$ are strictly less than unity;based on spectrum decomposition, $\mathbf{R}_t \to \mathbf{0}$ once the sum of $\epsilon^t_{j,\mathbf{U}_K}$ tends to zero as t approaches infinity. In addition, the eigenvalue $\epsilon_{j,\mathbf{U}_K}$, $j = 1, \ldots, KM$ are the eigenvalues of all $\mathbf{A}_k$, $k = 1. \ldots, K$, i.e., $\epsilon_{i,\mathbf{A}_k}$, $i = 1, \ldots, M$ and $k = 1, \ldots, K$. Hence we have that $\epsilon_{i,\mathbf{A}_k} < 1, \forall i, k$. Without loss of generality, by ordering the eigenvalues of each matrix $\mathbf{A}_k$ in descending order, i.e., $\epsilon_{1,\mathbf{A}_k} \geq \epsilon_{2,\mathbf{A}_k} \geq \cdots \geq \epsilon_{M,\mathbf{A}_k}$, the condition for an epidemic p_k (infection level k) to gradually extinct is then $\epsilon_{1,\mathbf{A}_k} < 1$. Therefore, based on Lemma 1, we obtain

$$\epsilon_{1,\mathbf{A}_k} = 1 - \sum_{l<k}\delta_{kl} + \beta\epsilon_{1,A} < 1 \Leftrightarrow \frac{1}{\epsilon_{1,A}} > \frac{\beta}{\sum_{l<k}\delta_{kl}} = \theta_k.$$

Corollary 1. *Let p_m be an epidemic and p_k be its transmutation, that is, $p_k \succ p_m$. Let the cure rates δ_{kl} be such that either $\delta_{kl} = \delta_l$ or $\delta_{kl} = \delta_{k-l}$. Then, epidemic p_k dies off if p_m dies off.*

Proof of Corollary 1. First consider the case that $\delta_{kl} = \delta_l$. If epidemic p_m dies off, then according to Eq. (25)

$$\frac{1}{\epsilon_{1,A}} > \frac{\beta}{\sum_{l<m}\delta_{ml}} = \frac{\beta}{\sum_{l=0}^{m-1}\delta_l} > \frac{\beta}{\sum_{l=0}^{k-1}\delta_l} = \frac{\beta}{\sum_{l<k}\delta_{kl}}.$$

This means that the largest eigenvalue of the matrix of infection level k, $\mathbf{A}_k$, is less than one and, therefore, epidemic p_k dies off. Consider now the case $\delta_{kl} = \delta_{k-l}$. Then, by a change of variables $v = m - l$ we obtain

$$\frac{1}{\epsilon_{1,A}} > \frac{\beta}{\sum_{l<m}\delta_{ml}} = \frac{\beta}{\sum_{v=1}^{m}\delta_v} > \frac{\beta}{\sum_{v=1}^{k}\delta_v} = \frac{\beta}{\sum_{l<k}\delta_{kl}}. \quad \square$$

Appendix B

B.1

The following expression is used for simplifying the Eq. (16) in Section 4.3. Specifically, if for a node j, which is neighbour of node i ($j \in V_i$), we set $f(x_j(t)) = P\{x_j(t)\} \cdot (1-\beta)^{x_j(t)^\top \cdot \sum_{l>k} p_l}$, $j = 1, \ldots, |V_i|$, then we have that,

$$\begin{aligned}\sum_{\mathbf{x}_{V_i}(t)}\prod_{j\in V_i} f(x_j(t)) &= \sum_{x_1(t)}\cdots\sum_{x_{|V_i|}} f(x_1(t))\cdots f(x_{|V_i|}(t))\\ &= \left(\sum_{x_1(t)} f(x_1(t))\right)\cdots\left(\sum_{x_{|V_i|}(t)} f(x_{|V_i|}(t))\right)\\ &= \prod_{j\in V_i}\sum_{x_j(t)} f(x_j(t)).\end{aligned}$$

B.2

The following expressions give the probabilities $P\{x_i(t) = p_0\}$, $P\{x_i(t) = p_1\}$, $P\{x_i(t) = p_2\}$ and $P\{x_i(t) = p_3\}$ that a node i is susceptible (p_0)or infected with epidemic p_k, $1 \leq k \leq 3$ at time t. Specifically, based on the Eqs. (6)–(13), we first obtain the probability of node i being susceptible at $t+1$:

$$\begin{aligned}P\{x_i(t+1) = p_0\} &= \prod_{j\in V_i}(1 - \beta + \beta P\{x_j(t) = p_0\})P\{x_i(t) = p_0\}\\ &+ \delta_{10}P\{x_i(t) = p_1\} + \delta_{20}P\{x_i(t) = p_2\} + \delta_{30}P\{x_i(t) = p_3\}.\end{aligned}$$

Similarly, we obtain the probability of node i being infected with epidemic p_1

$$\begin{aligned}&P\{x_i(t+1) = p_1\}\\ &= \prod_{j\in V_i}(1 - \beta + \beta(P\{x_j(t) = p_2\} + P\{x_j(t) = p_3\}))\\ &\quad\times P\{x_i(t) = p_0\} - \prod_{j\in V_i}(1 - \beta + \beta P\{x_j(t) = p_0\})P\{x_i(t) = p_0\}\\ &\quad + (1 - \delta_{10})\prod_{j\in V_i}(1 - \beta + \beta(P\{x_j(t) = p_2\} + P\{x_j(t) = p_3\}))\\ &\quad\times P\{x_i(t) = p_1\} + \delta_{21}P\{x_i(t) = p_2\} + \delta_{31}P\{x_i(t) = p_3\}\end{aligned}$$

and, the probability of node i being infected with epidemic p_2

$$
\begin{aligned}
&P\{x_i(t+1)=p_2\}\\
&\quad=\prod_{j\in V_i}(1-\beta P\{x_j(t)=p_3\})P\{x_i(t)=p_0\}\\
&\qquad-\prod_{j\in V_i}(1-\beta(P\{x_j(t)=p_2\}+P\{x_j(t)=p_3\}))\\
&\qquad\times P\{x_i(t)=p_0\}+\prod_{j\in V_i}(1-\beta P\{x_j(t)=p_3\})P\{x_i(t)=p_1\}\\
&\qquad-\prod_{j\in V_i}(1-\beta(P\{x_j(t)=p_2\}+P\{x_j(t)=p_3\}))\\
&\qquad\times(1-\delta_{10})P\{x_i(t)=p_1\}+(1-\delta_{20}-\delta_{21})\\
&\qquad\times\prod_{j\in V_i}(1-\beta P\{x_j(t)=p_3\})P\{x_i(t)=p_2\}+\delta_{32}P\{x_i(t)=p_3\}.
\end{aligned}
$$

Finally, for $P\{x_i(t+1)=p_3\}$ we have that

$$
\begin{aligned}
P\{x_i(t+1)=p_3\}&=\left[1-\prod_{j\in V_i}(1-\beta P\{x_j(t)=p_3\})\right]\\
&\times(P\{x_i(t)=p_0\}+P\{x_i(t)=p_1\}+P\{x_i(t)=p_2\})\\
&+(1-\delta_{32}-\delta_{31}-\delta_{30})P\{x_i(t)=p_3\}.
\end{aligned}
$$

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