This article has been accepted for publication in IEEE Transactions on Network Science and Engineering. This is the author's version which has not been fully edited and content may change prior to final publication. Citation information: DOI 10.1109/TNSE.2021.3125191

Discrete-time non-Markovian SEIS model on complex networks

Igor Tomovski, Lasko Basnarkov, and Alajdin Abazi

***Abstract*—We study a discrete-time variant of the non-Markovian SEIS (Susceptible-Exposed-Infectious-Susceptible) model, occurring on complex networks. The model assumes for an arbitrary form of the Discrete Temporal Probability Functions (DTPFs), that govern the transitions from Exposed to Infectious state (incubation period) and Exposed/Infectious back to Susceptible state (recovery period); this enables the model to address a wide range of real-world spreading phenomena. Theoretical analysis, based on methods from systems theory, leads to an expression that defines the epidemic threshold, for the analyzed model, as a critical relation between the DTPF's, infection rate and the network topology (the largest eigenvalue of the networks adjacency matrix), in a form that extends the result for the Markovian case. Validity of the suggested model, and the obtained theoretical result are confirmed by the numerical analyzes. We argue that the approach used in the paper, may be further extended to describe a wide variety of model variants and sub-models, occurring both on natural, as well as technological (engineering) networks.**

***Index Terms*—Complex networks, Stochastic processes, Discrete-time systems, Nonlinear network analysis, Discrete transforms, Stability analysis.**

I. Introduction

THE stochastic spreading processes, occurring on complex networks, raised serious interest among scientists that work in the field of complex systems, in the past two decades. This interest followed the rapid development of the transportation networks, telecommunication networks, computer networks, social networks etc, accompanied by the emergence of new types of stochastic spreading processes, either in a form of adverse byproducts (computer malware, inappropriate content spreading, etc) or engineered solutions, occurring on these networks. Extensive research in this field resulted in multitude of research papers that treated the issue from phenomenological and theoretical viewpoint, as well as from process control and engineering aspects.

Theoretical work on the subject focused on several research subareas. One of the main challenges was to adequately model the transfer of the spread material from an infectious to a susceptible neighbor in a heterogeneous media. In [1] Moore and Newman considered the site and bond percolation approach. Pastor-Sattoras and Vespignani in [2], [3] analyzed the dynamical evolution of state probabilities of nodes classified by their node degree. A huge leap forward was made with introduction of models that analyzed dynamical evolution of state probabilities of each network node, by considering statistical independence of joint events. Various models based on this approximation were developed both in discrete [4]–[6] and in continuous time [7], [8]. Cator and Van Mieghem in [9] worked towards improving the accuracy of these models, by considering dynamical evolution of the second-order joint events. In another subtopic, spreading models were developed to address processes occurring on special classes of network structures, including multi-layer networks [10] and time-varying networks [11], as well as processes characterized by specific transition dynamics, for example heterogeneous spreading rates [12], or specific form of recovery from failure [13]: recovery occurs τ steps after the failure, if the failure is internally, or τ' steps if the failure is externally driven (caused by a sufficient number of failed neighboring nodes).

In parallel, an extensive research effort was directed towards development of techniques to either control, or to utilize stochastic spreading processes as basis for different engineering solutions [14]–[16]. From protective aspect, different topology-manipulative algorithms and vaccination strategies were considered, for example in [17], [18], as well as algorithms that utilize selective content blocking approach in Social networks [19]. From the engineering viewpoint, stochastic spreading has been analyzed in order to maximize the influence spreading on complex networks [15], to maximize the effects of viral marketing [20], even as a tool to assess network topology [21].

The traditional outlook in the development and analysis of epidemic models, considers spreading phenomena as Markov processes, i.e. processes in which the state of each node, at given instance, is determined only by the state of the system at the moment immediately preceding it. The transition from one status into another, within a single time frame, occurs with a constant, time-independent probability; this notion places classical spreading models into even narrower class of Markov processes – Poisson processes. It is only in the last few years, that this attitude started to change towards the non-Markovian perspective in modeling spreading phenomena. This approach takes into account that, in reality, the status of a node, at given instance, depends on the status of the system at moments beyond the immediate preceding moment and that node status transition occurs following a temporal probability function, referenced by a trigger event (usually exposure to the agent).

I.Tomovski is with the Research Center for Computer Science and Information Technologies, Macedonian Academy of Sciences and Arts, Skopje, Macedonia
E-mail: igor@manu.edu.mk

L. Basnarkov is with the Faculty of Computer Science and Engineering, Ss Cyril and Methodius University and with Research Center for Computer Science and Information Technologies, Macedonian Academy of Sciences and Arts, Skopje, Macedonia

A. Abazi is with the Research Center for Computer Science and Information Technologies, Macedonian Academy of Sciences and Arts, Skopje and with South East European University, Tetovo, Macedonia

This article has been accepted for publication in IEEE Transactions on Network Science and Engineering. This is the author's version which has not been fully edited and content may change prior to final publication. Citation information: DOI 10.1109/TNSE.2021.3125191

The interest in this new approach to the problem, originally emerged in order to address the spreading of new forms of smart computer viruses, as well as information spreading on new types of social platforms. Occurrence of several large-scale epidemics, including the SARS, the MERS, the Avian influenza and finally the COVID-19 pandemics, affirmed the attitude that – proper understanding of real-world spreading processes requires diverting the approach in their analysis from Markovian to the non-Markovian realm. This is due to the fact that almost all status transitions in the epidemic diseases, primarily those related to incubation period and recovery, may by no means be treated as time-invariant Poisson processes, as may be seen from the corresponding collected medical data; for example, in the case of COVID-19 please refer to [22]–[24].

As a result, a number of publications, that suggest different forms of non-Markovian spreading models and analyze their effectiveness and applicability were published in the last decade. In one of the seminal works on the subject, Boguñá et al. [25] suggested Gillespie algorithm as most adequate tool in numerical investigation of non-Markovian spreading processes. Nowzari et al. in [26] introduced the non-Markovian SI*V* model, as a general framework for development of a whole range of epidemic sub-models. In a series of several papers [27]–[29], authors focused on the SIS model with Weilbullian and Gamma [28] distributed infection times and Markovian curing. Using the NIMFA model, adjusted for non-Markovian processes, the authors obtain the threshold relation in the form $\tau_c = 1/\ln(1+\lambda_1)$, with τ_c being the effective infection rate and λ_1 the leading eigenvalue of the networks adjacency matrix [28], [29]. A generalized framework that emulates stationary solution of a non-Markovian SIS model in Markovian realm is introduced in [30]. In [31], authors utilize non-Markovian and fractional calculus approach to study random link activation and deletion with heavy tailed Mittag-Leffler distribution for the inter-event times. Kiss et al. [32] and Sherborne et al. [33] work towards development of pairwise models of non-Markovian spreading, in order to reduce the errors that arise when different forms of mean-field approximation are implemented in modeling of spreading processes occurring on heterogeneous networks. Authors in [34] investigate the circumstances under which an equivalence between Markovian and non-Markovian dynamics exists. A common characteristics of the models inquired in these papers is that they are formulated in continuous time, with status transitions that follow functionally defined probability distributions. In another line of works, the Hawkes process, as a non-Markovian extension of the Poisson process [35], is utilized to study effects that endogenous and exogenous excitement have on epidemic spreading. In Zino et al. [36], authors suggest a mechanism in which self-excitement leads to new link formation in adaptive networks, and derive an epidemic threshold for a SIS type of a process. Kim et al. in [35] and Unwin et al. in [37] used similar methodology, extended with exogenous excitation, for capturing non-Poisson arrival of new cases in population-level models.

In this article, we introduce a novel discrete-time non-Markovian variant of an existing model, that, with adequate adjustments, tends to be "camera-ready" to accept an arbitrary form (either functionally defined, or collected from a process observation) of discrete temporal probability functions that describe the process transitions from Exposed to Infectious and Exposed/Infectious to Susceptible status. In that sense, the model addresses a wide range of spreading phenomena, including spreading of various infectious diseases, spreading of different forms of malware in computer networks, spreading of gossip and ideas in social networks etc. The analysis of the spreading process is based on a strictly dynamical system theory approach. Appropriate tools, including parts of Lyapunov's stability theory and z-transform, are used in order to derive the main result of this work – the epidemic threshold for the analyzed model.

The paper is organized as follows. In the Section 2 we introduce the model, while in the next Section 3 a thorough analysis of the stability of the epidemic origin is presented and the epidemic threshold is determined. Section 4 presents the numerical experiments and their analysis. The paper finishes with the conclusion in Section 5.

II. The Model

In this paper, we present a discrete-time non-Markovian SEIS (Susceptible- Exposed - Infectious- Susceptible) status model of epidemic spreading on complex networks. The SEIS model is well known in its classical (Markovian) form, for example in [38]–[41]. Recently, several research papers, considered the non-Markovian SEIS forms, or non-Markovian SIS models with similar dynamics, in continuous time [27]–[30], [34]. In this work, the concept is extended and further explored for spreading processes that, due to the character of the process dynamics, may be described and/or considered in discrete-time.

The discrete time modeling of stochastic spreading processes is characterized by several differences in comparison to continuous cases. Apparent from the obvious use of difference equations, instead of integro-differential forms, the main distinction lies in the description of the transfer of the spread content from an Infectious neighbor to a Susceptible node. In continuous time models, it is fairly assumed that, within an infinitesimal time frame, this process may only occur alongside a single contact line. In discrete time-models, except in special circumstances, the transfer, within a single time frame, may simultaneously occur from several Infectious neighbors (see Appendix A for details). Next, the tools used in the theoretical analysis, in particular for the non-Markovian models, differ: utilization of z-transform replaces the Laplace transform, adequate formulation and application of system stability criteria etc. In practice, discrete-time models, and especially discrete-time non-Markovian models, address processes in which, due to practical reasons, the data (DTPFs) is collected in distinctive regular time intervals, that might not necessarily represent a proper sampling of the underlying continuous processes. A good example is collection of data related to infectious diseases, where distributions of time to recovery, incubation period, time to hospitalization etc. are collected on per-day bases [23], [24].

This article has been accepted for publication in IEEE Transactions on Network Science and Engineering. This is the author's version which has not been fully edited and content may change prior to final publication. Citation information: DOI 10.1109/TNSE.2021.3125191

Prior to stating the model scenario, in order to stress the differences with the existing SEIS model variants encountered in the literature, as well as to clarify certain issues that might be ambiguous in the following text, a few notions would be addressed in regard to the terminology used in the paper:

- The node would be referred to as Exposed, providing it has contracted and caries the spreading agent, regardless of whether the node has acquired the capacity to spread the agent to the neighboring nodes or not.
- The node would be referred to as Infectious at an instance t, provided it is Exposed and is able to spread the virus further to its neighbors. In this sense, the Infectious status is a sub-state of the Exposed status, i.e. its manifesting state.
- The terms instance, time instance, time step, interval, and time cycle refer to moments in discrete time, in accordance with the time scale of the model, i.e. the models dynamical features. Depending on the rate with which status transitions occur, they may refer to days, hours or any other adequate (in respect to time sampling) time reference.
- *Disease cycle* of a node – T is the maximal time period needed for a node to complete a full cycle starting from Susceptible, through Exposed, possibly Infectious, and back to Susceptible status.
- *Trigger event* is a node-related process event, in respect to which all other subsequent events, within the disease cycle of the node, are time-referenced. In this model, and generally in stochastic spreading processes, the exposure of the Susceptible node to the agent, i.e. the $S \rightarrow E$ transition, plays that role.
- *Discrete temporal probability function* (DTPF) is a mapping $P(\tau)$ that defines probability of the node status, or probability of the node-status transition at time τ following the trigger event. Depending on the type of the process that it describes, the DTPF may be a probability mass function (pmf), cumulative distribution function (cdf), or a random mapping. We generally assume that it is constructed from collected statistical data from the real-world phenomena, or a function that mimic (approximate) such data.

The model presented in this paper is based on the following scenario (detailed flow diagram is presented in Fig. 1): a Susceptible node may become Exposed to the spreading agent, when in contact with an Infectious neighbor. The probability of the exposure at one moment t, providing certain transmission contact (duration and quality of the contact) between nodes occurs, is denoted by β (the infection rate). When exposed, the node, in the general case, does not instantly acquire the capacity to transmit the spread agent to its neighbours (does not become Infectious). Let $b(\tau)$ be a DTPF – the probability that the Exposed node will become Infectious at time τ following the exposure, while $B(\tau)$ a related DTPF – the probability that the Exposed node is Infectious at moment τ from the exposure. We would refer to $b(\tau)$ and $B(\tau)$ as *daily manifesting probability* and *manifesting probability* at time τ, respectively. In respect to manifesting probabilities, in this article we distinguish between two forms of manifestation:

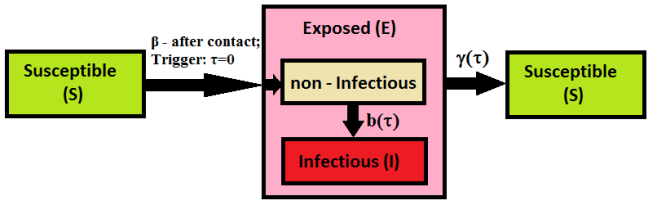


Fig. 1. Flow diagram for the analyzed non-Markovian SEIS model. Susceptible node contracts the spread agent following a contact with Infectious neighbor with probability β and becomes Exposed - trigger. Exposed, but non-Infectious node, becomes Infectious at time τ from the trigger event with probability $b(\tau)$. Exposed node (Infectious or non-Infectious) recovers and becomes Susceptible at time τ from the trigger event with probability $\gamma(\tau)$.

- Cumulative in nature: $B(\tau) = \sum_{k=0}^{\tau} b(k)$, with $\sum_{k=0}^{T-1} b(k) \leq 1$ (the "<" sign indicates that manifestation does not necessarily occur during the disease cycle). This type of manifestation is typical for infectious diseases, i.e. once the node becomes infectious, it remains infectious until recovery. In this case $b(\tau)$ is a pmf.
- Random in nature: $B(\tau) = b(\tau)$, with no restriction imposed on $b(\tau)$, except $0 \leq b(\tau) \leq 1$. This type of manifestation is more characteristic for technological and social networks. The manifesting pattern of exposure to the agent (information, computer virus) differs at each instance.

When the agent is eradicated from the node (recovery), the node transitions back to status Susceptible. The probability that the node will recover from the infection and become Susceptible again, τ time steps after the exposure, is denoted by $\gamma(\tau)$, with $\sum_{\tau=0}^{T-1} \gamma(\tau) = 1$ (the DTPF $\gamma(\tau)$ is pmf). For the parameters $\gamma(\tau)$ the term *daily recovering probabilities* will be used in the further text. Similarly, we consider the parameter $\Gamma(\tau) = \sum_{k=0}^{\tau} \gamma(k)$, and refer to it as *cumulative recovery probability* at time τ. Its complement $\overline{\Gamma(\tau)} = 1 - \Gamma(\tau)$ would be extensively used both in the model definition, as well as in the analysis. Finally, we assume that once a Susceptible node is exposed to the agent, it may not be re-exposed until it recovers.

A. Mathematical model

Consider a graph $G = G(V, E, w)$, with V representing the set of vertices (nodes), such that $|V| = N$, E the set of edges, $|E| = L$, and $w : E \rightarrow [0, 1]$ a mapping function, that associates a weight to each directed edge [42]. The interconnection among nodes in the network, is described with the adjacency matrix $\mathbf{A} = [a_{ij}]$, such that $0 \leq a_{ij} \leq 1$ if a link from the node j towards node i exists (direction of influence), and $a_{ij} = 0$ otherwise. The network (the graph) is, in the general case, asymmetrical and is assumed to be strongly connected.

The network G, as defined here, models the epidemiological contacts between individuals involved in the spreading process. Associated weights give assessment of these contacts

This article has been accepted for publication in IEEE Transactions on Network Science and Engineering. This is the author's version which has not been fully edited and content may change prior to final publication. Citation information: DOI 10.1109/TNSE.2021.3125191

in relation to their epidemiological significance like time averages of duration of particular contact, proximity of individuals, use of protective equipment, use of anti-virus programs etc.

One important issue that has to be addressed with caution, prior to formally stating the mathematical model, is the problem of the initial conditions. Consider p_i^{In} to be a probability that node i comes in contact with an external Infectious vector (wild animal(s), contaminated food or water, malware or malicious software source, information source etc) at time $t = 0$. The role of the external contacts, besides modeling the origin of the process, is to provide an initial process trigger: this could not be achieved by simply assigning Exposure/Infectiousness probabilities to nodes at $t = 0$, as done in the classical case. One should note that, in reality, subsequent exposures that originate from external sources, might occur for $t > 0$, as well. However, this seriously increases the complexity of both modeling and the analysis, to an extent that, by far, exceeds the scope of this paper, and would, therefore, be further omitted.

Let $p_i^S(t)$, $p_i^E(t)$, $p_i^I(t) \in [0,1]$ represent the probabilities that node i is Susceptible, Exposed and/or Infectious at time t, with $p_i^S(t) + p_i^E(t) = 1$ defining the probability conservation condition. In order to formulate the dynamical behavior of status probabilities for each node, as a function of time, the assumption of statistical independence of joint node-status events will be utilized, i.e. $p(s_i^S(t), s_j^I(t)) = p_i^S(t)p_j^I(t)$, with $s_i^S(t)$, $s_i^I(t)$ being random variables that represent the status of the node i as Susceptible and Infectious, at time t, respectively. Though this approximation introduces certain degree of error, its use is considered a golden standard in modeling spreading processes occurring in complex networks and is extensively applied in different studies [4], [5], [8], [43], [44]. By employing this approximation, and acting similarly as in [4], [5], the probability that the Susceptible node i, would be infected by its neighbors, at time t, is defined with the expression:

$$\pi_i(t) = 1 - \left(1 - p_i^{In}\delta(t)\right) \prod_{j=1}^{N} \left(1 - \beta a_{ij} p_j^I(t)\right),$$

where $\delta(t)$ is the the Kronecker's function, that is used for setting the initial seed of infection, while the product accounts for the probabilities of missing the infection at given moment while in contact with infectious neighbor. On the basis of the previous discussion, and considering that $\overline{\Gamma(T-1)} = 0$, the analyzed system may be described with the following set of equations:

$$\begin{aligned} p_i^S(t+1) &= 1 - p_i^E(t+1) \\ p_i^E(t+1) &= \sum_{\tau=0}^{T-2} \left(1 - p_i^E(t-\tau)\right) \pi_i(t-\tau)\overline{\Gamma(\tau)} \qquad (1) \\ p_i^I(t+1) &= \sum_{\tau=0}^{T-2} \left(1 - p_i^E(t-\tau)\right) \pi_i(t-\tau)\overline{\Gamma(\tau)}B(\tau). \end{aligned}$$

Notice that the second and the third equation of the dynamical system (1) fully describe the dynamical behavior of the model, with the first equation stating the conservation of probability constraint. For that reason, the first equation, unless otherwise stated, would be omitted when referring to the system (1) in the further text.

III. Theoretical analysis

The most important theoretical issue in the analysis of the spreading processes, is the derivation of the epidemic threshold. For re-occurring processes as the SEIS model, it is a relation between system parameters, characteristics of the network, status changing probabilities etc, that determines whether an initial spreading out-brake would turn into persistent epidemic, or will die out as time lapses.

In order to obtain the epidemic threshold for the analyzed SEIS model, in this Section a thorough stability analysis of the dynamical system (1) is conducted. Investigation of the conditions under which the origin of the system, i.e. $p_i^E(t) = p_i^I(t) = 0$, $p_i^S(t) = 1$, for all i, is globally stable, gives rise to critical relations among process parameters, that determine whether an initial infection will vanish or infinitely persist.

Remark 1. *Stability analysis of a dynamical system investigates the long-term behavior of the system, i.e. the system asymptotic dynamics when $t \to \infty$. From relation (1), one may conclude that the initial conditions terms $\left(1 - p_i^{In}(t)\delta(t)\right)$ directly contribute to the system dynamics only within the first $T-1$ time steps, and have no direct effect on the asymptotic behavior. In that sense, in the forthcoming analysis, it would be assumed that dynamical behavior is analyzed starting from some moment $t \geq T$, omitting the $\left(1 - p_i^{In}(t)\delta(t)\right)$ terms; however, values generated by the first $T-1$ time steps, would be included in the analysis as initial conditions.*

The conditions when the spreading cannot rise to an epidemic in the mathematical models are related to the stability of the epidemic origin, or the disease-free state. We address this issue by studying its linear and nonlinear stability in the following two theorems.

Theorem 1. Local asymptotic stability of the origin

Consider the dynamical system (1), and let $\mathbf{P}^E(t) = [p_i^E(t)]_{N\times 1}$ and $\mathbf{P}^I(t) = [p_i^I(t)]_{N\times 1}$, for $i = \overline{1,N}$. If the graph G is strongly connected, the vector $[\mathbf{P}^E(t)^{\mathrm{T}}\mathbf{P}^I(t)^{\mathrm{T}}]^{\mathrm{T}} = [\mathbf{0}_{N\times 1}^{\mathrm{T}}\mathbf{0}_{N\times 1}^{\mathrm{T}}]^{\mathrm{T}}$, i.e. the epidemic origin, is a locally stable fixed point of the dynamical system (1), provided all roots $z = r_{i,j}$, $i = 1,..,N$, $j = 0,..,T-2$, of the polynomial set:

$$P_i(z) \quad = \quad z^{T-1} - \beta\lambda_i(\mathbf{A}) \sum_{\tau=0}^{T-2} \overline{\Gamma(\tau)}B(\tau)z^{T-2-\tau} = 0, \qquad (2)$$

where $\lambda_i(\mathbf{A})$, $i = 1,..,N$ are the eigenvalues of the adjacency matrix $\mathbf{A}$, lie within the unit circle, i.e. $|r_{i,j}| < 1$.

Proof: From the system of equations (1), it is straightforward obvious that, by taking $\mathbf{P}^E(t) = \mathbf{0}$ and $\mathbf{P}^I(t) = \mathbf{0}$, for $t < T$, providing no external trigger exists ($p^{In} = 0$), one obtains $\mathbf{P}^E(T) = \mathbf{0}$ and $\mathbf{P}^I(T) = \mathbf{0}$. Similarly, for any $t > T$, by taking $\mathbf{P}^E(t-\tau) = \mathbf{0}$ and $\mathbf{P}^I(t-\tau) = \mathbf{0}$, $\tau = 0,..,T-1$, one obtains $\mathbf{P}^E(t+1) = \mathbf{0}$ and $\mathbf{P}^I(t+1) = \mathbf{0}$. Consequently, the vector $[\mathbf{P}^E(t)^{\mathrm{T}}\ \mathbf{P}^I(t)^{\mathrm{T}}]^{\mathrm{T}} = [\mathbf{0}_{N\times 1}^{\mathrm{T}}\ \mathbf{0}_{N\times 1}^{\mathrm{T}}]^{\mathrm{T}}$ is a fixed point of the dynamical system (1).

In order to prove the dynamical stability of the fixed point, we will consider the evolution of the perturbations $\delta p_i^E(t)$ and $\delta p_i^I(t)$ in the vicinity of the origin. Acting similarly as in [5, Lemma 1], by linearizing the equations (1) in the neighborhood of the point $[\mathbf{0}_{N\times 1}^{\mathrm{T}}\ \mathbf{0}_{N\times 1}^{\mathrm{T}}]^{\mathrm{T}}$, for $t > T$, one obtains:

$$\begin{aligned}\delta p_i^E(t+1) &= \sum_{\tau=0}^{T-2}\sum_{j=1}^{N}\beta a_{ij}\overline{\Gamma(\tau)}\delta p_j^I(t-\tau) \\ \delta p_i^I(t+1) &= \sum_{\tau=0}^{T-2}\sum_{j=1}^{N}\beta a_{ij}\overline{\Gamma(\tau)}B(\tau)\delta p_j^I(t-\tau).\end{aligned} \tag{3}$$

For the dynamical behavior of the perturbed system, for $t \leq T-1$, please refer to the Appendix B.

From the system of equations (3), it is apparent that the second group of N equations, that describe the dynamical evolution of the Infectious status of each node in the vicinity of the epidemic origin, alone, is self sufficient, and the dynamical stability of the system as a whole relies on the stability of this equation sub-set, only; vanishing of the infectious probabilities implies vanishing of the exposed ones, as well.

Let $\delta\mathbf{P}^I(t) = [\delta p_i^I(t)]^{\mathrm{T}}$ be the perturbed infection probability vector. By time-shifting the system $T-1$ steps forward, in accordance with the Remark 1, the second subset of system of equations (3) can now be re-written in a vector form as follows:

$$\delta\mathbf{P}^I(t+T) = \beta\sum_{\tau=0}^{T-2}\overline{\Gamma(\tau)}B(\tau)\mathbf{A}\delta\mathbf{P}^I(t+T-1-\tau). \tag{4}$$

The dynamical system described with the vector equation (4) is a discrete linear system of order $N\times(T-1)$, represented by N linear equations with latency $T-1$.

By performing unilateral z-transform on both sides of the equation (4) one obtains:

$$\begin{aligned}\mathbf{Q}(z)z^T - \sum_{\tau=0}^{T-1}\delta\mathbf{P}^I(\tau)z^{T-\tau} = \beta\mathbf{A}\sum_{\tau=0}^{T-2}\overline{\Gamma(\tau)}B(\tau)\mathbf{Q}(z)z^{T-1-\tau} \\ -\beta\mathbf{A}\sum_{\tau=0}^{T-2}\overline{\Gamma(\tau)}B(\tau)\sum_{k=0}^{T-2-\tau}\delta\mathbf{P}^I(k)z^{T-1-\tau-k},\end{aligned} \tag{5}$$

where $\mathbf{Q}(z) = \sum_{t=0}^{\infty} z^{-t}\delta\mathbf{P}^I(t)$ is the z-transform of the perturbed infection probability vector $\delta\mathbf{P}^I(t)$. The last relationship can be rearranged as:

$$\left(z^T - \beta\mathbf{A}\sum_{\tau=0}^{T-2}\overline{\Gamma(\tau)}B(\tau)z^{T-1-\tau}\right)\mathbf{Q}(z) = \mathbf{G}(z),$$

where for shortening the notation we have introduced the vector function that encapsulates the initial perturbations:

$$\begin{aligned}\mathbf{G}(z) &= \sum_{\tau=0}^{T-1}\delta\mathbf{P}^I(\tau)z^{T-\tau} \\ &- \beta\mathbf{A}\sum_{\tau=0}^{T-2}\overline{\Gamma(\tau)}B(\tau)\sum_{k=0}^{T-2-\tau}\delta\mathbf{P}^I(k)z^{T-1-\tau-k}.\end{aligned}$$

Now, $\mathbf{Q}(\mathbf{z})$ can be expressed as:

$$\begin{aligned}\mathbf{Q}(\mathbf{z}) &= \left(z^T\mathbf{I} - \beta\mathbf{A}\sum_{\tau=0}^{T-2}\overline{\Gamma(\tau)}B(\tau)z^{T-1-\tau}\right)^{-1}\mathbf{G}(z) \\ &= \frac{\mathbf{A}'(z)\mathbf{G}(z)}{\det\left(z^T\mathbf{I} - \beta\mathbf{A}\sum_{\tau=0}^{T-2}\overline{\Gamma(\tau)}B(\tau)z^{T-1-\tau}\right)},\end{aligned}$$

where $\mathbf{A}'(z)$ is a $N\times N$ matrix consisting of minors of the matrix $z^T\mathbf{I} - \beta\mathbf{A}\sum_{\tau=0}^{T-2}\overline{\Gamma(\tau)}B(\tau)z^{T-1-\tau}$, and which elements are polynomials of order $(N-1)\times T$. After dividing both numerator and denominator term with z^N, and by employing $f(z) = \beta\sum_{\tau=0}^{T-2}\overline{\Gamma(\tau)}B(\tau)z^{T-2-\tau}$, the determinant in the the denominator may be represented as:

$$\det(z^{T-1}\mathbf{I} - \mathbf{A}f(z)) = [f(z)]^N\det\left(\frac{z^{T-1}}{f(z)}\mathbf{I} - \mathbf{A}\right).$$

Considering that the characteristic polynomial of the matrix $\mathbf{A}$ is defined as $\det(\lambda\mathbf{I} - \mathbf{A}) = \prod_i(\lambda - \lambda_i(\mathbf{A}))$, with $\lambda_i(\mathbf{A})$ being the eigenvalues of the matrix$\mathbf{A}$, the following expression holds:

$$\begin{aligned}\det(z^{T-1}\mathbf{I} - \mathbf{A}f(z)) &= [f(z)]^N\prod_i\left(\frac{z^{T-1}}{f(z)} - \lambda_i(\mathbf{A})\right) \\ &= \prod_i\left(z^{T-1} - \lambda_i(\mathbf{A})f(z)\right).\end{aligned}$$

Consequently:

$$\mathbf{Q}(\mathbf{z}) = \frac{\mathbf{A}'(z)\mathbf{G}(z)}{\prod_{i=0}^{T-2}\left(z^{T-1} - \lambda_i(\mathbf{A})f(z)\right)} = \frac{\mathbf{H}(z)}{P(z)}, \tag{6}$$

where $\mathbf{H}(z)$ is an $N\times 1$ vector, which elements are polynomials of order $N\times(T-1)$ and $P(z) = \prod_{i=1}^{N}P_i(z)$ is polynomial of order $N\times(T-1)$ that is product of the polynomials:

$$P_i(z) = z^{T-1} - \lambda_i(\mathbf{A})\beta\sum_{\tau=0}^{T-2}\overline{\Gamma(\tau)}B(\tau)z^{T-2-\tau}.$$

From the equation (6), one may conclude that the stability of the dynamical system (1), in the vicinity of the epidemic origin, is determined by the roots of the polynomials $P_i(z)$, i.e. if all roots of the polynomials $P_i(z)$, $r_{i,j}$, lie within the unit circle, $|r_{i,j}| < 1$, than the dynamical system is stable in the point of epidemic origin. ■

Theorem 2. Global asymptotic stability of the origin

Consider the dynamical system (1), and let $\mathbf{P}^E(t) = [p_i^E(t)]$ *and* $\mathbf{P}^I(t) = [p_i^I(t)]$, *for* $i = 1,..,N$. *Providing all assumptions of Theorem 1 hold, the point of the epidemic origin is a global, on* $[0,1]^{2N}$, *asymptotically stable fixed point of the dynamical system (1).*

Proof: From the relation (1), considering that $p_i^E(t) \geq 0$, and by using the Weierstrass product inequality $\prod_i(1-\alpha_i) \leq 1 - \sum_i\alpha_i$, that holds for real numbers $0 \leq \alpha_i \leq 1$, one can obtain the following bounds:

$$\begin{aligned}p_i^E(t+1) &\leq \sum_{\tau=0}^{T-2}\sum_{j=1}^{N}\beta a_{ij}p_j^I(t-\tau)\overline{\Gamma(\tau)} \\ p_i^I(t+1) &\leq \sum_{\tau=0}^{T-2}\sum_{j=1}^{N}\beta a_{ij}p_j^I(t-\tau)\overline{\Gamma(\tau)}B(\tau).\end{aligned} \tag{7}$$

This article has been accepted for publication in IEEE Transactions on Network Science and Engineering. This is the author's version which has not been fully edited and content may change prior to final publication. Citation information: DOI 10.1109/TNSE.2021.3125191

Consider the following (auxiliary) dynamical system:

$$\begin{aligned} p_i'^E(t+1) &= \sum_{\tau=0}^{T-2}\sum_{j=1}^{N}\beta a_{ij}p_j'^I(t-\tau)\overline{\Gamma(\tau)} \\ p_i'^I(t+1) &= \sum_{\tau=0}^{T-2}\sum_{j=1}^{N}\beta a_{ij}p_j'^I(t-\tau)\overline{\Gamma(\tau)}B(\tau), \end{aligned} \tag{8}$$

or in a vector form:

$$\begin{aligned} \mathbf{P}'^E(t+1) &= \sum_{\tau=0}^{T-2}\beta\mathbf{A}\overline{\Gamma(\tau)}\mathbf{P}'^I(t-\tau) = \\ &= \mathbf{f}_1\left(\left(\mathbf{P}'^I(t-\tau)\right)_{\tau=0}^{T-2}\right) \\ \mathbf{P}'^I(t+1) &= \sum_{\tau=0}^{T-2}\beta\mathbf{A}\overline{\Gamma(\tau)}B(\tau)\mathbf{P}'^I(t-\tau) = \\ &= \mathbf{f}_2\left(\left(\mathbf{P}'^I(t-\tau)\right)_{\tau=0}^{T-2}\right), \end{aligned} \tag{9}$$

where we have denoted the sums as functions and $\left(\mathbf{P}'^I(t-\tau)\right)_{\tau=0}^{T-2} = \{\mathbf{P}'^I(t), \mathbf{P}'^I(t-1), \dots \mathbf{P}'^I(t-T+2)\}$ is a shorthand notation for a sequence of successive vectors $\mathbf{P}'^I(t)$. The system of equations (1) can be re-written in a vector form as:

$$\begin{aligned} \mathbf{P}^E(t+1) &= \mathbf{g}_1\left(\left(\mathbf{P}^E(t-\tau)\right)_{\tau=0}^{T-2}, \left(\mathbf{P}^I(t-\tau)\right)_{\tau=0}^{T-2}\right) \\ \mathbf{P}^I(t+1) &= \mathbf{g}_2\left(\left(\mathbf{P}^E(t-\tau)\right)_{\tau=0}^{T-2}, \left(\mathbf{P}^I(t-\tau)\right)_{\tau=0}^{T-2}\right) \end{aligned} \tag{10}$$

Likewise, the bounds (7) can be succinctly written with vectors as:

$$\begin{aligned} \mathbf{P}^E(t+1) &\leq \mathbf{f}_1\left(\left(\mathbf{P}^I(t-\tau)\right)_{\tau=0}^{T-2}\right) \\ \mathbf{P}^I(t+1) &\leq \mathbf{f}_2\left(\left(\mathbf{P}^I(t-\tau)\right)_{\tau=0}^{T-2}\right). \end{aligned} \tag{11}$$

One should observe first that the Lemma 1, and consequently the proposition (24) of Lemma 2, given in the Appendix C holds for the functions $\mathbf{f}_1$ and $\mathbf{f}_2$. Furthermore, for $t \geq T$, for any given sequences $\left(\mathbf{P}^I(t-\tau)\right)_{\tau=0}^{T-2}$ and $\left(\mathbf{P}^E(t-\tau)\right)_{\tau=0}^{T-2}$ from (10) and (11) one has $\mathbf{g}_2\left(\left(\mathbf{P}^E(t-\tau)\right)_{\tau=0}^{T-2}, \left(\mathbf{P}^I(t-\tau)\right)_{\tau=0}^{T-2}\right) \leq \mathbf{f}_2\left(\left(\mathbf{P}^I(t-\tau)\right)_{\tau=0}^{T-2}\right)$. Then, in accordance with Lemma 2, for any set of initial conditions $\left(\mathbf{P}'^I(\tau)\right)_{\tau=0}^{T-2} = \left(\mathbf{P}^I(\tau)\right)_{\tau=0}^{T-2}$, with corresponding $\left(\mathbf{P}^E(\tau)\right)_{\tau=0}^{T-2}$, the following relation holds:

$$0 \leq \mathbf{P}^I(t) \leq \mathbf{P}'^I(t). \tag{12}$$

Observe that the governing dynamics of the vector $\mathbf{P}'^I(t)$ in (9) is of identical form as the vector $\mathbf{P}^I(t)$ in (4), when the time-shift is disregarded. Using similar arguments as those following eq. (4) in the proof of Theorem 1, one may obtain that, providing all assumptions in the statement of the Theorem 2 hold, $\mathbf{P}'^I(t) \to 0$, when $t \to 0$. Consequently, from (12):

$$0 \leq \lim_{t\to\infty}\mathbf{P}^I(t) \leq \lim_{t\to\infty}\mathbf{P}'^I(t) = 0, \; i.e. \lim_{t\to\infty}p_i^I(t) = 0,$$

yielding, from (1) $\lim_{t\to\infty}p_i^E(t) = 0$, as well. The proof is completed. ■

The relationship between the model parameters which determine the epidemic threshold is given in the following theorem.

Theorem 3. *The dynamical system (1) is asymptotically stable, providing the graph G is strongly connected and the following condition holds:*

$$\beta\lambda_1(\mathbf{A}) < \frac{1}{\sum_{\tau=0}^{T-2}\overline{\Gamma(\tau)}B(\tau)}. \tag{13}$$

Proof: If G is strongly connected, the matrix $\mathbf{A}$ is non-negative and irreducible. From the Theorem of Perron - Frobenius for non-negative irreducible matrices, [45], [46], matrix $\mathbf{A}$ has a distinctive and positive eigenvalue $\lambda_1(\mathbf{A})$, such that all other eigenvalues of $\mathbf{A}$, satisfy $|\lambda_i(\mathbf{A})| \leq \lambda_1(\mathbf{A})$. We will consider separately the polynomial $P_1(z)$ associated with the leading eigenvalue $\lambda_1(\mathbf{A})$ of the adjacency matrix $\mathbf{A}$ and those corresponding to the remaining ones.

Case 1: Consider the polynomial $P_1(z)$, as defined with the relation (2), for $\lambda_i(\mathbf{A}) = \lambda_1(\mathbf{A})$ - the largest eigenvalue of $\mathbf{A}$. Since $P_1(z)$ is monic, there exists a Frobenius companion matrix $\mathbf{F}_1$:

$$\mathbf{F}_1 = \begin{pmatrix} 0 & 0 & \cdots & 0 & \beta\lambda_1(\mathbf{A})\overline{\Gamma(T-2)}B(T-2) \\ 1 & 0 & \cdots & 0 & \beta\lambda_1(\mathbf{A})\overline{\Gamma(T-3)}B(T-3) \\ \vdots & \vdots & \ddots & \vdots & \\ 0 & 0 & \cdots & 1 & \beta\lambda_1(\mathbf{A})\overline{\Gamma(0)}B(0) \end{pmatrix},$$

such that the eigenvalues of the matrix $\mathbf{F}_1$ are roots of $P_1(z)$, $\lambda_j(\mathbf{F}_1) = r_{1,j}$ [47]–[49].

Since $\lambda_1(\mathbf{A}) > 0$, $\mathbf{F}_1$ is non-negative. Let $0 \leq s \leq T-2$ be a positive number, such that $\overline{\Gamma(s)}B(s) > 0$ and for any $k, s < k \leq T-2$, $\overline{\Gamma(k)}B(k) = 0$. Then, the polynomial $P_1(z)$ has $T-2-s$ roots $r_{1,j} = 0$, $j = s+1, .., T-2$. The residual polynomial of $P_1(z)$, $P_1'(z)$:

$$P_1'(z) = z^{s+1} - \beta\lambda_1(\mathbf{A})\sum_{\tau=0}^{s}\overline{\Gamma(\tau)}B(\tau)z^{s-\tau} = 0,$$

is characterized by another Frobenius companion matrix $\mathbf{F}_1'$ of order $(s+1)\times(s+1)$, that is non-negative and irreducible. From the Theorem of Perron - Frobenius for non-negative irreducible matrices [45], [46], the matrix $\mathbf{F}_1'$ (and the polynomial $P_1'(z)$) is characterized by one distinctive real and positive eigenvalue (root) $\lambda_1(\mathbf{F}_1') = r_{1,1}$, such that all other eigenvalues (roots of the polynomial), satisfy: $|\lambda_j(\mathbf{F}_1')| = |r_{1,j}| \leq r_{1,1} = \lambda_1(\mathbf{F}_1')$.

To summarize, providing at least one $\overline{\Gamma(\tau)}B(\tau) \neq 0$ exists (otherwise the model is non-existent), the polynomial $P_1(z)$ has one distinctive real and positive root $r_{1,1}$. By taking $z = 1$ in $P_1(z)$, one obtains the following relationship:

$$\beta\lambda_1(\mathbf{A}) = \frac{1}{\sum_{\tau=0}^{T-2}\overline{\Gamma(\tau)}B(\tau)} = \delta_{eff}.$$

If:

$$\beta\lambda_1(\mathbf{A}) < \delta_{eff}, \tag{14}$$

from the Wielandt's Theorem [50], [51] follows that, the largest eignevalue of the matrix $\mathbf{F}_1$ (root of the polynomial $P_1(z)$) satisfies: $\lambda_1(\mathbf{F}_1) = r_{1,1} < 1$.

This article has been accepted for publication in IEEE Transactions on Network Science and Engineering. This is the author's version which has not been fully edited and content may change prior to final publication. Citation information: DOI 10.1109/TNSE.2021.3125191

Case 2: Let us now consider the remaining polynomials $P_i(z)$, where $\lambda_i(\mathbf{A}) \in \mathbb{C}$ and $\lambda_i(\mathbf{A}) \neq \lambda_1(\mathbf{A})$.

If $\lambda_i(\mathbf{A}) = 0$, then all roots of $P_i(z)$, $r_{i,j} = 0$. If $\lambda_i(\mathbf{A}) \neq 0$, to each polynomial $P_i(z)$, a Frobenius companion matrix $\mathbf{F}_i = [f^i_{kl}]$ might be assigned, such that $f^i_{k,T-2} = (\lambda_i(\mathbf{A})/\lambda_1(\mathbf{A}))\, f^1_{k,T-2}$, and $f^i_{k,l} = f^1_{k,l}$, $l = 0, .., T-3$.

Following the same arguments, as in Case 1, a residual polynomial $P'_i(z)$ exists and is accompanied by a Frobenius companion matrix $\mathbf{F}'_i = [f'^i_{k,l}]$ of order $(s+1) \times (s+1)$. Since $|f'^i_{k,s}| = |(\lambda_i(\mathbf{A})/\lambda_1(\mathbf{A}))| f'^1_{k,s} \leq f'^1_{k,s}$, with all other $f'^i_{k,l} = f'^1_{k,l}$, from the Wielandt's theorem [50], all eigenvalues of $\mathbf{F}'_i$ (roots of $P'_i(z)$), $\lambda_j(\mathbf{F}'_i) = r_{i,j}$, satisfy: $|\lambda_j(\mathbf{F}'_i)| = |r_{i,j}| \leq r_{1,1} = \lambda_1(\mathbf{A})$. Providing relation (14) holds, all roots $|r_{i,j}| < 1$.

From the discussion, and in accordance with Theorem 1 and Theorem 2, providing relation (14) holds, the epidemic origin is both locally and globally stable fixed point of the dynamical system (1). The proof is completed. ∎

From the last theorem one can conclude that the epidemic threshold is obtained from the following relationship:

$$\beta\lambda_1(\mathbf{A}) = \frac{1}{\sum_{\tau=0}^{T-2} \overline{\Gamma(\tau)} B(\tau)}, \tag{15}$$

which extends the known result for Markovian model $\beta\lambda_1(\mathbf{A}) = \delta$.

IV. Numerical simulations and analyzes

In order to test the validity of the model and the result for the epidemic threshold, in this section we present part of the results obtained from the numerical simulations, conducted on a number of synthetic (computer generated) networks. Two types of simulations were performed: simulation of the probabilistic dynamical system, i.e. the set of equations (1), and stochastic simulations. Stochastic simulations are conducted using the literal model narrative: once a susceptible node is exposed to the virus, a timer is started ($\tau = 0$) and a moment of recovery τ_{rec} is selected by a random generator with cumulative distribution $\Gamma(\tau)$. In cases in which a cumulative-like behavior of the manifesting state is considered, a moment of manifestation, τ_{inf}, is selected by a random generator with a cumulative probability $B(\tau)$; otherwise, at each instance in time, between the time of the exposure and the moment of recovery, the Exposed node may become Infectious, depending on the outcome generated by a Bernoulli random generator, with probability $B(\tau)$. For each of the investigated scenarios, simulations are repeated, under similar circumstances, in order to obtain 100 stochastic time series, that are subsequently averaged.

In the analysis presented bellow, results from the simulations conducted on two of the utilized networks are presented:

- Barabási–Albert [52] directed and weighted graph with $N = 1000$ nodes, total of $L = 3992$ uni-directional links , and the largest eigenvalue of the graph's adjacency matrix $\lambda_1(\mathbf{A}) = 5.2922$. The reference BA(1000,3992, rand(0:1)) or BA1000 for short, will be used for this network thought the paper. The network is derived from a symmetrical BA(1000,1996) graph, with $N = 1000$ nodes, generated with parameters $m_0 = 3$, $m = 2$;

TABLE I
DTPFs of the four cases stat 1, stat 2, stat 3 and stat 4

	Stat 1		Stat 2		Stat 3		Stat 4	
τ	$B(\tau)$	$\gamma(\tau)$	$B(\tau)$	$\gamma(\tau)$	$B(\tau)$	$\gamma(\tau)$	$B(\tau)$	$\gamma(\tau)$
0	0	0	0	0	0	0	0	0
1	0	0	0	0	0	0	1	0.022
2	0.10	0	0.10	0	0.10	0	1	0.043
3	0.25	0	0.25	0	0.25	0.1	1	0.062
4	0.50	0	0.50	0	0.50	0.15	0	0.078
5	0.75	0	0.75	0.1	0.75	0.25	0	0.091
6	0.90	0	0.90	0.15	0.90	0.40	0	0.100
7	1	0	1	0.25	1	0.10	1	0.104
8	1	0.10	1	0.40			1	0.104
9	1	0.15	1	0.10			1	0.100
10	1	0.25					0	0.091
11	1	0.40					0	0.078
12	1	0.10					0	0.062
13							1	0.043
14							1	0.022

- Watts–Strogatz [53] directed and weighted graph with $N = 1000$ nodes, total of $L = 6000$ uni-directional links , and the largest eigenvalue of the graphs adjacency matrix $\lambda_1(\mathbf{A}) = 3.26997$. This network would be further referenced as WS(1000,6000, rand(0:1)), or WS1000 for short. The network is derived from a symmetrical WS(1000,3000) graph, with $N = 1000$ nodes, generated with parameters $r = 3$, $p = 0.2$;

In order to test the functionality of the model, validity of the threshold and to derive additional conclusions that would become apparent from the simulations, four different DTPFs were used. To the first three DTPFs (stat 1, stat 2 and stat 3) we would refer to as *quasi-medical* DTPFs (qm for short). The qm DTPFs are constructed by arbitrary choice of parameters $\gamma(\tau)$ ($\Gamma(\tau)$) and $B(\tau)$, in a fashion to resemble statistical data related to epidemic diseases, i.e. $B(\tau)$ has a character of a cumulative probability and $b(\tau)$ of a density function (for reference consider Fig. 3 in [22]). The fourth DTPF (stat 4) has a non-cumulative character of $B(\tau)$, and is more characteristic for spreading processes occurring on technological and social networks. The four DTPF's are presented in Table I.

One may notice that the qm DTPFs, labeled as stat 1, stat 2 and stat 3, are almost identical, except for the time shift in the daily recovering probabilities. The aim of such construct is to clarify to what extent the overlapping between the manifestation and the recovery period affects the epidemic threshold. Although this notion is somewhat apparent from the expression (15), considering the cumulative nature of $B(\tau)$ in these sets, we intended to test it in practice.

The stat 4 DTPF is random in nature. Infectious status periods are interrupted by periods of dormicity. Though this behavior is seen in some viruses, HPV for example, it is more common for the computer viruses [27]–[29]. Recovering probabilities are generated by the function $\gamma(\tau) = \pi/(2T)\sin(\tau\pi/T)$.

Critical values for the parameter $\beta = \beta_c$, that define the epidemic threshold for stat 1, stat 2, stat 3 and stat 4 DTPFs and the utilized graphs, are given in Table II. The results from the numerical analysis on both BA1000 and WS1000 networks

This article has been accepted for publication in IEEE Transactions on Network Science and Engineering. This is the author's version which has not been fully edited and content may change prior to final publication. Citation information: DOI 10.1109/TNSE.2021.3125191

TABLE II
CRITICAL VALUES OF β_c

	β_c for BA1000	β_c for WS1000
stat 1	0.0328621	0.0531849
stat 2	0.0674848	0.109219
stat 3	0.162195	0.262501
stat 4	0.0472393	0.0764533

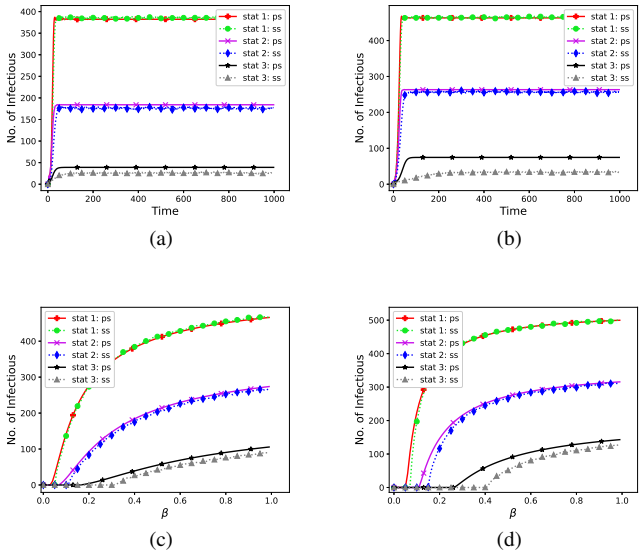


Fig. 2. Numerical simulations conducted on the BA1000 and WS1000 networks, and the qm DTPFs. Top row: Time evolution for the SEIS process occurring on the BA1000 graph, for $\beta = 0.4$ (a) and WS1000 graph, for $\beta = 0.45$ (b). ps-probabilistic system, ss - stochastic simulation. Bottom row: Number of Infectious nodes as function of β for the BA1000 (c) and WS1000 graph (d), obtained by sweeping the dynamical behavior of the model in the range $\beta \in [0, 1]$.

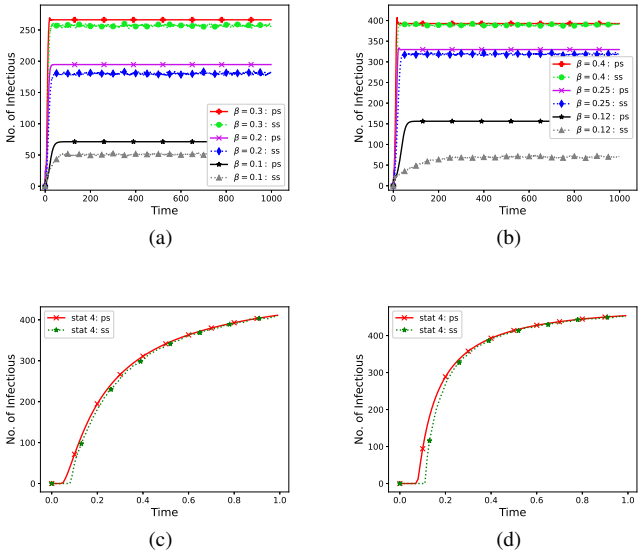


Fig. 3. Top row: Time evolution for the SEIS process occurring on the BA1000 graph (a) and WS1000 graph (b), for different values of the parameter β and the stat 4 DTPF. ps-probabilistic system, ss - stochastic simulation (averaged). Bottom row: Number of Infectious nodes as function of β for the BA1000 (c) and WS1000 graph (d), obtained by sweeping the dynamical behavior of the model in the range $\beta \in [0, 1]$.

for the qm DTPFs are presented in Fig. 2. Corresponding analysis for the stat 4 DTPFs is presented in Fig. 3.

From the results presented in the Fig. 2 and Fig. 3 and in Table II, the following conclusions may be drawn:

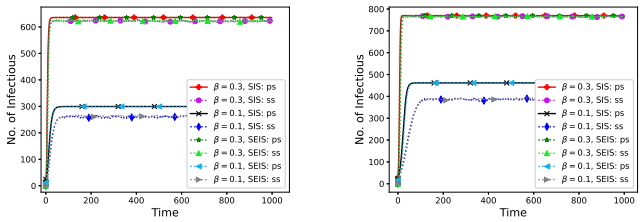
- There is a good overlap between the results obtained from the stochastic and probabilistic simulations, that confirms the validity of the SEIS model as defined with the system of equations (1). Differences might be noticed in low-level epidemics and are especially pronounced for the WS1000 graph (Fig. 2(b) for stat 3, Fig. 3(b) for $\beta = 0.12$). These type of discrepancies are expected when the system is near the threshold and acts in a non-stationary fashion: in these circumstances no mean-field type of approximation may be used to adequately model the process. This, to the same extent, also holds for the Markov spreading processes. To illustrate this claim, in Fig. 4 in the next section, we present the comparison of the classical Markov SIS model and the non-Markovian SEIS model with DTPF chosen to resemble the classical SIS model. From Fig. 4, one may conclude that the assumption of statistical independence of joint events in modeling both Markov SIS and non-Markovian SEIS, under identical circumstances (adequate choice of parameters β, δ and DTPFs, see Section V for details), in low-level epidemics (near the threshold), introduces the identical degree of error between reality (stochastic simulations) and corresponding models. This comes at no surprise, since the approximation, in our model, is utilized in the same fashion as in the Markov spreading models (see eqs. (19) and (20) in the Appendix A).
  As system moves away from the threshold, the accuracy of the model improves and excellent overlap between the system (1) and the reality (stochastic simulations) is reached;
- There is a perfect overlap between the theoretical results for the epidemic threshold, i.e. critical values of the parameter β_c as calculated from the relation (15) and presented in Table II, and results obtained from numerical simulations of the system (1), for example Fig. 2(c), Fig. 2(d), Fig. 3(c) and Fig. 3(d).

From the Fig. 2(a) and Fig. 2(b) it is apparent that, for the processes in which $B(\tau)$ is cumulative in nature, the degree of overlap between the manifestation and recovery period significantly affects the epidemic threshold. With a reduction in the degree of overlap (increase of the time lag of the recovery stage in respect to manifestation stage), the epidemic threshold (i.e. β_c) decreases, and diseases, with these characteristics, tend to have higher epidemic capacity and grow into epidemics.

V. DISCUSSION AND CONCLUSIONS

As previously mentioned, the non-Markovian SEIS model tends to give rise to a broad range of models specific to various diseases and various scenarios related to the course of such diseases. This might be achieved by manipulation of statistical parameters that define the model, $\Gamma(\tau)$ and $B(\tau)$. This notion

This article has been accepted for publication in IEEE Transactions on Network Science and Engineering. This is the author's version which has not been fully edited and content may change prior to final publication. Citation information: DOI 10.1109/TNSE.2021.3125191



(a) (b)

Fig. 4. Comparison of the Markov SIS model, with the non-Markovian SEIS model with DTPF chosen to resemble a Markov SIS. (a) BA1000 graph; (b) WS1000 graph

that the non-Markovian models may extend their usability to a broad range of epidemic sub-models, was first introduced by the Nowzari et al. in [26]. In what follows, we mention just few scenarios that might be derived from the native model:

- Let $B(\tau) = 1$, for all τ, and $\gamma(T-1) = 1$. In this case $\delta_{eff} = 1/(T-2)$, and the epidemic threshold is maximized. When $T \to \infty$, one obtains the classical SI model;
- Let $B(\tau) = 1$, for all τ, and $\gamma(\tau) = \delta(1-\delta)^{\tau-1}s(\tau - 1)$, with $0 < \delta \leq 1$ and where $s(\tau)$ is the Heaviside function. When $T \to \infty$, one obtains the classical SIS model. Numerical simulation of this scenario is presented in Fig. 4;
- The model may be utilized as a base for development of other models that include factors that take into account long term immunity following original exposure, permanent immunity, different sub-stages of a disease etc. In such circumstances, the original system (1) should be accompanied by sets of equations that will modulate the manifesting and recovering probabilities as time lapses. This, however, exceeds by far the scope of this paper and would be addressed elsewhere.

In conclusion, we should note the non-Markovian models have a great potential to more accurately describe spreading processes in comparison with the classical forms, emphasizing certain aspects of the processes, that otherwise would be omitted. In that sense, the presented model may be considered a valid candidate to become an important tool in the future research of spreading phenomena, and in particular in the analysis of contagious diseases epidemics. Furthermore, these models provide the opportunity to be exploited as a base for engineering and design of a wide variety of applications based on stochastic spreading occurring on technological, primarily computer networks and accompanying social platforms. This may be achieved by utilizing different forms of the DTPFs $B(\tau)$ and $\Gamma(\tau)$.

TABLE III
LIST OF LOGICAL VARIABLES AND OPERATIONS USED IN THE ANALYSIS.

Symbol	Meaning
$S_i(t)$	Node i is Susceptible at time t (T/F)
$E_i(t)$	Node i is Exposed at time t (T/F)
$I_i(t)$	Node i is Infectious at time t (T/F)
$\Pi_i(t)$	Susceptible node i contract the agent at time t (T/F)
$A_{ij}(t)$	Link acting in the $j \to i$ direction is active at time t (T/F)
$\beta_{ij}(t)$	Provided $A_{ij}(t) = T$, agent is transferred in the $j \to i$ direction at time t (T/F)
$G_i(t;\tau)$	Node that transitioned $S \to E$ at time $t - \tau$ recovers in the next τ steps (T/F)
$B_i(t;\tau)$	Node that transitioned $S \to E$ at time $t - \tau$ manifest infectiousness at time t (T/F)
$\vee_{i=0}^{N} A_i$	$\vee_{i=0}^{N} A_i = A_0\ OR\ A_1\ OR ... OR\ A_N$
$\wedge_{i=0}^{N} A_i$	$\vee_{i=0}^{N} A_i = A_0\ AND\ A_1\ AND .. AND\ A_N$
$\oplus_{i=0}^{N} A_i$	$\oplus_{i=0}^{N} A_i = A_0\ XOR\ A_1\ XOR .. XOR\ A_N$

APPENDIX A
MODEL DERIVATION

Consider the notation introduced in Table III:

We consider that, for $t \geq T$, the status of the node i as Exposed (Infectious) at time $t+1$ is a result of an occurrence of one of $T-1$ exclusive events: node i that was Susceptible contracted the spread agent at $t-\tau$, and did not recover in the following τ steps (and is manifesting infectiousness at time t):

$$E_i(t+1) = \oplus_{\tau=0}^{T-1} S_i(t-\tau) \wedge \Pi_i(t-\tau) \wedge \overline{G_i(t;\tau)} \quad (16)$$
$$I_i(t+1) = \oplus_{\tau=0}^{T-1} S_i(t-\tau) \wedge \Pi_i(t-\tau) \wedge \overline{G_i(t;\tau)} \wedge B_i(t;\tau)$$

with

$$\Pi_i(t) = \vee_{j=1}^{N} (\beta_{ij}(t) \wedge A_{ij}(t) \wedge I_j(t)) \quad (17)$$

By taking expectations on both sides of the equation (16), and considering $\beta = E[\beta_{ij}(t)]$, $a_{ij} = E[A_{ij}(t)]$, $\Gamma(\tau) = E[G(t;\tau)]$, $B(\tau) = E[B(t;\tau)]$, one obtains:

$$p_i^E(t+1) = \sum_{\tau=0}^{T-1} E[S_i(t-\tau) \wedge \Pi_i(t-\tau)]\overline{\Gamma(\tau)} \quad (18)$$
$$p_i^I(t+1) = \sum_{\tau=0}^{T-1} E[S_i(t-\tau) \wedge \Pi_i(t-\tau)]\overline{\Gamma(\tau)}B(\tau)$$

For convenience, in the following text we will replace the $\wedge$ symbol with simple (logical) multiplication. Consider:

$$E[S_i(t)\Pi_i(t)] = E[\vee_{j=1}^{N}(\beta_i j(t) A_{ij}(t) S_i(t) I_j(t)] \quad (19)$$
$$= \sum_{j=1}^{N} \beta a_{ij} E[S_i(t) I_j(t))]$$
$$- \sum_{j_1 \neq j_2 = 1}^{N} \beta^2 a_{ij1} a_{ij2} E[S_i(t) I_{j1}(t) I_{j2}(t)]$$
$$+ \sum_{j1 \neq j2 \neq j3 = 1}^{N} \beta^3 a_{ij1} a_{ij2} a_{ij3} E[S_i(t) I_{j1}(t) I_{j2}(t) I_{j3}(t)] -$$
$$... + (-1)^{d_i} \beta^{d_i} a_{ij1} a_{ij2} a_{ijd_i} E[S_i(t) I_{j1}(t) I_{j2}(t) .. I_{jd_i}(t)]$$

By employing the assumption of statistical independence of joint events, $E[C_1, C_2, ...C_k] = E[C_1]...E[C_k]$, considering

This article has been accepted for publication in IEEE Transactions on Network Science and Engineering. This is the author's version which has not been fully edited and content may change prior to final publication. Citation information: DOI 10.1109/TNSE.2021.3125191

$E[S_i(t)] = p_i^S(t)$, $E[E_i(t)] = p_i^E(t)$ and $E[I_i(t)] = p_i^I(t)$, one obtains the well known result:

$$E[S_i(t)\Pi_i(t)] = p_i^S \pi_i(t) = (1 - p_i^E(t))\pi_i(t) \quad (20)$$
$$\pi_i(t) = 1 - \prod_{j=1}^{N} \left(1 - \beta a_{ij} p_j^I(t)\right)$$

Notice that for sufficiently small β in (19), higher order terms may be neglected, and, by additionally applying the assumption of statistical independence of joint events, one obtains:

$$E[S_i(t)\Pi_i(t)] = p_i^S(t) \sum_{j=1}^{N} \beta a_{ij} p_j^I(t) \quad (21)$$

This form, with adequate adjustments, has been widely used for Markovian [8], as well as non-Markovian [28], [29], [34] spreading models in continuous time, where it is assumed that within infinitesimal time period, a Susceptible node may be infected only by one of its neighbors (no multiple infectious events may occur).

APPENDIX B
DYNAMICAL BEHAVIOR OF THE PERTURBED SYSTEM (1) FROM $t = 0$ TO $t = T - 1$

For $t \leq T - 1$, the perturbed system (1) takes form:

$$\begin{aligned}
\delta p_i^E(t+1) &= \sum_{\tau=0}^{t} \overline{\Gamma(\tau)} \\
&\times \left(\sum_{j=1}^{N} \beta a_{ij} \delta p_j^I(t-\tau) + \delta p_i^{In} \delta(t-\tau) \right) \\
\delta p_i^I(t+1) &= \sum_{\tau=0}^{t} \overline{\Gamma(\tau)} B(\tau) \\
&\times \left(\sum_{j=1}^{N} \beta a_{ij} \delta p_j^I(t-\tau) + \delta p_i^{In} \delta(t-\tau) \right),
\end{aligned} \quad (22)$$

where $\delta(t)$ is the Kronecker's function. We assume that the dynamical evolution of the system (22) is initiated by a small external (in respected to the network) impulses, denoted with the probabilities δp_i^{In}. The initial conditions are given with $\delta p_i^E(0) = \delta p_i^I(0) = 0$, for all i. System is evolved for $T - 1$ cycles, and sets of initial conditions for the system (3) are obtained:$\{\delta p_i^E(t), \delta p_i^I(t)\}$, $t = 1, .., T - 1$. Dynamical evolution from $t > T$ proceeds with system (3).

APPENDIX C
LEMMAS 1 AND 2

Though trivial, we will prove here two Lemmas that are used in the proof of Theorem 2.

Lemma 1. *Let:*

$$\mathbf{P}(t+1) = \sum_{\tau=0}^{T-1} a_\tau \mathbf{A}\mathbf{P}(t-\tau), \quad (23)$$

is a mapping $\mathbf{P}(t) \in (R^+)^N$, $\mathbf{A}$ *an* $N \times N$ *non-negative matrix and* $a_\tau \in R \geq 0$. *Let* $(\mathbf{P}'(t-\tau))_{\tau=0}^{T-1}$ *and* $(\mathbf{P}''(t-\tau))_{\tau=0}^{T-1}$ *be two sets of initial conditions for the system (23), such that* $\mathbf{P}'(\mathbf{t}-\tau) \leq \mathbf{P}''(\mathbf{t}-\tau)$, $\tau = 0, .., T-1$. *Then:*

$$\mathbf{P}'(t+1) \leq \mathbf{P}''(t+1).$$

Proof: Let $\mathbf{P}'(t-\tau) = \mathbf{P}''(t-\tau) - \delta(t-\tau)$, with $\delta(t-\tau) \geq 0$, $\tau = 0, .., T-1$. Then for each τ one has:

$$\mathbf{A}\mathbf{P}'(t-\tau) \quad = \quad \mathbf{A}\mathbf{P}''(t-\tau) - \mathbf{A}\delta(t-\tau) \leq \mathbf{A}\mathbf{P}''(t-\tau),$$

since the product $\mathbf{A}\delta(t-\tau)$ is non-negative. Then, due to $a_\tau \geq 0$ one has:

$$\sum_{\tau=0}^{T-1} a_\tau \mathbf{A}\mathbf{P}'(t-\tau) \leq \sum_{\tau=0}^{T-1} a_\tau \mathbf{A}\mathbf{P}''(t-\tau)$$

which is equivalent to the assertion of the lemma. Thus the lemma is proved. ■

Lemma 2. *Let* $\mathbf{f}$ *be a vector function such that*

$$\mathbf{f}\left((\mathbf{x}_\tau)_{\tau=0}^{T-2}\right) \geq \mathbf{f}\left((\mathbf{y}_\tau)_{\tau=0}^{T-2}\right), \quad (24)$$

for each $\mathbf{x}_\tau \geq \mathbf{y}_\tau; \mathbf{x}_\tau, \mathbf{y}_\tau \in (R^+)^N$. *Let* $\mathbf{g}$ *be another vector function for which:*

$$\mathbf{f}\left((\mathbf{x}_\tau)_{\tau=0}^{T-2}\right) \geq \mathbf{g}\left((\mathbf{z}_\tau)_{\tau=0}^{T-2}, (\mathbf{x}_\tau)_{\tau=0}^{T-2}\right), \quad (25)$$

for each $\mathbf{x}_\tau, \mathbf{z}_\tau \in [0,1]^N$. *Let* $(\mathbf{R}(\tau))_{\tau=0}^{\infty}$ *be an arbitrary sequence of vectors* $\mathbf{R}(\tau) \in [0,1]^N$. *Let* $\mathbf{P}(t)$ *and* $\mathbf{Q}(t)$ *be sequences obtained by successive application of the functions* $\mathbf{f}$ *and* $\mathbf{g}$ *as:*

$$\begin{aligned}
\mathbf{P}(t+1) &= \mathbf{f}\left((\mathbf{P}(t-\tau))_{\tau=0}^{T-2}\right), \\
\mathbf{Q}(t+1) &= \mathbf{g}\left((\mathbf{R}(t-\tau))_{\tau=0}^{T-2}, (\mathbf{Q}(t-\tau))_{\tau=0}^{T-2}\right),
\end{aligned} \quad (26)$$

Than for identical initial conditions of the sequences $(\mathbf{P}(\tau))_{\tau=0}^{T-2} = (\mathbf{Q}(\tau))_{\tau=0}^{T-2}$ *one has* $\mathbf{P}(t) \geq \mathbf{Q}(t)$, *for any* $t \geq T-1$.

Proof: It is obvious that due to relationship between the functions $\mathbf{f}$ and $\mathbf{g}$ (25), one has $\mathbf{P}(T-1) \geq \mathbf{Q}(T-1)$. Now let us assume that for certain $t \geq T-1$ and all $k; t \geq k$, one has $\mathbf{P}(k) \geq \mathbf{Q}(k)$. Then, from (24) and (25):

$$\begin{aligned}
\mathbf{P}(t+1) &= \mathbf{f}\left((\mathbf{P}(t-\tau))_{\tau=0}^{T-2}\right) \geq \mathbf{f}\left((\mathbf{Q}(t-\tau))_{\tau=0}^{T-2}\right) \\
&\geq \mathbf{g}\left((\mathbf{R}(t-\tau))_{\tau=0}^{T-2}, (\mathbf{Q}(t-\tau))_{\tau=0}^{T-2}\right) = \mathbf{Q}(t+1)
\end{aligned}$$

Therefore, by induction, one can conclude that $\mathbf{P}(t) \geq \mathbf{Q}(t)$ holds for all $t \geq T-1$. ■

ACKNOWLEDGMENT

This research was partially supported by the Faculty of Computer Science and Engineering, at the SS Cyril and Methodius University in Skopje, Macedonia.

REFERENCES

[1] C. Moore and M. E. J. Newman, "Epidemics and percolation in small-world networks," *Phys. Rev. E*, vol. 61, pp. 5678–5682, May 2000.

[2] R. Pastor-Satorras and A. Vespignani, "Epidemic spreading in scale-free networks," *Phys. Rev. Lett.*, vol. 86, no. 14, pp. 3200–3203, Apr 2001.

[3] ——, "Epidemic dynamics and endemic states in complex networks," *Phys. Rev. E*, vol. 63, no. 6, p. 066117, may 2001.

[4] Y. Wang, D. Chakrabarti, C. Wang, and C. Faloutsos, "Epidemic spreading in real networks: An eigenvalue viewpoint," in *In Proc. of the 22nd International Symposium on Reliable Distributed Systems - IEEE SRDS'03*, Oct 2003, pp. 25–34.

[5] D. Chakrabarti, Y. Wang, C. Wang, J. Leskovec, and C. Faloutsos, "Epidemic tresholds in real networks," *ACM Trans. Inf. Syst. Secur.*, vol. 10, no. 4, pp. 13:1 – 13:26, Jan 2008.

[6] M. Draief, A. Ganesh, and L. Massoulié, "Thresholds for virus spread on networks," *Ann. Appl. Probab.*, vol. 18, no. 2, pp. 359 – 378, Apr 2008.

[7] A. J. Ganesh, L. Massoulie, and D. F. Towsley, "The effect of network topology on the spread of epidemics," in *In Proc. IEEE Infocom*, vol. 2, Mar 2005, pp. 1455–1466.

[8] P. Van Mieghem, J. Omic, and R. Kooij, "Virus spread in networks," *IEEE/ACM Trans. Netw.*, vol. 17, no. 1, pp. 1 – 14, Feb 2009.

[9] E. Cator and P. Van Mieghem, "Second-order mean-field susceptible-infected-susceptible epidemic threshold," *Phys. Rev. E*, vol. 85, no. 5, p. 056111, May 2012.

[10] M. Salehi, R. Sharma, M. Marzolla, M. Magnani, P. Siyari, and D. Montesi, "Spreading processes in multilayer networks," *IEEE Transactions on Network Science and Engineering*, vol. 2, no. 2, pp. 65–83, 2015.

[11] M. Ogura and V. M. Preciado, "Stability of spreading processes over time-varying large-scale networks," *IEEE Transactions on Network Science and Engineering*, vol. 3, no. 1, pp. 44–57, 2016.

[12] B. Qu and H. Wang, "Sis epidemic spreading with heterogeneous infection rates," *IEEE Transactions on Network Science and Engineering*, vol. 4, no. 3, pp. 177–186, 2017.

[13] A. Majdandzic, B. Podobnik, S. V. Buldyrev, D. Y. Kenett, S. Havlin, and H. Eugene Stanley, "Spontaneous recovery in dynamical networks," *Nature Physics*, vol. 10, no. 1, pp. 34–38, Jan 2014. [Online]. Available: https://doi.org/10.1038/nphys2819

[14] C. Nowzari, V. M. Preciado, and G. J. Pappas, "Analysis and control of epidemics: A survey of spreading processes on complex networks," *IEEE Control Systems Magazine*, vol. 36, no. 1, pp. 26–46, 2016.

[15] G. Tong, W. Wu, S. Tang, and D. Du, "Adaptive influence maximization in dynamic social networks," *IEEE/ACM Transactions on Networking*, vol. 25, no. 1, pp. 112–125, 2017.

[16] P. Basaras, G. Iosifidis, D. Katsaros, and L. Tassiulas, "Identifying influential spreaders in complex multilayer networks: A centrality perspective," *IEEE Transactions on Network Science and Engineering*, vol. 6, no. 1, pp. 31–45, 2019.

[17] Yan Wan, S. Roy, and A. Saberi, "Network design problems for controlling virus spread," in *2007 46th IEEE Conference on Decision and Control*, 2007, pp. 3925–3932.

[18] Y. Wan, S. Roy, and A. Saberi, "Designing spatially heterogeneous strategies for control of virus spread," *IET Syst Biol*, vol. 2, no. 4, pp. 184–201, Jul 2008.

[19] J. Guo, T. Chen, and W. Wu, "A multi-feature diffusion model: Rumor blocking in social networks," *IEEE/ACM Transactions on Networking*, vol. 29, no. 1, pp. 386–397, 2021.

[20] H. T. Nguyen, M. T. Thai, and T. N. Dinh, "A billion-scale approximation algorithm for maximizing benefit in viral marketing," *IEEE/ACM Transactions on Networking*, vol. 25, no. 4, pp. 2419–2429, 2017.

[21] B. Prasse and P. Van Mieghem, "Network reconstruction and prediction of epidemic outbreaks for general group-based compartmental epidemic models," *IEEE Transactions on Network Science and Engineering*, vol. 7, no. 4, pp. 2755–2764, 2020.

[22] J. Y. Chun, G. Baek, and Y. Kim, "Transmission onset distribution of covid-19," *International Journal of Infectious Diseases*, vol. 99, pp. 403–407, 2020. [Online]. Available: https://www.sciencedirect.com/science/article/pii/S1201971220306123

[23] J. Qin, C. You, Q. Lin, T. Hu, S. Yu, and X.-H. Zhou, "Estimation of incubation period distribution of covid-19 using disease onset forward time: A novel cross-sectional and forward follow-up study," *Science Advances*, vol. 6, no. 33, 2020. [Online]. Available: https://advances.sciencemag.org/content/6/33/eabc1202

[24] J. Sreevalsan-Nair, R. R. Vangimalla, and P. R. Ghogale, "Analysis of clinical recovery-period and recovery rate estimation of the first 1000 covid-19 patients in singapore," *medRxiv*, 2020. [Online]. Available: https://www.medrxiv.org/content/early/2020/04/24/2020.04.17.20069724

[25] M. Boguñá, L. F. Lafuerza, R. Toral, and M. A. Serrano, "Simulating non-markovian stochastic processes," *Phys. Rev. E*, vol. 90, p. 042108, Oct 2014. [Online]. Available: https://link.aps.org/doi/10.1103/PhysRevE.90.042108

[26] C. Nowzari, M. Ogura, V. M. Preciado, and G. J. Pappas, "A general class of spreading processes with non-markovian dynamics," in *2015 54th IEEE Conference on Decision and Control (CDC)*, 2015, pp. 5073–5078.

[27] P. Van Mieghem and R. van de Bovenkamp, "Non-markovian infection spread dramatically alters the susceptible-infected-susceptible epidemic threshold in networks," *Phys. Rev. Lett.*, vol. 110, p. 108701, Mar 2013. [Online]. Available: https://link.aps.org/doi/10.1103/PhysRevLett.110.108701

[28] P. Van Mieghem and Q. Liu, "Explicit non-markovian susceptible-infected-susceptible mean-field epidemic threshold for weibull and gamma infections but poisson curings," *Phys. Rev. E*, vol. 100, p. 022317, Aug 2019. [Online]. Available: https://link.aps.org/doi/10.1103/PhysRevE.100.022317

[29] Q. Liu and P. Van Mieghem, "Burst of virus infection and a possibly largest epidemic threshold of non-markovian susceptible-infected-susceptible processes on networks," *Phys. Rev. E*, vol. 97, p. 022309, Feb 2018. [Online]. Available: https://link.aps.org/doi/10.1103/PhysRevE.97.022309

[30] M. Starnini, J. P. Gleeson, and M. Boguñá, "Equivalence between non-markovian and markovian dynamics in epidemic spreading processes," *Phys. Rev. Lett.*, vol. 118, p. 128301, Mar 2017. [Online]. Available: https://link.aps.org/doi/10.1103/PhysRevLett.118.128301

[31] N. Georgiou, I. Z. Kiss, and E. Scalas, "Solvable non-markovian dynamic network," *Phys. Rev. E*, vol. 92, p. 042801, Oct 2015. [Online]. Available: https://link.aps.org/doi/10.1103/PhysRevE.92.042801

[32] I. Z. Kiss, G. Röst, and Z. Vizi, "Generalization of pairwise models to non-markovian epidemics on networks," *Phys. Rev. Lett.*, vol. 115, p. 078701, Aug 2015. [Online]. Available: https://link.aps.org/doi/10.1103/PhysRevLett.115.078701

[33] N. Sherborne, J. C. Miller, K. B. Blyuss, and I. Z. Kiss, "Mean-field models for non-markovian epidemics on networks," *Journal of Mathematical Biology*, vol. 76, no. 3, pp. 755–778, Feb 2018. [Online]. Available: https://doi.org/10.1007/s00285-017-1155-0

[34] M. Feng, S.-M. Cai, M. Tang, and Y.-C. Lai, "Equivalence and its invalidation between non-markovian and markovian spreading dynamics on complex networks," *Nature Communications*, vol. 10, no. 1, p. 3748, Aug 2019. [Online]. Available: https://doi.org/10.1038/s41467-019-11763-z

[35] M. Kim, D. Paini, and R. Jurdak, "Modeling stochastic processes in disease spread across a heterogeneous social system," *Proceedings of the National Academy of Sciences*, vol. 116, no. 2, pp. 401–406, 2019. [Online]. Available: https://www.pnas.org/content/116/2/401

[36] L. Zino, A. Rizzo, and M. Porfiri, "Modeling memory effects in activity-driven networks," *SIAM Journal on Applied Dynamical Systems*, vol. 17, no. 4, pp. 2830–2854, 2018. [Online]. Available: https://doi.org/10.1137/18M1171485

[37] H. J. T. Unwin, I. Routledge, S. Flaxman, M.-A. Rizoiu, S. Lai, J. Cohen, D. J. Weiss, S. Mishra, and S. Bhatt, "Using hawkes processes to model imported and local malaria cases in near-elimination settings," *PLOS Computational Biology*, vol. 17, no. 4, pp. 1–18, 04 2021. [Online]. Available: https://doi.org/10.1371/journal.pcbi.1008830

[38] M. Fan, M. Y. Li, and K. Wang, "Global stability of an SEIS epidemic model with recruitment and a varying total population size," *Mathematical Biosciences*, vol. 170, no. 2, pp. 199 – 208, 2001. [Online]. Available: http://www.sciencedirect.com/science/article/pii/S0025556400000675

[39] M. López, A. Peinado, and A. Ortiz, "A SEIS model for propagation of random jamming attacks in wireless sensor networks," in *International Joint Conference SOCO'16-CISIS'16-ICEUTE'16*, M. Graña, J. M. López-Guede, O. Etxaniz, Á. Herrero, H. Quintián, and E. Corchado, Eds. Cham: Springer International Publishing, 2017, pp. 668–677.

[40] A. Korobeinikov, "Lyapunov functions and global properties for SEIR and SEIS epidemic models," *Math Med Biol*, vol. 21, no. 2, pp. 75–83, Jun 2004.

[41] E. Foxall, "The SEIS model, or, the contact process with a latent stage," *Journal of Applied Probability*, vol. 53, no. 3, p. 783–801, 2016.

[42] G. Blelloch, "Lecture 13 — shortest weighted paths," pp. 1–8, October 2012. [Online]. Available: http://www.cs.cmu.edu/afs/cs/academic/class/15210-f12/www/lectures/lecture13.pdf

[43] S. Gómez, A. Arenas, J. Borge-Holthoefer, S. Meloni, and Y. Moreno, "Discrete-time markov chain approach to contact-based disease spreading in complex networks," *Eur. Phys. Lett.*, vol. 89, no. 3, p. 38009, Feb 2010.

[44] ——, "Probabilistic framework for epidemic spreading in complex networks," *Int. J. Complex Systems in Science*, vol. 1, pp. 47–51, Feb 2011.

[45] G. Frobenius, *Über Matrizen aus nicht negativen Elementen*, ser. Preussische Akademie der Wissenschaften Berlin: Sitzungsberichte der Preußischen Akademie der Wissenschaften zu Berlin. Reichsdr., 1912.

[46] O. Perron, "Zur theorie der matrices," *Mathematische Annalen*, vol. 64, no. 2, pp. 248–263, Jun 1907. [Online]. Available: https://doi.org/10.1007/BF01449896

[47] F. de Terán, F. M. Dopico, and J. Pérez, "Backward stability of polynomial root-finding using fiedler companion matrices," *IMA Journal of Numerical Analysis*, vol. 36, no. 1, pp. 133–173, 2016.

[48] A. Ivanov, H. Aliyev, and B. Bakiyev, "Analytical method of finding polynomial roots by using the eigenvectors, eigenvalues apparatus," in *2015 Twelve International Conference on Electronics Computer and Computation (ICECCO)*, 2015, pp. 1–3.

[49] J. L. Aurentz, R. Vandebril, and D. S. Watkins, "Fast computation of the zeros of a polynomial via factorization of the companion matrix," *SIAM Journal on Scientific Computing*, vol. 35, no. 1, pp. A255–A269, 2013. [Online]. Available: https://doi.org/10.1137/120865392

[50] C. Meyer, *Matrix Analysis and Applied Linear Algebra*, ser. Other Titles in Applied Mathematics. Society for Industrial and Applied Mathematics, 2000.

[51] A. Jeffrey and D. Zwillinger, *Table of Integrals, Series, and Products*, 7th ed. Elsevier Science, 2007. [Online]. Available: https://books.google.mk/books?id=aBgFYxKHUjsC

[52] R. Albert and A.-L. Barabási, "Statistical mechanics of complex networks," *Reviews of Modern Physics*, vol. 74, no. 1, pp. 47 – 97, Jan 2002.

[53] S. H. Watts, D. J.and Strogatz, "Collective dynamics of 'small-world' networks," *Nature*, vol. 393, no. 6684, p. pp. 440–442, Jun 1998.

Igor Tomovski received his BSc, MSc and PhD in 1997,2008 and 2012 respectively from the Faculty of Electrical Engineering, Ss Cyril and Methodius University in Skopje. He is a research scientist with the Research Center for Computer Sciences and Information Technologies at the Macedonian Academy of Sciences and Arts (MANU). His research interests include complex systems and networks and nonlinear dynamics.

Lasko Basnarkov received the BSc degree in electronics and telecommunications in 2000, and the MSc and PhD degrees in theoretical physics in 2005, and 2009, respectively, all at the Ss. Cyril and Methodius University in Skopje, Macedonia. He is professor of physics at the faculty of Computer Science and Engineering, Ss. Cyril and Methodius University, Skopje and associated with the Research Center for Computer Sciences and Information Technologies at the Macedonian Academy of Sciences and Arts (MANU). His research interests are mainly in nonlinear dynamics and statistical physics and their applications in econophysics, financial engineering, game theory, and mathematical epidemiology.

Alajdin Abazi received his BSc, MSc and PhD in Electrical Engineering from the University of Zagreb in 1965, 1973 and 1977 respectively. In the period 1979-81, he was appointed Dean of the Technical Faculty in Prishtina. Acad. Abazi is founding member of the South East European University (SEEU) in Tetovo and later appointed SEEU Rector. In 2006, he was elected a full member in the Department of Technical Sciences of the Macedonian Academy of Sciences and Arts. Since 2011 he is a member-founder of the Albanian Academy of Arts and Sciences, and then in 2012 he was elected a foreign member of the Academy of Sciences and Arts of Kosovo. Acad. Abazi is a former Secretary of the Department of Technical Sciences at MANU. From 2020 he is the Head of the Research Center for Computer Sciences and Information Technologies at MANU. His research interests include higher education, energy and sustainable development, theory of power, power quality, and information technologies for sustainable development.