# Spread of epidemics in time-dependent networks

V. S. Bokharaie, O. Mason and F. Wirth

***Abstract*— We consider SIS models for the spread of epidemics. In particular we consider the so called nonhomogeneous case, in which the probability of infection and recovery are not uniform but depend on a neighborhood graph which describes the possibility of infection between individuals. In addition it is assumed, that infection, recovery probabilities as well as the interconnection structure may change with time. Using the concept of the joint spectral radius of a family of matrices conditions are provided that guarantee robust extinction of the epidemics.**

## I. Introduction

Mathematical modelling of the spread of diseases is a classical topic in mathematical biology, [1], [2]. Interestingly, in recent times models of disease spread have been applied by the computer science community to model the spread of malignant software in computer networks, [3], [4], [5], [6], [7]. This has the benefit that in many cases the spread of computer viruses is much better documented than the spread of viruses in biological populations.

The modelling of epidemics distinguishes two cases of fundamental difference. In one case the state of an individual may change from susceptible to infected and after recovery to susceptible again, the corresponding acronym is SIS models. On the other hand the change of states may be evolve from susceptible to infected to recovered with the assumption that recovered individuals cannot become infected again (SIR models). In this paper we concentrate on SIS models.

In general, there are several approaches to modelling SIS epidemics. These range from discrete-time models and ordinary differential equations to Markov chain models. Most notably however the difference between homogeneous and nonhomogeneous models has been widely discussed in recent years. In homogeneous models a basic assumption is that infection probabilities are uniform over the whole population and the same assumption is made for recovery probabilities. While this leads to appealingly simple models the drawback to be expected is that too much is oversimplified. In non-homogeneous models a graph is introduced that describes the interaction between individuals and the possibilities of one individual infecting another. Indeed the influence of the graph structure on the dynamics of the infection is well documented, [4], [5].

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The paper is organized as follows. In the next Section II we provide a description of the problem being considered, develop a discrete-time model for non-homogeneous disease spread and present criteria for extinction and epidemic terms of the *joint spectral radius* of a set of matrices. In Section III, the corresponding analysis for continuous models is described.

## II. Problem Description

We consider an undirected graph $G = (V, E)$ of $n = |V|$ individuals. Each individual can either be healthy or infected. Infections can spread in one time step among neighbors of the graph. That is, if there is an edge $(i, j)$ between two vertices $i$ and $j$ and one of the vertices, say $i$, is infected, then there is a certain probability $\beta$ that in the next time step $j$ will be infected. Conversely, there is also a certain probability $\delta$ that node $i$ will be cured in the next time step. In the following we develop a model for the evolution of probabilities of infection in the graph. In contrast to results in the literature we will not assume that the graph $G$, or $\delta$ and $\beta$ are fixed quantities but allow some time dependence.

We denote the set of neighbors of node $i$ at time $t$ by $N_i(t)$. We assume that the process of infection between neighbors is independent from the other probabilistic events. The probability that node $i$ is infected at time $t$ is denoted by $p_i(t)$. Following [3] the probability $\zeta_i(t)$ that in the $t$-th time interval node $i$ is not infected by its neighbours is

$$\zeta_i(t) = \prod_{j\in N_i(t)} (p_j(t)(1-\beta_j(t)) + (1-p_j(t))) \quad (1)$$

$$= \prod_{j\in N_i(t)} (1-\beta_j(t)p_j(t)) \ , t \in \mathbb{N}\,. \quad (2)$$

Assuming independence of the probabilistic events that can take place the time evolution of the health probability of node $i$ thus satisfies

$$1 - p_i(t+1) = \quad (3)$$

$$(1-p_i(t))\zeta_i(t) + \delta p_i(t)\zeta_i(t) + \frac{1}{2}\delta p_i(t)(1-\zeta_i(t)) = \quad (4)$$

$$\frac{1}{2}\delta p_i(t) + \zeta_i(t)\left(1 + \left(\frac{1}{2}\delta - 1\right)p_i(t)\right), \quad t \in \mathbb{N}.$$

In (4) we have first a term that quantifies the probability of not being infected at time $t$ and not receiving infection from $t$ to $t+1$, then we have a term characterising recovery after being infected at time $t$ and finally there is a term that characterises recovery and reinfection from $t$ to $t+1$. In this formula the factor $1/2$ is somewhat arbitrary and would depend on the particular sequence of events. We thus assume

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implicitly that in the time interval from $t$ to $t+1$ the event of recovery as well as the event of being infected by a neighbor happens in a uniform manner.

To simplify the previous equations note that

$$\zeta_i(t)\left(1+\left(\frac{1}{2}\delta-1\right)p_i(t)\right)= \quad (5)$$
$$1+\left(\frac{1}{2}\delta-1\right)p_i(t)+\sum_{j\in N_i(t)}\beta_j(t)p_j(t)+o(\|p\|)\,.$$

Defining $D(t)=\operatorname{diag}\begin{pmatrix}\delta_1(t) & \dots & \delta_n(t)\end{pmatrix}$ as the diagonal matrix of recovery probabilities at time $t$ and $A(\beta)$ as the weighted adjacency matrix of the graph of connections this leads to the final formulation of the evolution of $p(t)$ as

$$p(t+1)=(I-D(t)+A(\beta(t)))\,p(t)+F_{\delta(t),\beta(t)}(p(t))\,, \quad (6)$$

where the $i$th component of $F_{\delta(t),\beta(t)}(t)$ contains the higher order products contained in (5) , $i=1,\dots,n$ and the index denotes the dependence on the special choice of $\delta$ and $A(\beta)$. We note for later use, that the set of possible $A(\beta)$ describing the interconnection structure and infection probabilities is bounded because it is contained in the set of nonnegative matrices with entries in the interval $[0,1]$. Thus for every $r>0$ there exists a uniform constant $\eta$ (depending on $r$) such that for all $p, \|p\|\le r$ and all choices of $\delta,\beta$ we have

$$\|F_{\delta,\beta}(p)\|\le\eta\|p\|^2\,. \quad (7)$$

Thus if we linearize the equation (3) in the equilibrium of interest $p^*=\begin{pmatrix}p_1 & \dots & p_n\end{pmatrix}^T=0$ the linear system becomes

$$p(t+1)=(I-D(t))p(t)+A(\beta(t))p(t)\,. \quad (8)$$

We now assume we have a set $\mathcal{M}\subset\mathbb{R}_+^{n\times n}$ of matrices describing the possible matrices that can appear in (8). It is reasonable to assume, that

$$\mathcal{M}=I-\mathcal{D}+\mathcal{A}\,,$$

where $\mathcal{D}$ represents the set of possible recovery matrices and $\mathcal{A}$ is the set of possible disease transmission matrices, which are all the weighted adjacency matrices appearing. By assumption the matrices in $\mathcal{A}$ have zero diagonal and entries in $[0,1]$.

Associated to the set of matrices $\mathcal{M}$ is the joint spectral radius, which is defined by

$$\rho(\mathcal{M}):=\lim_{t\to\infty}\sup\{\|M(t-1)M(t-2)\dots M(0)\|^{1/t} \quad (9)$$
$$\mid M(s)\in\mathcal{M}, s=0,\dots,t-1\}\,. \quad (10)$$

Recall that $\rho(\mathcal{M})<1$ is equivalent to the existence of constants $C\ge1,\gamma\in(0,1)$ such that for all $t\in\mathbb{N}$ we have

$$\|M_{t-1}\cdots M_1M_0\|\le C\gamma^t \quad \text{for all } M_s\in\mathcal{M}, s=0,\dots,t\,. \quad (11)$$

Thus $\rho(\mathcal{M})<1$ characterizes exponential stability of the linear inclusion

$$p(t+1)\in\{Mp(t)\mid M\in\mathcal{M}\}\ .$$

It is known that this stability property is equivalent to the existence of norms with respect to which all matrices $M\in\mathcal{M}$ are contractions. This is the content of the following theorem, [8].

*Theorem 2.1:* Let $\mathcal{M}\subset\mathbb{R}^{n\times n}$ be a nonempty compact set of matrices, then $\rho(\mathcal{M})<1$ is equivalent to the existence of a norm $v$ on $\mathbb{R}^n$ and a constant $\alpha$ such that

$$v(M)\le\alpha<1\,,\quad \text{for all } M\in\mathcal{M}\,.$$

This is also of relevance to the nonlinear system as the next two results show.

*Theorem 2.2:* Assume that the joint spectral radius of $\mathcal{M}$ satisfies

$$\rho(\mathcal{M})<1 \quad (12)$$

then $p^*=0$ is a locally exponentially stable fixed point of (6).

*Proof:* The proof follows a classical approach of linearization theory for stability. According to Theorem 2.1 we can pick a norm $v$ on $\mathbb{R}^n$ such that $v(M)\le\alpha<1$ for all $M\in\mathcal{M}$. Let $C>1$ be a constant such that $v(x)\le C\|x\|$ for all $x\in\mathbb{R}^n$. Using the norm $v$ as a Lyapunov function along the solutions of (6) we obtain for $\|p(t)\|\le r$ and a fixed $r>0$ that

$$\begin{aligned}v(p(t+1))&\le v(M_{\delta(t),\beta(t)}p(t))+v(F_{\delta(t),\beta(t)}(p(t)))\\&\le\alpha v(p(t))+v(F_{\delta(t),\beta(t)}(p(t)))\\&\le\alpha v(p(t))+C\eta v(p(t))\end{aligned}$$

If we choose $r$ sufficiently small, so that $\alpha+C\eta<1$, we see that solutions are decaying no matter which choice of $\delta$ and $A(\beta)$ is applied. This shows the assertion. ∎

In other words the condition $\rho(\mathcal{M})<1$ guarantees that infections will die out for all possible time evolutions of the nonlinear system (3). Similarly, a criterion for epidemics is

*Theorem 2.3:* Assume that the joint spectral radius of $\mathcal{M}$ satisfies

$$\rho(\mathcal{M})>1 \quad (13)$$

then $p^*=0$ is a locally exponentially unstable fixed point of (3).

*Proof:* By the generalized spectral radius theorem, due to Berger and Wang, [9], [8] if $\rho(\mathcal{M})>1$ then there is a finite sequence $(M_0,M_1,M_2,\dots,M_{t-1})\in\mathcal{M}^t$ such that the spectral radius of the product

$$r\left(M_{t-1}\cdots M_1M_0\right)>1\,.$$

If we consider the corresponding sequence of choices for the infection and recovery probabilities $(\delta_s,A(\beta_s)), s=1,\dots,t$ and consider the periodic nonlinear system (6) obtained by periodically applying this sequence, then we obtain a system which has the periodic linear system given by the matrices $M_0,\dots,M_{t-1}$ as its linearization. As the linearization is exponentially unstable, so is the fixed point $p^*=0$ for the corresponding nonlinear system. ∎

It should be noted that the content of the previous result is that for certain choices of sequences $(\delta_s,A(\beta_s)), s\in\mathbb{N}$ the nonlinear system is unstable. This does not rule out the possibility that for particular choices the fixed point is stable.

## III. Continuous Models

The results that have been obtained in the previous section translate readily to continuous time SIS models. Again $p_i(t)$ denotes the probability that node $i$ is infected at time $t$, but now $t \in \mathbb{R}_+$. A model class of this type is given in [7] as

$$\dot{p}_i(t) = \beta(1 - p_i(1)) \sum_{j \in N_i} p_j(t) - \delta_i p_i(t)\,, \qquad (14)$$

where now the constant $\beta$ may be interpreted as the infection rate of the disease and $\delta_i$ is the recovery rate of individual $i$. As in the discrete time case we generalize this model to the time-varying case by considering

$$\dot{p}_i(t) = (1 - p_i(1)) \sum_{j \in N_i(t)} \beta_{ij}(t) p_j(t) - \delta_i(t) p_i(t)\,, \quad (15)$$

where we assume that the functions $\beta_{ij}, \delta_i, N_i$ are sufficiently regular to ensure existence of solutions. E.g. by considering standard switching signals or by imposing a measurability assumption. The linearization of (15) at $p^* = 0$ is then given by

$$\dot{p}_i(t) = \sum_{j \in N_i(t)} \beta_{ij}(t) p_j(t) - \delta_i(t) p_i(t)\,. \qquad (16)$$

which is easily seen to be the infinitesimal formulation of the discrete-time systems described in (8). Formulated as a system we obtain

$$\dot{p} = \left(A(\beta(t)) - D(t)\right) p(t)\,, \qquad (17)$$

where $D(t) = \operatorname{diag}\begin{pmatrix}\delta_1(t) & \dots & \delta_n(t)\end{pmatrix}$ is the diagonal matrix of recovery rates at time $t$ and $A(\beta(t))$ as the weighted adjacency matrix of the graph of connections.

We now assume we have a set $\mathcal{M} \subset \mathbb{R}_+^{n \times n}$ of matrices that describe the possible matrices that can appear in (17). It is reasonable to assume, that

$$\mathcal{M} = \mathcal{A} - \mathcal{D}\,,$$

where $\mathcal{D}$ represents the set of possible recovery matrices and $\mathcal{A}$ is the set of possible disease transmission matrices, which are all the weighted adjacency matrices appearing.

As in the discrete time case, we can define the continuous time-version of the joint spectral radius (also called maximal Lyapunov exponent) $\rho$ corresponding to the linear inclusion

$$\dot{p} \in \{Mp \mid M \in \mathcal{M}\}\,, \qquad (18)$$

see [10], [11]. The proof of the following two results is then similar to that of the discrete time case. In particular, also in the continuous time case it is known that exponential stability of the inclusion (18) is equivalent to the existence of a norm which serves as a Lyapunov function.

*Theorem 3.1:* Assume that the joint spectral radius of the linear inclusion (18) satisfies

$$\rho(\mathcal{M}) < 1 \qquad (19)$$

then $p^* = 0$ is a locally exponentially stable fixed point of the system given by (15).

*Theorem 3.2:* Assume that the joint spectral radius of the linear inclusion (18) satisfies

$$\rho(\mathcal{M}) > 1 \qquad (20)$$

then $p^* = 0$ is a locally exponentially unstable fixed point of the system given by (15).

## IV. Conclusion and Future Work

In this note we have presented a brief introduction into the modelling of the spread of epidemics in time-varying situations. For certain cases the conditions on the joint spectral radius can be significantly simplified. We will explore this in future publications. Similarly, we plan to investigate related Markov models.

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