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The Solution of the NIMFA Epidemic Model around the Epidemic Threshold

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Abstract: Non-linear differential equations are a common approach to modelling the spread of infectious diseases. Unfortunately, a closed-form solution is not known for the majority of epidemic models, which restricts an in-depth understanding of the evolution of the virus. In this work, we solve the differential equations of the NIMFA epidemic model around the epidemic threshold, provided that the initial viral state is small or proportional to the steady-state. The solution of the NIMFA model around the epidemic threshold is of particular importance for disease control measures that aim to eradicate the infectious disease.

Keywords: NIMFA differential equations, Viral state dynamics, Epidemiology

1. INTRODUCTION

Rooted in the study of infectious diseases, modern epidemiology encompasses a plethora of spreading phenomena such as trends, voter dynamics, and posts on online social media. Two characteristics are mutual to all epidemic processes. First, each individual can be infected by the virus (trend, opinion, etc.) or healthy. Second, the virus spreads from an infected individual to a healthy individual, if the two individuals are in contact. In this work, we consider the spread of a virus between groups of individuals, where a group corresponds to, for instance, a household or a contiguous geographic region.

More precisely, we study the non-linear differential equations of the N-Intertwined Mean-Field Approximation (NIMFA) of the Susceptible-Infected-Susceptible (SIS) epidemic process (Van Mieghem et al., 2009; Van Mieghem, 2011). Since there seems to be no closed-form solution of the NIMFA equations, the precise behaviour of the viral state $v(t)$ over time t is not fully understood. Our main result in Theorem 1 below specifies the solution $v_{\text{apx}}(t)$ of the NIMFA equations just above the epidemic threshold for small initial viral states $v(0)$. The solution $v_{\text{apx}}(t)$ around the epidemic threshold is useful for two reasons. First, the solution $v_{\text{apx}}(t)$ is an accurate approximation also for viral spreads that are further above the epidemic threshold. Second, to effectively mitigate the prevalence of the virus, disease control measures (e.g., vaccinations or quarantine) must confine the viral spread as close as possible to the epidemic threshold, i.e., in the regime where the solution $v_{\text{apx}}(t)$ is accurate.

2. THE NIMFA EPIDEMIC MODEL

We consider the spread of a virus between N disjoint groups of individuals that are either healthy or infected. For every group $i = 1, ..., N$, the *viral state* $v_i(t) \in [0, 1]$ equals to the fraction of infected individuals at continuous time $t \geq 0$. The viral state vector is defined as $v(t) = (v_1(t), ..., v_N(t))^T$. The *NIMFA epidemic model* (Van Mieghem et al., 2009; Van Mieghem, 2011; Lajmanovich and Yorke, 1976) describes the viral state evolution for every group i as

$$\frac{dv_i(t)}{dt} = -\delta_i v_i(t) + (1 - v_i(t)) \sum_{j=1}^{N} \beta_{ij} v_j(t). \qquad (1)$$

Here, $\delta_i > 0$ is the *curing rate* of group i, and $\beta_{ij} \geq 0$ is the *infection rate* from group j to group i. Hence, if the infection rate β_{ij} is strictly greater zero, then infections do occur from group j to group i, for instance, because group i and j correspond to two adjacent geographical regions. We denote the $N \times N$ *effective infection rate matrix* by W with the elements $(W)_{ij} = \beta_{ij}/\delta_i$. We assume that the effective infection rate matrix W corresponds to a strongly connected graph. The *basic reproduction number* R_0 of the NIMFA epidemic model (1) is defined as the spectral radius of the effective infection rate matrix W. Around the *epidemic threshold criterion* $R_0 = 1$, there is a phase transition (Lajmanovich and Yorke, 1976; Khanafer et al., 2016): If $R_0 \leq 1$, then the only equilibrium is the all-healthy state $v(t) = 0$, which is globally asymptotically stable. Else, if $R_0 > 1$, then there is a second equilibrium, the $N \times 1$ steady-state vector v_∞ with positive components, and the steady-state v_∞ is globally asymptotically stable for every initial viral state $v(0) \neq 0$. Hence, the steady-state v_∞ is the long-term, or endemic, viral state, which can be computed in three ways. First, the steady-state v_∞ follows by solving (1) with $\frac{dv_i(t)}{dt} = 0$ for all i numerically. Second, the steady-state v_∞ can be obtained via a Taylor expansion (Van Mieghem, 2012). Third, the steady-state v_∞ can be expressed as a continued fraction expansion (Van Mieghem et al., 2009; Van Mieghem, 2011). In discrete time, the viral state dynamics of the NIMFA epidemic model have been studied in (Ahn and Hassibi, 2013; Paré et al., 2018; Prasse and Van Mieghem, 2019b), and a method for estimating the parameters from observing the viral state $v(t)$ has been proposed in (Prasse and Van Mieghem, 2018). An extension of NIMFA (1) is to

consider that the spreading parameters δ_i and β_{ij} can be controlled. We refer the reader to (Nowzari et al., 2016) for an overview of approaches that aim to control the epidemic outbreak.

3. SOLUTION OF THE NIMFA MODEL AROUND THE EPIDEMIC THRESHOLD

By solving the NIMFA equations "around the epidemic threshold", we mean solving the differential equations (1) in the limit $R_0 \downarrow 1$. Since the basic reproduction number R_0 is the spectral radius of the effective infection rate matrix W, the basic reproduction number R_0 approaches 1 due to the following disease control measures: the curing rates δ_i are increased or the infection rates β_{ij} are decreased (or both). Since the endemic viral state v_∞ approaches zero as $R_0 \downarrow 1$, the objective of any disease control is indeed to steer the basic reproduction number R_0 as close as possible to 1.

For the vast majority of real-world epidemics, the initial viral state $v_i(0)$ is below the steady-state $v_{\infty,i}$ for every group i. In other words, at time $t = 0$ the prevalence has not reached its maximum yet. If the initial viral state $v(0)$ is smaller than the steady-state v_∞, then the viral state $v_i(t)$ does not overshoot the steady-state $v_{\infty,i}$ at any time $t \geq 0$:

Lemma 1. (Prasse and Van Mieghem, 2019a).
Suppose that the basic reproduction number R_0 is greater than 1, the infection rates satisfy $\beta_{ij} = \beta_{ji}$ for all groups i, j, and that the initial viral state $v_i(0)$ of every group i is in $[0, v_{\infty,i}]$. Then, it holds that $v_i(t) \in [0, v_{\infty,i}]$ for every group i at every time $t \geq 0$.

Based on Lemma 1, we confine our analysis of the viral state dynamics to the positive invariant set

$$\mathcal{V} = \left\{ v \in \mathbb{R}^N \middle| 0 \leq v_i \leq v_{\infty,i},\ \forall i = 1, ..., N \right\}.$$

In (Prasse and Van Mieghem, 2018), we observed that the $N \times 1$ viral state vector $v(t)$ remains, approximately, in a subspace of dimension $m << N$ at all times $t \geq 0$. More precisely, at every time t it holds that

$$v(t) \approx c_1(t)y_1 + ... + c_m(t)y_m, \tag{2}$$

where the functions $c_1(t), ..., c_m(t)$ are scalar and the vectors $y_1, ..., y_m$ are orthogonal. More so, numerical simulations indicate that, if the basic reproduction number R_0 is close to one, then the viral state $v(t)$ is well approximated by only $m = 1$ scalar function $c_1(t)$ and vector y_1, which is the focus of this work. Since the viral state $v(t)$ converges to the steady-state vector v_∞ as $t \to \infty$, a natural choice for the vector y_1 is $y_1 = v_\infty$. We drop the subscript of the scalar function $c_1(t)$, and decompose the initial viral state vector $v(0)$ as

$$v(0) = c(0)v_\infty + \xi(0), \tag{3}$$

where the $N \times 1$ vector $\xi(0)$ is orthogonal to the steady-state vector v_∞, and the scalar $c(0)$ equals $c(0) = v_\infty^T v(0)/\|v_\infty\|_2^2$. Then, the NIMFA epidemic model (1) has a closed-form solution around the epidemic threshold $R_0 = 1$ when the vector $\xi(0)$ is small:

Theorem 1. (Prasse and Van Mieghem, 2019a).
Suppose that the infection rates satisfy $\beta_{ij} = \beta_{ji}$ for all groups i, j and that the initial viral state $v_i(0)$ of every group i is in $[0, v_{\infty,i}]$. Furthermore, suppose that for some constant $p > 1$, $\|\xi(0)\|_2 = \mathcal{O}\left((R_0 - 1)^p\right)$ when $R_0 \downarrow 1$, and define

$$v_{\text{apx}}(t) = \frac{1}{2}\left(1 + \tanh\left(\frac{w}{2}t + \Upsilon(v(0))\right)\right) v_\infty. \tag{4}$$

Here, the scalars $\Upsilon(v(0))$ and w equal

$$\Upsilon(v(0)) = \operatorname{arctanh}\left(2\frac{v_\infty^T v(0)}{\|v_\infty\|_2^2} - 1\right)$$

and

$$w = (R_0 - 1)\sum_{l=1}^{N} \delta_l \left(x_1\right)_l^2,$$

where $x_1 \in \mathbb{R}^N$ denotes the principal eigenvector of the effective infection rate matrix W belonging to the eigenvalue R_0. Then, there exists some constant $\sigma > 0$ such that

$$\frac{\|v(t) - v_{\text{apx}}(t)\|_2}{\|v_\infty\|_2} \leq \sigma(R_0 - 1)^{s-1} \quad \forall t \geq 0, \tag{5}$$

where $s = \min\{p, 2\}$, when the basic reproduction number R_0 approaches 1 from above.

Theorem 1 states a convergence of the viral state $v(t)$ to the approximation $v_{\text{apx}}(t)$ that is uniform in time t. The upper bound (5) on the approximation error is best possible (Prasse and Van Mieghem, 2019a) (up to the constant σ) when $p \leq 2$. Furthermore, both the viral state $v(t)$ and the approximation $v_{\text{apx}}(t)$ converge to the steady-state v_∞, which implies that $v_{\text{apx}}(t) \to v(t)$ when $t \to \infty$. From the definition (3) of the vector $\xi(0)$, it follows that $\|\xi\|_2 \leq \|v(0)\|_2$. Hence, $\|v(0)\|_2 = \mathcal{O}\left((R_0 - 1)^p\right)$ as $R_0 \downarrow 1$ implies $\|\xi(0)\|_2 = \mathcal{O}\left((R_0 - 1)^p\right)$ as $R_0 \downarrow 1$. In real-world epidemics, the total number of individuals per group is large and the number of infected individuals at time $t = 0$ is small. Hence, the initial viral state $v(0)$ is indeed small in practice, and Theorem 1 is applicable to real-world epidemics with a basic reproduction number R_0 that is close to one.

4. NUMERICAL EVALUATION

Figure 1 gives an impression of the accuracy of the approximation $v_{\text{apx}}(t)$ when the initial viral state is set to $v(0) = 0.01v_\infty$, such that the vector $\xi(0) = 0$.

We are interested in the accuracy of the approximation $v_{\text{apx}}(t)$ with respect to the basic reproduction number R_0, and we define the approximation error ϵ_V as

$$\epsilon_V = \frac{1}{N t_{\text{conv}}} \sum_{i=1}^{N} \int_0^{t_{\text{conv}}} \frac{1}{v_{\infty,i}} \left| v_i\left(\tilde{t}\right) - v_{\text{apx},i}\left(\tilde{t}\right) \right| d\tilde{t}.$$

Here, the convergence time t_{conv} is defined as the smallest time t at which

$$|v_i(t_{\text{conv}}) - v_{\infty,i}| \leq 0.01$$

holds for every group i. Thus, at the convergence time t_{conv} the viral state $v(t_{\text{conv}})$ has practically converged to the steady-state v_∞. Figure 2 shows that the approximation error ϵ_V converges quickly to zero when the basic reproduction number R_0 approaches 1 from above.

To study whether the approximation $v_{\text{apx}}(t)$ is accurate for *general* initial viral states $v(0)$, we set the initial viral state $v_i(0)$ for every group i to a uniformly distributed random number in $[0, v_{\infty,i}]$. Figure 3 shows that also for

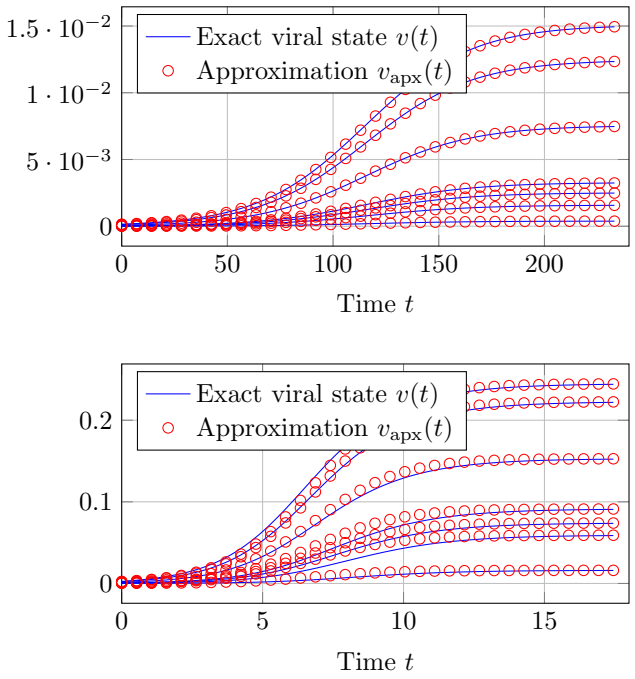

Fig. 1. For a Barabási-Albert random graph with $N = 500$ nodes and heterogeneous spreading parameters β_{ij}, δ_i, the approximation accuracy of Theorem 1 is depicted when the initial viral state equals $v(0) = 0.01v_\infty$. The upper and lower sub-plot shows the viral state traces $v_i(t)$ of seven different nodes i, including the node i with the greatest steady-state $v_{\infty,i}$, for a basic reproduction number of $R_0 = 1.01$ and $R_0 = 1.2$, respectively.

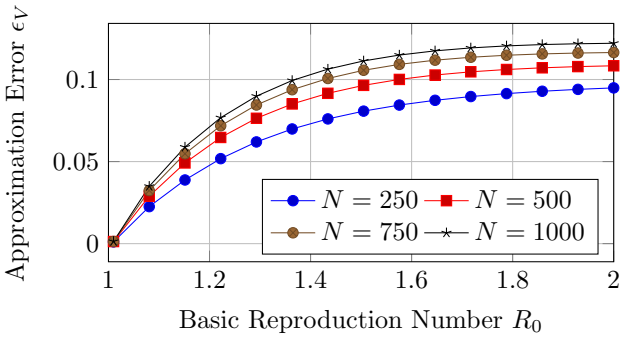

Fig. 2. The approximation error ϵ_V versus the basic reproduction number R_0 for Barabási-Albert random graphs for different network sizes N when the initial viral state equals $v(0) = 0.01v_\infty$.

randomly generated initial viral states $v_i(0) \in [0, v_{\infty,i}]$, the approximation $v_{\text{apx}}(t)$ is accurate. In particular, the exact viral state $v(t)$ seems to converge rapidly to the approximation $v_{\text{apx}}(t)$ as time t evolves.

5. CONCLUSION

We solved the non-linear differential equations of the NIMFA epidemic model around the epidemic threshold $R_0 = 1$ for small initial viral states $v(0)$. Numerical simulations demonstrate that the solution around the epidemic threshold $R_0 = 1$ is accurate – also when the

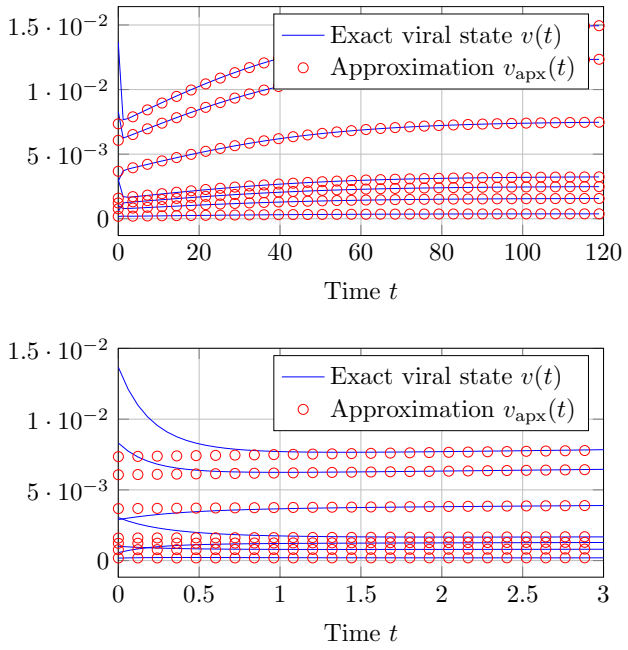

Fig. 3. For a Barabási-Albert random graph with $N = 500$ nodes and heterogeneous spreading parameters β_{ij}, δ_i, a basic reproduction number $R_0 = 1.01$ and a randomly generated initial viral state $v(0)$, the approximation accuracy of Theorem 1 is depicted. The upper and the lower sub-plot show the viral state traces $v_i(t)$ of seven different nodes i until time $t = 120$ and $t = 3$, respectively.

basic reproduction number R_0 is greater than 1 and for general initial viral states $v(0)$. Potential applications of the solution around the threshold, in particular for the stabilisation of the all-healthy viral state $v(t) = 0$, stand on the agenda of future research. Furthermore, Theorem 1 is based on the decomposition (2) with $m = 1$ scalar function $c_1(t)$ and vector y_1. It is an open problem, how to consider $m > 1$ orthogonal functions in (2) to derive an approximation $v_{\text{apx}}(t)$ of the viral state $v(t)$, which potentially is accurate also when $R_0 >> 1$.

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