A Networked Competitive Multi-Virus SIR Model: Analysis and Observability

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Abstract: This paper proposes a novel discrete-time multi-virus SIR (susceptible-infected-recovered) model that captures the spread of competing SIR epidemics over a population network. First, we provide a sufficient condition for the infection level of all the viruses over the networked model to converge to zero in exponential time. Second, we propose an observation model which captures the summation of all the viruses’ infection levels in each node, which represents the individuals who are infected by different viruses but share similar symptoms. We present a sufficient condition for the model to be locally observable. We propose a Luenberger observer for the system state estimation and show via simulations that the estimation error of the Luenberger observer converges to zero before the viruses die out.

Keywords: Biological networks and epidemics dynamics

1. INTRODUCTION

The history of human civilization has been a narrative of undergoing, battling, and outmatching various pandemics (Benedictow and Benedictow, 2004; Johnson and Mueller, 2002). Suffering severe life and economic loss, the research of modeling and monitoring the spread of multiple diseases concurrently has grown significantly through the inspection of each epidemic. In this paper, we investigate the modeling, analysis, and observation of the spread of multi-viruses over population networks.

A considerable amount of effort has been expended on the study of multi-virus models (Paré et al., 2017; Prakash et al., 2012; Sahneh and Scoglio, 2014; Paré et al., 2020b; Santos et al., 2015; Liu et al., 2016; Paré et al., 2021), which focus on the competing susceptible-infected-susceptible (SIS) networked virus model. In this paper our focus is on the competing susceptible-infected-recovered (SIR) epidemic model over a network, as the SIR model can capture the behavior of a diverse set of different epidemics such as: H1N1 (Coburn et al., 2009), Ebola (Berge et al., 2017), and COVID-19 (Chen et al., 2020). The single virus SIR epidemic networked model has been studied extensively, e.g., (Hota et al., 2021; Mei et al., 2017; Paré et al., 2020a). However, to the best of our knowledge, the competing SIR epidemics has not been studied in the literature. Thus, in this work we propose a discrete-time competing SIR virus networked model. The multi-virus model captures the presence and spread of viruses such as influenza and the SARS-CoV-2 virus over a population and could also be utilized to represent different behaviors of variants of the SARS-CoV-2 virus (Lopez Bernal et al., 2021).

Beyond the modeling and analysis of the epidemic models, the epidemic monitoring and infection level estimation have been crucial to the research on contagions. Given that the SARS-CoV-2 pandemic has provided us with an enormous amount of data, how to accurately infer the infection levels of the infectious diseases has become a topic requiring urgent attention (Barmparis and Tsironis, 2020; Meyerowitz-Katz and Merone, 2020). However, the various symptoms caused by diseases such as influenza (Monto et al., 2000) and SARS-CoV-2 (Tostmann et al., 2020) affect the measurement of the cases of different diseases and pose difficulties for the estimation of the states of different epidemics, especially when tests are limited as was witnessed at the beginning of the pandemic and at various peaks of different waves.

In this paper, we propose what we believe to be the first multi-virus model of SIR networked epidemic spreading, along with specifications that ensure the model is well defined. We then provide sufficient conditions for the infection level of each virus to converge to zero in exponential time. Moreover, we explore the system state estimation with an observation model which captures the summation of all cases that exhibit similar signs of illness.

1.1 Notation

We denote the set of real numbers and the set of non-negative integers by \( \mathbb{R} \) and \( \mathbb{Z}_{\geq 0} \), respectively. For any positive integer \( n \), we have \( |n| := \{1, 2, \ldots, n\} \). The spectral radius and an eigenvalue of a matrix \( A \in \mathbb{R}^{n \times n} \) are denoted by \( \rho(A) \) and \( \lambda(A) \), respectively. A diagonal matrix...
Fig. 1. The competing SIR networked model. Each node in the network can only be in one of three states: S, I, R, where \( k \in [m] \).

is denoted by \( \text{diag}(\cdot) \). The transpose of a vector \( x \in \mathbb{R}^n \) is \( x^\top \). The Euclidean norm is denoted by \( \| \cdot \| \). We use \( I \) to denote the identity matrix. We use \( 0 \) to denote the vectors whose entries are all 0, where the dimensions of the vectors are determined by context. Given a matrix \( A \), \( A \) is positive definite, whereas \( \gamma_k \) is the \( k \)-th virus spreading over the network and each individual can be infected by no more than one virus. We denote \( h > 0 \) where \( h \) is the infection rate of the \( k \)-th virus from node \( i \) to node \( j \), and by \( x_k^i \) the healing rate for node \( i \) with respect to virus \( k \). We denote by \( s_i \) and \( r_i \) the susceptible and recovered proportions of subpopulation \( i \), respectively.

We use \( x_k^i[t] \), where \( k \in [m] \), to denote the fraction of individuals infected with virus \( k \) at time instant \( t \). A graphical depiction of this model is given in Figure 1. The discrete-time dynamics of the time-invariant competing virus of SIR networked epidemic model are written as:

\[
\begin{align*}
 s_i[t+1] &= s_i[t] - h \left( s_i[t] \sum_{k=1}^{m} \sum_{j=1}^{n} \beta_{ij}^k x_j^k[t] \right), \quad (1a) \\
x_k^i[t+1] &= x_k^i[t] + h \left( s_i[t] \sum_{j=1}^{n} \beta_{ij}^k x_j^k[t] - \gamma_k x_k^i[t] \right), \quad (1b) \\
r_i[t+1] &= r_i[t] + h \left( \sum_{k=1}^{m} \gamma_k x_k^i[t] \right), \quad (1c)
\end{align*}
\]

where \( h > 0 \) is the sampling parameter, \( t \) is the time index, and \( k \in [m] \) indicates the \( k \)-th virus. Notice that \( s_i[t] + x_1^i[t] + \cdots + x_m^i[t] + r_i[t] = 1 \), capturing the fact that in the competing virus scenario, all the viruses are exclusive: an individual cannot be infected by more than one virus concurrently. We now rewrite (1b) as:

\[
x_k^i[t+1] = x_k^i[t] + h \left( S_i[t] B_k - \Gamma_k \right) x_k^i[t], \quad (2)
\]

where \( S_i[t] = \text{diag}(s_i[t]) \), \( B_k \) is a matrix with \((i,j)\)-th entry \( \beta_{ij}^k \), and \( \Gamma_k[t] = \text{diag}(\gamma_k) \).

We now introduce the following assumptions to ensure that the model in (1) is well-defined.

Assumption 1. For all \( i \in [n] \) and \( k \in [m] \), we have \( s_i[0], x_k^i[0], r_i[0] \in [0, 1] \).

Assumption 2. For all \( i \in [n] \) and \( k \in [m] \), we have \( \beta_{ij}^k \geq 0, \gamma_k^i > 0 \).

Assumption 3. For all \( i \in [n] \) and \( k \in [m] \), we have \( h \sum_{k=1}^{m} \sum_{j=1}^{n} \beta_{ij}^k \leq 1 \) and \( h \sum_{k=1}^{m} \gamma_k^i \leq 1 \).

Remark 4. Assumptions 1 and 2 can be interpreted as the initial proportion of susceptible, infected, and recovered individuals all lying in the interval of \([0, 1]\) and we assume that the healing rates are always positive, which are both reasonable. Assumption 3 ensures the sampling rate is frequent enough for the states of the model to remain well defined.

We next build an observation model which produces the output as the proportion of individuals who show flu-like symptoms from infection of all viruses. The observation model is written as (where we repeat (1b) for convenience):

\[
x_k^i[t+1] = x_k^i[t] + h \left( s_i[t] \sum_{j=1}^{n} \beta_{ij}^k x_j^k[t] - \gamma_k x_k^i[t] \right), \quad (3a) \\
y_i[t] = \sum_{k=1}^{m} c_k^i x_k^i[t], \quad (3b)
\]

where \( c_k^i \) is the measurement coefficient.

Assumption 5. The coefficient \( c_k^i \in (0, 1) \) for all \( i \in [n], k \in [m] \).

Remark 5. The coefficient \( c_k^i \) from Eq. (3b) can capture the probability of showing symptoms from the \( k \)-th virus at subpopulation \( i \). Therefore, \( 1 - c_k^i \) captures the probability of individuals infected with the \( k \)-th virus in subpopulation \( i \) being asymptomatic. The coefficient \( c_k^i \) can also represent how each subpopulation \( i \) defines and measures the cases based on the symptoms of each virus \( k \). For example, the symptoms of influenza can include but are not limited to fever, muscle aches, cough, runny nose, headaches, fatigue, etc.

Remark 7. In the observation model, Eq. (3b) can be interpreted as the summation of all the number of symptomatic patients in each subpopulation, which is an indicator for the decision-makers to be able to judge adequacy of the local hospital capacity and the availability of medical resources against the need.

We then have the following results for the system model under Assumptions 1-3.

Lemma 8. Suppose that \( s_i[0], x_k^i[0], r_i[0] \in [0, 1] \), \( s_i[0] + \sum_{k=1}^{m} x_k^i[0] + r_i[0] = 1 \) for all \( k \in [m], i \in [n] \), and Assumptions 1, 2, and 3 hold. Then, for all \( i \in [n] \) and \( t \in \mathbb{Z}_{\geq 0} \),

(1) \( s_i[t], x_k^i[t], r_i[t] \in [0, 1] \), for all \( k \in [m] \), and \( s_i[t] + \sum_{k=1}^{m} x_k^i[t] + r_i[t] = 1 \), and

(2) \( s_i[t+1] \leq s_i[t] \).

Proof: We prove this result by induction. Base Case: By the assumptions made, \( s_i[0], x_k^i[0], r_i[0] \in [0, 1] \), \( s_i[0] + \sum_{k=1}^{m} x_k^i[0] + r_i[0] = 1 \) for all \( k \in [m], i \in [n] \).

From Assumptions 1-3, we know that \( s_i[0] \geq 0 \) and \( h \left( \sum_{k=1}^{m} \sum_{j=1}^{n} \beta_{ij}^k x_j^k[0] \right) \geq 0 \), hence \( s_i[1] = s_i[0](1 - h \left( \sum_{k=1}^{m} \sum_{j=1}^{n} \beta_{ij}^k x_j^k[0] \right)) \). Since \( -h(s_i[1] \sum_{k=1}^{m} \sum_{j=1}^{n} \beta_{ij}^k x_j^k[0]) \leq \)
Lemma 10. (Ying, 1991) The system in (4) is locally observable at $x[t]$ if and only if the map $x[t] \rightarrow g(x[t]), g(f^{1}(x[t])), \ldots, g(f^{n-1}(x[t]))$ is injective, where $n$ is the dimension of $x[t]$.

3. HEALTHY STATE ANALYSIS

This section presents sufficient conditions that guarantee that each virus $k$ converges to zero exponentially fast, and provides the associated rates of convergence for each virus. We then present the conditions for all the viruses to converge to zero in exponential time. Similar to the standard SIR model, the multi-competitive SIR network model converges to a healthy state regardless of the system parameters and initial conditions; however, it is important to study the exponential convergence as it guarantees that the viruses die out at a faster rate and fewer individuals become infected over the course of the outbreak.

Let

$$M^k := I - hS^k + hB^k,$$

$$\tilde{M}^k[t] := I + h(S^k - S[t]),$$

and note that $\tilde{M}^k$ is the state transition matrix of Eq. (2):

$$\tilde{M}^k[t] = M^k - h(I - S[t])B^k.$$

We first present a sufficient condition, in terms of $M^k$, for the viruses to converge to zero exponentially.

Theorem 15. Under Assumptions 1-3, if $r(M^k) < 1$, then the $k$-th virus of the system in (1) converges to zero in exponential time, and this holds for all $k \in [m]$.

Proof: Consider the candidate Lyapunov function:

$$V^k(t, x^k) = (x^k)^\top \tilde{M}^k x^k.$$  

Since $\tilde{P}^k$ is diagonal and positive definite, $(x^k)^\top \tilde{P}^k x^k > 0$, for all $x^k \neq 0$. Therefore, $V^k(t, x^k) > 0$ for all $k \in [m], t \in \mathbb{Z}_{\geq 0}$, $x^k \neq 0$. Since $\tilde{P}^k$ is positive definite,

$$\lambda_{\min}(\tilde{P}^k)I \leq \tilde{P}^k \leq \lambda_{\max}(\tilde{P}^k)I,$$

which implies that

$$\sigma_1^k \|x^k\|^2 \leq V^k(t, x^k) \leq \sigma_2^k \|x^k\|^2,$$

where $\sigma_1^k = \lambda_{\min}(\tilde{P}^k)$ and $\sigma_2^k = \lambda_{\max}(\tilde{P}^k)$, with $\sigma_1^k, \sigma_2^k > 0$.

Now we turn to computing $\Delta V^k(t, x^k)$. For $x^k \neq 0$ and for all $k \in [m]$, using (2) and (6)-(7), we have

$$\Delta V^k(t, x^k) = (x^k)^\top \tilde{M}^k[t]\top P^k \tilde{M}^k[t] x^k - (x^k)^\top P^k x^k$$

$$= (x^k)^\top [(\tilde{M}^k)^\top P^k \tilde{M}^k - P^k] x^k$$

$$- 2h(x^k)^\top (B^k)^\top I - S[t])P^k \tilde{M}^k x^k + h^2(x^k)^\top (B^k)^\top (I - S[t])P^k (I - S[t])B^k x^k.$$  

Note that the second and third terms of (11) can be reorganized as

$$(x^k)^\top [-2h(B^k)^\top (I - S[t])P^k M^k$$

$$+ h^2(B^k)^\top (I - S[t])P^k (I - S[t])B^k x^k$$

$$(x^k)^\top [h(B^k)^\top (I - S[t])P^k$$

$$- 2M^k + h(I - S[t])B^k) x^k$$

$$= (x^k)^\top (h(B^k)^\top (I - S[t])P^k$$

$$- 2[M^k + h(I - S[t])B^k] x^k$$

where the last equality follows from (6), and the inequality follows from Assumptions 2-3 and Lemma 8. Thus, by plugging (12) into (11), we obtain

$$\Delta V^k(t, x^k) \leq (x^k)^\top [(\tilde{M}^k)^\top P^k \tilde{M}^k - P^k] x^k.$$  

(13)
Since \((M^k)\top P^k M^k - P^k\) is negative definite, we have, from Eq. (13),
\[
\Delta V(t, x^k) \leq -\sigma^k_3 \|x^k\|^2,
\]
where \(\sigma^k_3 = \lambda_{\text{min}}[P^k - (M^k)\top P^k M^k]\), with \(\sigma^k_3 > 0\).

Therefore, from (10) and (14), \(V^k(t, x^k)\) is a Lyapunov function, with an exponential decay, and hence, \(x^k\) converges to zero at an exponential rate.

**Corollary 16.** Under the assumptions of Theorem 15, and with \(P^k\) as defined in the proof of Theorem 1, convergence of \(x^k[t]\) generated by Eq. (2) has an exponential rate of at least \(\sqrt{1 - \frac{\sigma^k_3}{\sigma^k_2}}\), where \(\sigma^k_2 = \lambda_{\text{max}}(P^k)\), \(\sigma^k_3 = \lambda_{\text{min}}[P^k - (M^k)\top P^k M^k]\) for each \(k \in [m]\).

**Proof:** From Lemma 11, (10), and (14), the rate of convergence of virus \(k\) is upper bounded by \(\sqrt{1 - \frac{\sigma^k_3}{\sigma^k_2}}\). We then need to show that the rate is well defined, which is \(\sqrt{1 - \frac{\sigma^k_3}{\sigma^k_2}} \in [0, 1]\). Since \(\sigma^k_2 > 0\) and \(\sigma^k_3 > 0\), it will be sufficient to show that \(\sigma^k_3 \geq \sigma^k_2\).

Since \(P^k\) is positive definite and \((M^k)\top P^k M^k\) is nonnegative definite, we have
\[
\sigma^k_3 I \leq P^k - (M^k)\top P^k M^k \leq P^k \leq \sigma^k_2 I,
\]
from which \(\sigma^k_3 \geq \sigma^k_2\) and the rate of convergence \(\sqrt{1 - \frac{\sigma^k_3}{\sigma^k_2}}\) is well defined.

4. **STATE OBSERVATION MODEL**

In this section, we use the measurement of \(y_i[t]\), the fraction of individuals who show similar symptoms from all viruses, to determine the infection level of each virus. We first construct the observability matrix for the system from Eq. (3b), writing Eq. (3b) as:
\[
y[t] = CX[t],
\]
where \(y[t] = [y_1[t] \ y_2[t] \cdots y_n[t]]\top \in \mathbb{R}^n\) is the measurement, and \(C \in \mathbb{R}^{m \times n}\) is:
\[
C = [C^1 \ C^2 \cdots C^m]
\]
with \(C^k = \text{diag}(c_1^k, c_2^k, \cdots, c_m^k)\) for all \(k \in [m]\), \(X[t] \in \mathbb{R}^{mn\times 1}\) is:
\[
X[t] = \begin{bmatrix} x^1[t] \\ x^2[t] \\ \vdots \\ x^m[t] \end{bmatrix}
\]

Therefore, the measurement \(y[t]\) can be reorganized as:
\[
y[t] = C^1 x^1[t] + C^2 x^2[t] + \cdots + C^m x^m[t].
\]
We can express the measurement at each time step over the time interval \([t, t+m-1]\) as:
\[
\begin{bmatrix} y[t] \\ y[t+1] \\ y[t+2] \\ \vdots \\ y[t+m-1] \end{bmatrix} = \begin{bmatrix} C^1 x^1[t] + \cdots + C^m x^m[t] \\ C^1 x^1[t+1] + \cdots + C^m x^m[t+1] \\ C^1 x^1[t+2] + \cdots + C^m x^m[t+2] \\ \vdots \\ C^1 x^1[t+m-1] + \cdots + C^m x^m[t+m-1] \end{bmatrix}
\]

where
\[
O^k[t] = \begin{bmatrix} C^k \\ C^k M^k[t] \\ \vdots \\ C^k M^m[t] \end{bmatrix} = \begin{bmatrix} C^k M^k[t] M^k[t+1] \\ C^k M^m[t] M^m[t+1] \\ \vdots \\ C^k M^m[t] M^m[t+m-2] \end{bmatrix}
\]
with \(O^k[t] \in \mathbb{R}^{mn\times n}\) for all \(k \in [m]\). We now define the observability matrix of the system in Eq. (3) as:
\[
O[t] = [O^1[t] \ O^2[t] \cdots O^m[t]]
\]
where \(O[t] \in \mathbb{R}^{mn\times mn}\).

We now consider the case when \(s_i[t] = 0, \forall i \in [n]\). Then the observability matrix in Eq. (19) becomes
\[
O_0[t] = [O_0^1[t] \ O_0^2[t] \cdots O_0^m[t]],
\]
where
\[
O_0^k[t] = \begin{bmatrix} C^k \\ C^k (I - h\Gamma^k) \\ \vdots \\ C^k (I - h\Gamma^k)^{m-1} \end{bmatrix}
\]
for all \(k \in [m]\).

**Theorem 17.** Under Assumptions 1-5, if, for each \(i \in [n]\), \(\gamma_i^k\) is a distinct value for all \(k \in [m]\), the competing virus model (3) is locally observable at \(s_i[t] = 0, \forall i \in [n]\).

**Proof:** From the assumptions \(h \sum_{k=1}^m \gamma_i^k \leq 1, h > 0, \) and \(\gamma_i^k > 0\) for all \(i \in [n], k \in [m]\), we obtain that \(1 - h\gamma_i^k > 0\).
for all $i \in [n], k \in [m]$. In addition, since we assume that $c^k_i > 0$ for all $i \in [n], k \in [m]$, we can conclude that the entries of Eq. (20): $c^k_i (1 - h \gamma^k_i) \in (0, 1)$ for all $i \in [n], k \in [m]$.

We let $0^n := [0 \ 0 \cdots 0]^T \in \mathbb{R}^{1 \times n}$ and $0^0 := 0$. Consider Eq. (21) and recall that every matrix in it is diagonal; Hence, Eq. (20) is the concatenation of a set of block diagonal matrices. For all $i \in [n]$, the $i$-th row of the observability matrix (20) can be written as:

$$[0^{i-1} c^1_i \ 0^{i-1} \ 0^{i-1} c^2_i \ 0^{i-1} \cdots \ 0^{i-1} c^m_i \ 0^{i-1}]$$

which is linearly independent with the $(i + b_n)$-th row of (20) for all $l \in [m - 1]$:

$$[0^{l-1} c^1_i (1 - h \gamma^l_i) \ 0^{n-i} \cdots \ 0^{l-1} c^m_i (1 - h \gamma^m_i) \ 0^{n-i}]$$

under our assumption that, for each $i \in [n]$, $\gamma^k_i$ is a distinct value across all $k \in [m]$. Thus, the observability matrix in Eq. (20) has full row rank. Since the observability matrix is a square matrix, we conclude that Eq. (20) is full rank.

Notice that whenever we add another virus to the system model (3), we increase the dimension of (20) from $mn \times mn$ to $(m + 1) n \times (m + 1) n$ by adding $m$ blocks, and the rank of the observability matrix will change from $mn$ to $(m + 1) n$, by the same logic as above. Therefore, by Lemma 14, the competing virus model in (3) is locally observable at $s_i[t] = 0$, $\forall i \in [n]$.

Remark 18. The assumption in Theorem 17, namely that, for each $i \in [n]$, $\gamma^k_i$ is a distinct value across every $k \in [m]$, can be interpreted as each virus having a different recovery rate. This assumption is reasonable as the recovery rate represents the inverse of the average duration of an infected individual being sick, and the average amount of time for an individual to recover from different types/strands of viruses varies drastically (Whitley and Roizman, 2001).

5. SIMULATIONS

In this section, we consider the special case of two viruses: the SARS-CoV-2 virus and influenza spreading over the network depicted in Figure 2. In the network, each node represents a major country in Europe: UK, Turkey, Germany, Spain, and Russia, and the edges represent transportation between two node countries. The system parameters are listed in Table 1 and Table 2. We choose the SARS-CoV-2 virus and influenza because they cause patients to display similar symptoms such as fever, fatigue, headache, etc. It is difficult to distinguish between the two contagions in the early stage of the epidemic without proper testing facilities. This section includes no real data; however, the viral spreading parameters are inspired by the behavior of the viruses (Anderson et al., 2020), namely, the SARS-CoV-2 virus is more contagious than influenza. We also acknowledge that these two viruses are not necessarily competitive; there are cases where people have been infected with both SARS-CoV-2 and influenza (Wu et al., 2020).

The evolution of the infection levels of both viruses are illustrated in Figure 3. We estimate the infection level by using the following proposed Luenberger observer:
\[
\dot{x}_i^k[t + 1] = \dot{x}_i^k[t] + h \left\{ \dot{s}_i[t] \sum_{j=1}^{n} \beta_{ij}^k \dot{x}_j^k[t] - \gamma_{ij}^k \dot{x}_i^k[t] \right\} + L_x(y_i^k[t] - \dot{y}_i[t]). \tag{22}
\]

where \( \dot{s}_i[t] = 1 - \sum_{k=1}^{m} \dot{x}_i^k[t] - \dot{r}_i[t], \) in which the recovered level is estimated through: \( \dot{r}_i[t] = h \sum_{q} \sum_{k=1}^{m} \gamma_{ik}^q \dot{x}_i^q[t] \) at each time step recursively. We first simulate the state estimation in Figure 4 and we can see that the estimation error converges to zero before the viruses die out. Moreover, the magnitude of the estimation error of each virus in each node is less than 10% of its infection proportion respectively. Hence, the Luenberger observer is an adequate system state estimator for our system model (2).

6. CONCLUSION

This paper has investigated the stability and observability of a novel discrete-time networked multi-virus SIR model. We have provided a sufficient condition for each virus to converge to zero exponentially. We have then specified a necessary and sufficient condition for the system to be locally observable at \( s_i[t] = 0, \forall i \in [n]. \) In simulation, we utilized a Luenberger state observer to estimate the system states and the results illustrate that the Luenberger observer is suitable for state estimation of our new model.

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