# Modeling Network Contagion Via Interacting Finite Memory Pólya Urns

Somya Singh, Fady Alajaji and Bahman Gharesifard Department of Mathematics and Statistics Queen's University Kingston, ON K7L 3N6, Canada Email: {17ss262, fa, bahman.gharesifard}@queensu.ca

Abstract—We construct a system of interacting *finite memory* Pólya urns to model contagion spread in a network. The urns, which are composed of red and black balls (representing degrees of infection and healthiness, respectively) interact in the sense that the probability at any time instant of drawing a red ball for a given urn not only depends on that urn's ratio of red balls, but also on the ratio of red balls in the other urns of the network, hence accounting for the effect of spatial contagion. The urns have a finite memory, M, in the sense that reinforcing (black or red) balls added to each urn at time t are only kept in that urn for M future time instants (until time t + M). The resulting vector of all urn drawing variables forms an Mth order time-invariant irreducible and aperiodic Markov chain. We analytically examine the properties of the underlying Markov process and derive its asymptotic behaviour for the case of homogeneous system parameters. We further use mean-field approximation to obtain a class of approximating linear and nonlinear dynamical systems for the non-homogeneous case. Finally, we present simulations to assess the quality of these mean-field approximations.

## I. INTRODUCTION

The ongoing pandemic of SARS-CoV-2 has led to a recent surge in the study of epidemics [1]-[9]. Numerous mathematical models have been proposed in the literature to mimic the dynamics of contagion in a population. Specifically, Pólya urns are widely used to construct such models because the reinforcement scheme for Pólya urns is a preferential attachment scheme [10], [11] which makes it a suitable choice to track spreads of infection [12]-[15]. Most of these models consist of two-color Pólya urns, in which red balls represent a degree of infection and black balls represent a degree of immunity or healthiness for the individuals portrayed by the network urns. Moreover, the use of contagion schemes is not just limited to modeling the dynamics of biological outbreaks. It has also been used to study the behaviour of error bursts in communication channels with memory [16], [17], the spread of innovations or rumors in a social network [18]–[22], opinion dynamics [23]–[25], image segmentation methods [26], the propagation of viruses in computers [27], [28], etc.

In this paper, we develop a model for contagion using an interacting network of *finite memory* Pólya urns. The concept of finite memory Pólya urn was introduced in [16] as a

variation of the classical (infinite memory) Pólya urn [29]-[31]. For a memory M Pólya urn, at each time instant t > M. we remove the balls which were added to the urn at time t-M along with the addition of balls. The step of removing balls ensures that all the added balls remain in the urn for a finite amount (M) of time and hence accounts for latency (e.g., limited duration of infections when adding red balls and of curing (via strengthened immunity or therapeutics) when adding black balls). In this model, the interaction between finite memory Pólya urns (which are analogous to individuals in a population network) is given by a weighted adjacency matrix. This setup ensures that the model replicates spread of infection and the weights in the adjacency matrix can be decided based on factors such as immunity of an individual, spreading rate of the disease, and vicinity between individuals. It is worth pointing our that the interaction between Pólya urns has been examined in several other ways in the literature [14], [32]-[34]. In [14], the Pólya urns interact via super*urns*. A super urn for an urn i consists of all the balls in the neighbouring urns and the urn *i*. Unlike our model, the weight given to urns in the super-urn model in [14] changes with time.

We show that our finite memory interacting Pólya model results in a network-wide time-invariant Markov draw process of memory M that is irreducible and aperiodic (c.f., Lemma 1 and Sec. II.B). Due to the complexity of the Markov chain caused by the fact that its number of states increases exponentially with both M and the number of nodes, it is difficult to solve analytically for its (unique) stationary distribution. We are however able to characterize in closed-form the asymptotic marginal distributions of the draw variables when the system parameters are homogeneous across all urns (see Theorem 1).

Since our model becomes complex in non-homogeneous scenarios (i.e., for differing system parameters), we use *mean-field approximations* (similar to [35]) to predict the asymptotic behaviour of the Markov chain. Mean-field approximation is a variational Bayesian method in which a joint posterior distribution is approximated by the product of individual posterior distributions [36]. Interestingly when M = 1, we obtain an exact linear dynamical system in the (general) non-homogeneous setting without using any approximations (c.f., Theorem 2). We then derive the equilibrium point of this linear dynamical system, hence precisely determining the limiting distribution of each drawing variable (see Theorem 3).

The work of the authors was funded by the Natural Sciences and Engineering Research Council (NSERC) of Canada. The last author wishes to thank the Alexander von Humboldt Foundation for their generous support.

When M > 1, we use the mean-field approximation that for every urn, the most recent M drawing variables are independent of each other (see Theorem 4). Similar mean-field approximations have been used in literature to study spread of diseases in compartmental models [37], [38]. Finally, we emphasize that the model presented in this paper, which shares similarities with susceptible-infection-susceptible (SIS) models [37], [39], is merely motivated by the spread of biological diseases. It can be used in the context of other applications such as the spread of rumors or opinions in a social network. The actual adoption of our model for capturing biological infection spread in a population based on real data (including comparing its effectiveness vis-à-vis established epidemiological models in the literature) is an interesting future direction.

This paper is organised as follows. In Section II, we describe our model and its stochastic properties for the homogeneous and non-homogeneous cases. We also construct the linear and non-linear dynamical systems which give an exact representation of our model when M = 1 and an approximation when M > 1. In Section III, we illustrate the asymptotic behaviour of the underlying network Markov process and the dynamical systems through simulations. We analyse the quality of dynamical system approximations and the changes in asymptotic behaviour with changes in the initial parameters. Finally in Section IV, we conclude the paper and discuss future directions. Due to space limitations, we herein omit all proofs and derivations; full details are available in the longer version of this paper [15].

#### II. MODEL DESCRIPTION AND MAIN RESULTS

We denote our interacting Pólya contagion network with N urns and memory M by IPCN(M, N). Each of the urns in this network contains some red and black balls. We denote the initial number (at time t = 0) of red and black balls in urn i by  $R_i$  and  $B_i$  respectively. We also assume that each urn has at least one red and one black ball initially (i.e.,  $R_i > 0$  and  $B_i > 0$  for all i). In the context of a contagious disease, the initial proportion of black balls in each urn may represent an individual's initial level of immunity, while in the context of opinion dynamics, it may represent an individual's initial belief about the viewpoint portrayed by black balls.

We next define the reinforcement scheme, in the form of *draw variables*,  $Z_{i,t}$ , associated with urn *i* at time  $t \ge 1$ :

$$Z_{i,t} = \begin{cases} 1 & \text{if a red ball is drawn for urn } i \text{ at time } t \\ 0 & \text{if a black ball is drawn for urn } i \text{ at time } t \end{cases}$$

where the process of drawing a ball for urn i is defined in (2) below; this drawing mechanism is applied simultaneously to all urns. If a red ball (respectively, a black ball) is drawn for urn i, we add  $\Delta_{r,i} \ge 0$  red balls (respectively,  $\Delta_{b,i} \ge 0$  balls) to urn i. We assume that  $\Delta_{r,i} + \Delta_{b,i} \ne 0$  for all urns i. The initial total number of balls in urn i is  $T_i = R_i + B_i$ . For every time instance t > M, along with drawing and addition of balls according to the above reinforcement scheme, we remove the balls which were added to urn i at time t - M. Let  $U_{i,t}$  denote the ratio of red balls in urn i at time t. The draw variables for the urns are defined using the interaction matrix:

$$S = \begin{bmatrix} s_{11} & s_{12} & \cdots & s_{1N} \\ s_{21} & s_{22} & \cdots & s_{2N} \\ \vdots & \ddots & \vdots & \vdots \\ s_{N1} & s_{N2} & \cdots & s_{NN} \end{bmatrix}$$
(1)

where S is a row stochastic matrix with non-negative entries, and hence is a weighted adjacency matrix for IPCN(M, N). The draw random variable for urn *i* satisfies:

$$Z_{i,t} = \begin{cases} 1 & \text{w.p.} & \sum_{j=1}^{N} s_{ij} U_{j,t-1} \\ 0 & \text{w.p.} & 1 - \sum_{j=1}^{N} s_{ij} U_{j,t-1} \end{cases}$$
(2)

where "w.p." stands for "with probability." Note that the draw variables of all the urns are conditionally independent, i.e., at every time instant t,

$$P\left(Z_{1,t},\ldots,Z_{N,t}|\{Z_{1,k}\}_{k=1}^{t-1},\ldots,\{Z_{N,k}\}_{k=1}^{t-1}\right)$$
$$=\prod_{i=1}^{N}P\left(Z_{i,t}|\{Z_{1,k}\}_{k=1}^{t-1},\ldots,\{Z_{N,k}\}_{k=1}^{t-1}\right).$$
 (3)

Before analyzing the properties of this model, We normalize the initial and reinforcement parameters as follows:

$$\rho_i := \frac{R_i}{T_i}, \ \delta_{r,i} := \frac{\Delta_{r,i}}{T_i}, \ \delta_{b,i} := \frac{\Delta_{b,i}}{T_i} \text{ for } i = 1, \dots, N.$$

Since our model has a finite memory, every added ball remains in the system for a finite time (which is the memory M of the system). Therefore, for an IPCN(M, N) system, the ratio of red balls in each urn depends on its initial and reinforcement parameters as well as its recent M draws. More precisely,

$$U_{i,t} = \frac{\rho_i + \sum_{n=t-M+1}^{t} \delta_{r,i} Z_{i,n}}{1 + \sum_{n=t-M+1}^{t} (\delta_{r,i} Z_{i,n} + \delta_{b,i} (1 - Z_{i,n}))}.$$
 (4)

We now establish the Markov property for IPCN(M, N). We denote  $Z_t := (Z_{1,t}, Z_{2,t}, \cdots, Z_{N,t})$  to be the vector of all the drawing variables at time t. For t > M, let  $a_t = (a_{1,t}, \cdots, a_{N,t}) \in \{0, 1\}^N$ . Using (2) and (3) we obtain:

$$P[Z_{t+1} = a_{t+1} | Z_t = a_t, \cdots, Z_1 = a_1]$$
  
=  $\prod_{i=1}^N P[Z_{i,t+1} = a_{i,t+1} | Z_t = a_t, \cdots, Z_1 = a_1]$   
=  $\prod_{i=1}^N \left( a_{i,t+1} \sum_{j=1}^N s_{ij} U_{j,t} + (1 - a_{i,t+1})(1 - \sum_{j=1}^N s_{ij} U_{j,t}) \right).$  (5)

Now, using (4) in (5), we have:

$$P[Z_{t+1} = a_{t+1} | Z_t = a_t, \cdots, Z_1 = a_1]$$

$$= \prod_{i=1}^{N} \left( (1 - a_{i,t+1}) + \left( (2a_{i,t+1} - 1) \times \right) \right)$$

$$\left( \sum_{j=1}^{N} \frac{s_{ij} \left( \rho_j + \sum_{n=t-M+1}^{t} \delta_{r,j} a_{j,n} \right)}{1 + \sum_{n=t-M+1}^{t} \left( \delta_{r,j} a_{j,n} + \delta_{b,j} (1 - a_{j,n}) \right)} \right) \right)$$

$$= P[Z_{t+1} = a_{t+1} | Z_t = a_t, \cdots, Z_{t-M+1} = a_{t-M+1}].$$
(6)

Therefore, for an IPCN(M, N) system,  $\{Z_t\}_{t=1}^{\infty}$  is a time-invariant Mth order Markov chain.

In order to simplify the analysis, we construct the Markov process  $W_t = \{Z_t, Z_{t+1}, \cdots, Z_{t+M-1}\}$  for IPCN(M, N) system. Since  $\{Z_t\}_{t=1}^{\infty}$  is an *M*th order Markov chain, the order of  $\{W_t\}_{t=1}^{\infty}$  is 1. We now use (6) to give a general formula for the entries of the transition probability matrix  $Q^{(M,N)}$  of the Markov process  $\{W_t\}_{t=1}^{\infty}$ , which has  $2^{MN}$  states. Note that the transition probability,  $q_{ab}^{(M,N)}$ , of going from state

$$a = ((a_{11}, a_{21}, \cdots, a_{N1}), \cdots, (a_{1M}, a_{2M}, \cdots, a_{NM}))$$

to state

$$b = ((b_{11}, b_{21}, \cdots, b_{N1}), \cdots, (b_{1M}, b_{2M}, \cdots, b_{NM}))$$

in one time step is nonzero if and only if  $a_{ij} = b_{i(j-1)}$  for  $i \in \{1, \dots, N\}$  and  $j \in \{2, \dots, M\}$ , where a, b are binary NM tuples. If  $q_{ab}^{(M,N)}$  is nonzero, it is given by

$$q_{ab}^{(M,N)} := \widetilde{q}_{ab}^{(1)} \widetilde{q}_{ab}^{(2)} \cdots \widetilde{q}_{ab}^{(N)}$$

where

 $\widetilde{q}_{ab}^{(d)} =$ 

$$\begin{cases} 1 - \sum_{i=1}^{N} s_{di} \frac{(\rho_i + \sum_{k=1}^{M} \delta_{r,i} a_{ik})}{\sum\limits_{k=1}^{M} (\delta_{r,i} a_{ik} + \delta_{b,i} (1 - a_{ik}))} & \text{if} \quad b_{dM} = 0 \\ \\ \sum_{i=1}^{N} s_{di} \frac{(\rho_i + \sum\limits_{k=1}^{M} \delta_{r,i} a_{ik})}{\sum\limits_{k=1}^{M} (\delta_{r,i} a_{ik} + \delta_{b,i} (1 - a_{ik}))} & \text{if} \quad b_{dM} = 1, \end{cases}$$

$$(7)$$

with  $d \in \{1, \dots, N\}$ . We next investigate the asymptotic behaviour of the drawing variables for different cases.

## A. Homogeneous case

In the homogeneous case, we set  $R_i = R$  and  $T_i = T$  and  $\Delta_{r,i} = \Delta_{b,i} = \Delta$  for all *i*; hence, we have  $\rho_i = \rho := R/T$  and  $\delta_{r,i} = \delta_{b,i} = \delta := \Delta/T$  for all *i*. From a contagion perspective, having identical initial and reinforcement parameters for all urns ensures similar immunity levels and infection/curing rates. However, the interaction parameters, given by (1), are different for each urn even in the homoge-

neous case; therefore the susceptibility of individuals is still different. We first examine the stochastic properties of the Markov process  $\{Z_t\}_{t=1}^{\infty}$ . We obtain that (6) simplifies to

$$P[Z_{t+1} = a_{t+1} | Z_t = a_t, \cdots, Z_1 = a_1]$$
  
= 
$$\prod_{i=1}^N \left( (2a_{i,t+1} - 1) \sum_{j=1}^N \frac{s_{ij}(\rho + \delta \sum_{k=t-M+1}^t a_{j,k})}{1 + \delta M} + (1 - a_{i,t+1}) \right)$$
  
= 
$$P[Z_{t+1} = a_{t+1} | Z_t = a_t, \cdots, Z_{t-M+1} = a_{t-M+1}].$$
 (8)

The above simplification yields the following result.

**Lemma 1.** For the homogeneous IPCN(M, N), the transition probability matrix  $Q^{(M,N)}$  is irreducible and aperiodic.

It is difficult to derive a closed form for the stationary distribution of the Markov process  $\{W_t\}_{t=1}^{\infty}$ . However, we can determine its asymptotic marginals as follows.

**Theorem 1.** For a homogeneous IPCN(M, N) system

$$\lim_{t \to \infty} P(Z_{i,t} = 1) = \rho \tag{9}$$

for all urns i in the network.

#### B. Non-Homogeneous Case

The Markov process  $\{W_t\}_{t=1}^{\infty}$  remains irreducible and aperiodic for the non-homogeneous case (the proof is similar to that of Lemma 1). However, it is much harder to obtain the analogous result to Theorem 1 when the initial parameters differ across the urns. This is why we resort to constructing dynamical systems for  $P_i(t) := P(Z_{i,t} = 1)$ . Interestingly for the IPCN(1, N) network, we derive an exact linear dynamical system, as follows:

$$P(Z_{i,t} = 1 | Z_{1,t-1}, Z_{2,t-1}, \cdots, Z_{N,t-1})$$

$$= \sum_{j=1}^{N} \frac{s_{ij}(\rho_j + \delta_{r,j}Z_{j,t-1})}{1 + \delta_{r,j}Z_{j,t-1} + (1 - Z_{j,t-1})\delta_{b,j}}$$
(10)
$$= \sum_{j=1}^{N} [s_{ij}\beta_1^{(j)}(1)Z_{j,t-1} + s_{ij}\beta_1^{(j)}(0)(1 - Z_{j,t-1})]$$

where

$$\beta_1^{(j)}(k) := \frac{\rho_j + k\delta_{r,j}}{1 + k\delta_{r,j} + (1 - k)\delta_{b,j}}$$

for  $j \in \{1, \dots, N\}, k \in \{0, 1\}$ . Now taking expectation with respect to  $(Z_{1,t-1}, \dots, Z_{N,t-1})$  on both sides of (10) yields

$$P_i(t) = \sum_{j=1}^{N} [\beta_1^{(j)}(1)s_{ij}P_j(t-1) + s_{ij}\beta_1^{(j)}(0)(1-P_j(t-1))]$$
(11)

To this end, defining the column vector P(t) as

$$P(t) = (P_1(t), P_2(t), \cdots, P_N(t))^{\mathbf{T}}$$

where <sup>T</sup> denotes transposition, we obtain the following dynamical system for the IPCN(1, N) network.

**Theorem 2.** For the IPCN(1, N) system, the infection vector satisfies the equation

$$P(t) = J_{N,1}P(t-1) + C_{N,1}$$
(12)

where  $J_{N,1} \in \mathbb{R}^{N \times N}$ ,  $C_{N,1} \in \mathbb{R}^{N \times 1}$  are matrices with respective entries:

$$[J_{N,1}]_{i \times j} = \frac{s_{ij}(\rho_j + \delta_{r,j})}{(1 + \delta_{r,j})} - \frac{s_{ij}\rho_j}{(1 + \delta_{b,j})}$$
$$= s_{ij}(\beta_1^{(j)}(1) - \beta_1^{(j)}(0))$$
and 
$$[C_{N,1}]_{1 \times i} = \sum_{j=1}^N \frac{s_{ij}\rho_j}{(1 + \delta_{b,j})} = \sum_{j=1}^N s_{ij}\beta_1^{(j)}(0).$$

For the IPCN(1, N) system, we next determine the limit of  $P_i(t)$  as time t grows without bound for each i (this result is an extension of Theorem 1 from the homogeneous to the non-homogeneous case).

**Theorem 3.** The linear dynamical system for the IPCN(1, N) system given by (12) has a unique equilibrium point given by  $P^* = (I - J_{N,1})^{-1}C_{N,1}$  and

$$\lim_{t \to \infty} P_i(t) = P_i^*$$

for all  $i \in \{1, ..., N\}$ .

For IPCN(M, N) networks with M > 1, we next use mean-field approximation to construct approximate non linear dynamical systems. Specifically, we make the following simplifying assumption:

 We assume that for every time instant t > M, for each urn i, Z<sub>i,t-1</sub>, ··· , Z<sub>i,t-M</sub> are approximately independent of each other; i.e., at any given time instant t > M, we assume that for all j ∈ {1, 2, ··· , N},

$$P[Z_{j,t-1}, Z_{j,t-2}, \cdots, Z_{j,t-M}] \approx \prod_{k=1}^{M} P[Z_{j,t-k}].$$
 (13)

Under the above assumption, the following theorem gives the approximating dynamical systems for IPCN(M, N) systems with M > 1.

**Theorem 4.** For the IPCN(M, N) system, the approximating dynamical system is given by:

$$P_{i}(t) \approx \sum_{j=1}^{N} s_{ij} \beta_{M}^{(j)}(0) + \sum_{\substack{j=1 \ n=1}}^{N} \sum_{n=1}^{M} \left[ \left( \sum_{k=0}^{n} \left( (-1)^{n-k} \binom{n}{k} s_{ij} \beta_{M}^{(j)}(k) \right) \right) \times \left( \sum_{\substack{(d_{1}, \cdots, d_{n}) \\ \in H_{n,M}}} P_{j}(t-d_{1}) \cdots P_{j}(t-d_{n}) \right) \right], (14)$$

where

$$H_{n,M} := \{ (d_1, \cdots, d_n) \mid d_i \in \{1, \cdots, M\}, \\ d_i \neq d_j \; \forall i, j \in \{1, \cdots, n\} \}.$$

**Example:** To illustrate (14), consider a simple IPCN(2,3) system depicted by the graph

with interaction matrix given by:

 $\begin{bmatrix} s_{11} & 1 - s_{11} & 0\\ s_{21} & 1 - s_{21} & 0\\ s_{13} & 0 & 1 - s_{13} \end{bmatrix}.$ 

Using (14), the approximate expression of  $P_1(t)$  is explicitly given by:

$$P_{1}(t) \approx s_{11}\beta_{2}^{(1)}(0) + (1 - s_{11})\beta_{2}^{(2)}(0) + s_{11}(\beta_{2}^{(1)}(1) - \beta_{2}^{(1)}(0))(P_{1}(t - 1) + P_{1}(t - 2)) + (1 - s_{11})(\beta_{2}^{(2)}(1) - \beta_{2}^{(2)}(0))(P_{2}(t - 1) + P_{2}(t - 2)) + s_{11}(\beta_{2}^{(1)}(2) - 2\beta_{2}^{(1)}(1) + \beta_{2}^{(1)}(0))P_{1}(t - 1)P_{1}(t - 2) + (1 - s_{11})(\beta_{2}^{(2)}(2) - 2\beta_{2}^{(2)}(1) + \beta_{2}^{(2)}(0))P_{2}(t - 1)P_{2}(t - 2).$$

**Observation:** Note that the dynamical system derived in Theorem 4 is linear and exact for M = 1 (as it reduces to the system derived in Theorem 2) and is a non-linear approximation for M > 1. Since the analysis of non-linear dynamical systems is much harder, it might be useful to drop the non-linear terms in (14) and analyse the linear part of the system which is given by:

$$P_{i}(t) \approx \sum_{j=1}^{N} s_{ij} \beta_{M}^{(j)}(0) + \sum_{j=1}^{N} \sum_{k=1}^{M} s_{ij} \left( \beta_{M}^{(j)}(1) - \beta_{M}^{(j)}(0) \right) P_{j}(t-k).$$
(15)

In the next section, we demonstrate the quality of these constructed linear and non-linear dynamical systems using simulation results.

## III. SIMULATIONS

We provide two typical scenarios of simulations, each presenting the empirical sum of the corresponding Markov process and the linear and non-linear approximations for M = 1, 2, 3. In the first scenario, the  $\delta_r$  values are chosen to be much larger than the  $\delta_b$  values; this scenario models contagion spread situations where the infection rate is much higher than the curing rate. In contrast, in the second scenario, we choose  $\delta_b$  values much larger than the  $\delta_r$  values.

The empirical sum for the Markov process is computed as follows. For an IPCN(M, N) system, we plot the average empirical sum at time t:  $\frac{1}{N}\sum_{i=1}^{N} I_t(i)$ , where  $I_t(i) = \frac{1}{t}\sum_{n=1}^{t} Z_{i,n}$ . For each plot, the average empirical sum is computed 100 times and the mean value is plotted against time. For the dynamical system simulations, we plot  $\frac{1}{N}\sum_{i=1}^{N} P_i(t)$ , which we refer to as the (average) infection rate.

In both scenarios, we observe that when M = 1, the average empirical sum and the linear dynamical system achieve

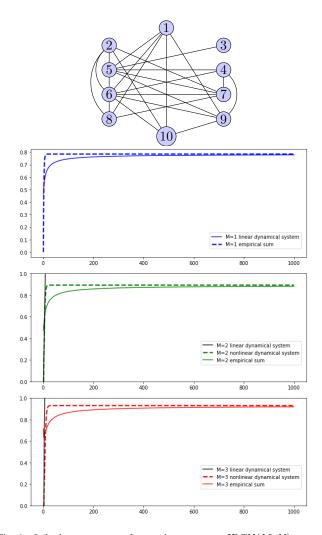


Fig. 1. Infection rate curves for non-homogeneous IPCN(M, N) systems with N = 10 nodes and memory M = 1, 2, 3. At time t = 0, each urn has a total of 25 balls. The number of red balls in each urn at t = 0 is chosen randomly between range 5 to 23 so that  $\rho's$  lie in the range 0.2 to 0.92.  $\Delta'_r s$  are chosen randomly between range 60 to 70 and  $\Delta'_b s$  are randomly chosen between range 20 to 29. For simplicity, we set the initial values  $P_i(0), P_i(1), \dots, P_i(M-1)$  all equal to zero for all urns *i*.

the same asymptotic value given by  $P_i^*$  in Theorem 3 (as expected since the linear dynamical system exactly represents the IPCN(1, N) network).

When M = 2, 3, for the first scenario (Fig. 1), we observe that the corresponding non-linear approximations (given by (14)) perform quite well, while the linear approximations (of (15)) do not approximate the asymptotic behaviour of the system. The failure of the linear dynamical systems in this case is attributed to the higher  $\delta_r$  values. The coefficients in (15) become so large that  $P(Z_{i,t} = 1)$  becomes greater than 1, making the linear dynamical system irrelevant. In the second scenario (Fig. 2), both linear and non-linear dynamical systems perform well. More simulations are provided in [15].

## IV. CONCLUSIONS AND FUTURE DIRECTIONS

We introduced a memory-M Pólya urn based interacting network to model epidemic spread in a population. We studied the stochastic properties of the underlying Mth order Markov process, determined its limiting marginal distributions when M = 1 and constructed dynamical system approximations for M > 1. Our simulation results provide clear evidence that the approximating dynamical systems perform well. Future work includes analysing the asymptotic behaviour of the approximating non-linear dynamical system. Another direction is the development of judicious intervention strategies under limited resources that mitigate network infection propagation.

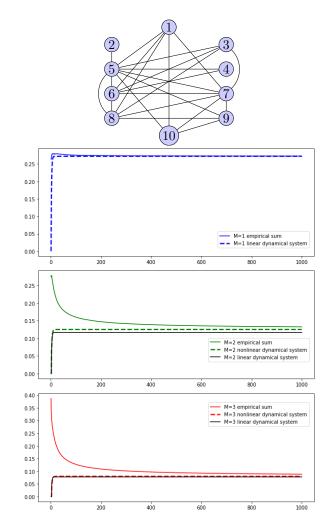


Fig. 2. Infection rate curves for non-homogeneous IPCN(M, N) systems with N = 10 nodes and memory M = 1, 2, 3. At time t = 0, the total number of balls in each urn is 25. The number of red balls in each urn at time t = 0 are chosen randomly between the range 2 to 17 so that  $\rho's$  lie in the range 0.08 to 0.68.  $\Delta'_r s$  are chosen randomly in the range 12 to 30.  $\Delta'_b s$  are chosen in the range 61 to 80. For simplicity, we set the initial values  $P_i(0), P_i(1), \dots, P_i(M-1)$  all equal to zero for all urns *i*.

### REFERENCES

- A. R. Hota and S. Sundaram, "Game-theoretic vaccination against networked SIS epidemics and impacts of human decision-making," *IEEE Transactions on Control of Network Systems*, vol. 6, no. 4, pp. 1461–1472, 2019.
- [2] P. E. Paré, C. L. Beck, and T. Başar, "Modeling, estimation, and analysis of epidemics over networks: An overview," *Annual Reviews in Control*, vol. 50, pp. 345–360, 2020.
- [3] A. Zeb, E. Alzahrani, V. S. Erturk, and G. Zaman, "Mathematical model for coronavirus disease 2019 (COVID-19) containing isolation class," *BioMed Research International*, 2020.
- [4] B. Ivorra, M. Ferrández, M. Vela-Péroz, and A. Ramos, "Mathematical modeling of the spread of the coronavirus disease 2019 (COVID-19) taking into account the undetected infections. the case of China," *Commun. Nonlinear Science & Numerical Simulation vol.* 88, 2020.
- [5] N. R. Barraza, G. Pena, and V. Moreno, "A non-homogeneous Markov early epidemic growth dynamics model. application to the SARS-CoV-2 pandemic," *Chaos, Solitons & Fractals*, p. 110297, 2020.
- [6] A. B. Gumel, E. A. Iboi, C. N. Ngonghala, and E. H. Elbasha, "A primer on using mathematics to understand COVID-19 dynamics: Modeling, analysis and simulations," *Infectious Disease Modelling*, vol. 6, pp. 148–168, 2021.
- [7] M. Hayhoe, F. Barreras, and V. M. Preciado, "Multitask learning and nonlinear optimal control of the COVID-19 outbreak: A geometric programming approach," *Annual Reviews in Control*, vol. 52, pp. 495– 507, 2021.
- [8] J. M. Read, J. R. Bridgen, D. A. Cummings, A. Ho, and C. P. Jewell, "Novel coronavirus 2019-ncov (COVID-19): Early estimation of epidemiological parameters and epidemic size estimates," *Philosophical Trans. Royal Society B*, vol. 376, no. 1829, p. 20200265, 2021.
- [9] M. T. Sofonea, B. Reyné, B. Elie, R. Djidjou-Demasse, C. Selinger, Y. Michalakis, and S. Alizon, "Memory is key in capturing COVID-19 epidemiological dynamics," *Epidemics*, vol. 35, p. 100459, 2021.
- [10] N. Berger, C. Borgs, J. T. Chayes, and A. Saberi, "Asymptotic behavior and distributional limits of preferential attachment graphs," *The Annals* of *Probability*, vol. 42, no. 1, pp. 1–40, 01 2014.
- [11] A. Collevecchio, C. Cotar, and M. LiCalzi, "On a preferential attachment and generalized Pólya's urn model," *The Annals of Applied Probability*, vol. 23, no. 3, pp. 1219–1253, 2013.
- [12] G. Harrington, F. Alajaji, and B. Gharesifard, "Initialization and curing policies for Pólya contagion networks," *SIAM Journal on Control and Optimization*, to appear, 2022.
- [13] M. Hayhoe, F. Alajaji, and B. Gharesifard, "Curing epidemics on networks using a Polya contagion model," *IEEE/ACM Transactions on Networking*, vol. 27, no. 5, pp. 2085–2097, 2019.
- [14] —, "A Polya contagion model for networks," *IEEE Transactions on Control of Network Systems*, vol. 5, no. 4, p. 1998–2010, 12 2017.
- [15] S. Singh, F. Alajaji, and B. Gharesifard, "A finite memory interacting Pólya contagion network and its approximating dynamical systems," *SIAM Journal on Control and Optimization*, to appear, 2022 (see also arXiv preprint arXiv:2010.00463v3, Dec. 2021).
- [16] F. Alajaji and T. Fuja, "A communication channel modeled on contagion," *IEEE Transactions on Information Theory*, vol. 40, no. 6, pp. 2035–2041, 1994.
- [17] L. Zhong, F. Alajaji, and G. Takahara, "A binary communication channel with memory based on a finite queue," *IEEE Transactions on Information Theory*, vol. 53, no. 8, pp. 2815–2840, 2007.
- [18] E. M. Rogers, Diffusion of Innovations. Simon and Schuster, 2010.

- [19] D. Kempe, J. Kleinberg, and É. Tardos, "Maximizing the spread of influence through a social network," in *Proceedings of the Ninth ACM* SIGKDD International Conference on Knowledge Discovery and Data Mining, 2003, pp. 137–146.
- [20] D. Shah and T. Zaman, "Rumors in a network: Who's the culprit?" *IEEE Transactions on Information Theory*, vol. 57, no. 8, pp. 5163– 5181, 2011.
- [21] W. Dong, C. Tan, and W. Zhang, "Rooting out the rumor culprit from suspects," in *Proceedings of the IEEE Symposium on Information Theory*, 2013, pp. 2671–2675.
- [22] P.-D. Yu, C. W. Tan, and H.-L. Fu, "Rumor source detection in unicyclic graphs," in *Proceedings of the IEEE Information Theory Workshop*, 2017, pp. 439–443.
- [23] B. Skyrms and R. Pemantle, "A dynamic model of social network formation," *Proceedings of the National Academy of Sciences*, vol. 97, no. 16, pp. 9340–9346, 2000.
- [24] E. Adar and L. A. Adamic, "Tracking information epidemics in blogspace," in *Proceedings of the IEEE/WIC/ACM International Conference on Web Intelligence (WI'05)*, 2005, pp. 207–214.
- [25] A. Jadbabaie, A. Makur, E. Mossel, and R. Salhab, "Opinion dynamics under social pressure," arXiv preprint arXiv:2104.11172v1, 2021.
- [26] A. Banerjee, P. Burlina, and F. Alajaji, "Image segmentation and labeling using the Pólya urn model," *IEEE Transactions on Image Processing*, vol. 8, no. 9, pp. 1243–1253, 1999.
- [27] M. Garetto, W. Gong, and D. Towsley, "Modeling malware spreading dynamics," in *Proceedings of IEEE INFOCOM, 22nd Annual Joint Conference of the IEEE Comput. and Commun. Societies*, vol. 3, 2003, pp. 1869–1879.
- [28] N. Berger, C. Borgs, J. Chayes, and A. Saberi, "On the spread of viruses on the internet," in *Proceedings of the ACM-SIAM Symposium* on Discrete Algorithms, vol. 5, 2005, pp. 301–310.
- [29] F. Eggenberger and G. Pólya, "Über die statistik verketteter vorgänge," ZAMM-Journal of Applied Mathematics and Mechanics/Zeitschrift für Angewandte Mathematik und Mechanik, vol. 3, no. 4, pp. 279–289, 1923.
- [30] —, "Sur l'interprétation de certaines courbes de fréquence," CR Acad. Sci. Paris, vol. 187, pp. 870–872, 1928.
- [31] G. Pólya, "Sur quelques points de la théorie des probabilités," in Annales de l'institut Henri Poincaré, vol. 1, no. 2, 1930, pp. 117–161.
- [32] M. Benaïm, I. Benjamini, J. Chen, and Y. Lima, "A generalized Pólya's urn with graph based interactions," *Random Structures & Algorithms*, vol. 46, no. 4, pp. 614–634, 2015.
- [33] G. Aletti, I. Crimaldi, and A. Ghiglietti, "Synchronization of reinforced stochastic processes with a network based interaction," *The Annals of Applied Probability*, vol. 27, no. 6, pp. 3787–3844, 2017.
  [34] P. Pra, P. Y. Louis, and I. G. Minelli, "Synchronazation via interacting
- [34] P. Pra, P. Y. Louis, and I. G. Minelli, "Synchronazation via interacting reinforcement," *Journal of Applied Probability*, vol. 51, pp. 556–568, 03 2016.
- [35] P. Van Mieghem, J. Omic, and R. Kooij, "Virus spread in networks," *IEEE/ACM Transactions on Networking*, vol. 17, no. 1, pp. 1–14, 2009.
- [36] D. J. C. MacKay, Information Theory, Inference & Learning Algorithms. USA: Cambridge University Press, 2002.
- [37] N. A. Ruhi, T. Christos, and B. Hassibi, "Improved bounds on the epidemic threshold of exact SIS models on complex networks," *Proceedings of the IEEE Conference on Decision and Control (CDC)*, pp. 3560–3565, 2016.
- [38] 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.
- [39] D. Easley and J. Kleinberg, Networks, Crowds, and Markets: Reasoning about a Highly Connected World. Cambridge University Press, 2010.