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A Branching Process Approximation of the Final Size of a Multitype Collective Reed-Frost Model

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Abstract: We consider the asymptotic behavior of the final size of a multitype collective Reed-Frost process. This type of models was introduced by [9] and include most known epidemic models of the type SIR (Susceptible, Infected, Removed) as special cases. Under certain conditions, we show that, when the initial number of susceptible is very large and the initial number of infected individuals is finite, the infection process behaves as a multitype Galton-Watson process. This fact is proved using a simple argument based on Bernstein polynomials. We use this result to study the final size of the epidemic.

Keywords: Collective Reed-Frost epidemic, Final size, Galton-Watson process, Bernstein polynomials.

1 Introduction

There is a rich literature concerning the description and analysis of the spread of contagious diseases using mathematical models. One important class of such models is the type referred to as SIR (Susceptible, Infected, Removed) [6]. In these models the fundamental question of interest concerns the final size of the epidemic, i.e. the number of individuals to ever contract the disease. Prior to 1975, two models were used to study the final size: the so-called general epidemic model and the Reed-Frost model [2]. Later on much effort was deployed in order to generalize these models. [9] introduced the general collective Reed-Frost model. As will be seen, this model generalizes most epidemic models proposed in the literature. In the case of a homogeneous population, the Picard-Lefèvre model uses the following hypotheses:

a) At time t, $t \in IN$, let S_t and I_t denote the numbers of susceptible and infected individuals, respectively, and let the vector (S_t, I_t) represents the state of the population. Let further $(S_0, I_0) = (n, m)$ describes the initial conditions. Then we have

$$S_t = S_{t+1} + I_{t+1}, \quad t \in IN \tag{1}$$

b) $(S_t, I_t,\ t \in IN)$ is a Markov chain with transitions governed by the following rule: consider, among the n initial susceptibles, any possible subset of size k, $k \in [0, n]$. Then, all the infectives of every generation behave independently. Moreover, each of them fails to transmit infection within such a subset of susceptibles actually present, with the (known) probability $q(k, n)$ which depends only on the sizes k and n.

Using a certain family of martingales combined with properties of the Gontcharoff polynomials, [9] could determine the exact distribution of the final size. The asymptotic result in the same direction was obtained by[7]
Of course, the assumption that the underlying population is homogeneous is not very realistic. In the case of AIDS modelling for example, it is possible [5] to divide the total population into a number of subgroups (according to sex, sexual orientation, drug abuse status...) with different risk factors. To take such heterogeneities into account, [9] have described generalizations of the above cited models. Accordingly, the total population can be partitioned into J homogeneous groups differing from each other such that the infection within each subgroup is transmitted according to the laws of a model of

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the collective type. For the resulting model, it is possible to apply the techniques used in the homogeneous case to describe the distribution of the final size of the epidemic as the initial number of susceptibles grows while the initial number of infectives is kept finite.

2 Collective Reed-Frost epidemic model with several populations

Consider a closed population divided into J distinct homogeneous groups. Suppose that the infection is transmitted within each group according to the following rules.

a) The propagation of the disease is described through successive generations of infectives. At each time t, $t \in IN$, the state of the population is given by $(S_t^{(j)}, I_t^{(j)}, j = 1,\ldots,J)$, where $S_t^{(j)}$ and $I_t^{(j)}$ denote, respectively, the numbers of susceptibles and infecteves in group j, $j = 1,\ldots,J$, at time t. Initially, $(S_0^{(j)}, I_0^{(j)}) = (n_j, m_j)$ which implies that

$$S_t^{(j)} = S_{t+1}^{(j)} + I_{t+1}^{(j)}, \quad j = 1,\ldots,J \text{ and } t \in IN \ . \tag{2}$$

b) $\{(S_t^{(j)}, I_t^{(j)}, \ \ j = 1,\ldots,J); \ \ t \in IN\}$ is a Markov chain with transitions governed by the following rule. Consider any possible subset in J initial classes of susceptibles of sizes $k_1 \in [0, n_1], \ldots, k_J \in [0, n_J]$ with $k_1 + \ldots + k_J \geq 1$. Then all the infectives of every generation behave independently. Moreover, each of those in group j, $j = 1,\ldots,J$ fail to transmit infection within these (at most J) subsets of susceptibles actually present, with the (known) probability $q^{(j)}(k_1,\ldots,k_J)$ which depends only on the current group j and the sizes $k_1, k_2, \ldots, k_J$ and $n_1,\ldots,n_J$.

We observe that the probabilities $q^{(j)}(k_1,\ldots,k_J)$ are evaluated independently of the susceptibles outside the subset. In addition, these probabilities allow us to determine the conditional distribution of survivals by generation. A direct use of formula (3.5) in [8], gives

$$P[S_{t+1}^{(j)} = s_j, j = 1,\ldots,J / (S_t^{(j)}, I_t^{(j)}, \ \ j = 1,\ldots,J)]$$

$$= \sum_{k_1=s_1}^{S_t^{(1)}} \ldots \sum_{k_J=s_J}^{S_t^{(J)}} \prod_{j=1}^{J} \left(C_{S_t^{(j)}}^{k_j} C_{k_j}^{s_j} (-1)^{k_j - s_j} [q^{(j)}(k_1,\ldots,k_J)]^{I_t^{(j)}} \right), \tag{3}$$

for $s_1 \in [0, S_t^{(1)}], \ldots, s_J \in [0, S_t^{(J)}]$ and $t \in itIN$.
Moreover, the process $\left(S_t^{(j)}, I_t^{(j)}, j = 1,\ldots,J, \ \ t \in IN\right)$ is terminated at the moment

$$K = inf\{t / I_t^{(1)} = I_t^{(2)} = \ldots = I_t^{(J)} = 0\}. \tag{4}$$

$S_K^{(j)}$ is thus the ultimate number of susceptibles in group j, $j = 1,\ldots,J$, which have avoided contact with all infected and $T^{(j)} = n^{(j)} - S_K^{(j)}, j = 1,\ldots,J$, denotes the final size of the epidemic in group j. The following notation will be needed in the sequel

$$\begin{aligned} M_j &= \begin{cases} n_j, \ j = 1,\ldots,J \\ \quad \text{or} \\ n = n_1 + n_2 + \ldots + n_J, \end{cases} \\ \pi_n^{(j)} &= \tfrac{n_j}{M_j}, \ \ j = 1,\ldots,J, \\ \mathbf{T}_n &= (T^{(1)},\ldots,T^{(J)}). \end{aligned} \tag{5}$$

3 A branching process approximation

In order to approximate the epidemical process by branching process, we need the following assumptions :

(i) $m = m_1 + \ldots + m_J$ is finite and $\pi_n^{(j)} \longrightarrow \pi_j$, whenever $n_j \longrightarrow +\infty$, $j = 1,\ldots,J$.
(ii) There exists a continuous function $\hat{g}$ from $[0,1]^J$ in $[0,1]^J$ such that
$|q^{(j)}(k_1,\ldots,k_J) - \hat{g}^{(j)}(1 - \frac{k_1}{M_1},\ldots,1 - \frac{k_J}{M_J})| \longrightarrow 0$, whenever $n_j \longrightarrow +\infty$, $j = 1,\ldots,J$, uniformly in $k_1,\ldots,k_J$.

Let $\mathbf{I}_t=(I_t^{(1)},\ldots,I_t^{(J)})$, $\mathbf{S}_t=(S_t^{(1)},\ldots,S_t^{(J)})$, $\mathbf{z}=(z_1,\ldots,z_J)$, and denote by $g_{\mathbf{S}_{t+1}}(\mathbf{z}/\mathbf{S}_t,\mathbf{I}_t)$ (respectively $f_{\mathbf{I}_{t+1}}(\mathbf{z}/\mathbf{S}_t,\mathbf{I}_t)$), $t=0,1,\ldots$, the conditional generating function $\mathbf{S}_{t+1}$ (respectively of $\mathbf{I}_{t+1}$). Using the formula (3.3) in [8] we obtain

$$\begin{aligned} g_{\mathbf{S}_{t+1}}(\mathbf{z}/\mathbf{S}_t,\mathbf{I}_t) &= E(\prod_{j=1}^{J} z_j^{S_{t+1}^{(j)}}/\mathbf{S}_t,\mathbf{I}_t) \\ &= \sum_{k_1=0}^{S_t^{(1)}}\cdots\sum_{k_J=0}^{S_t^{(J)}}\prod_{j=1}^{J} C_{S_t^{(j)}}^{k_j}(z_j-1)^{k_j}[q^{(j)}(k_1,\ldots,k_J)]^{I_t^{(j)}}. \end{aligned} \tag{6}$$

Since $I_{t+1}^{(j)}=S_t^{(j)}-S_{t+1}^{(j)}$, $j=1,\ldots,J$ and $t\in itIN$. We have

$$\begin{aligned} f_{\mathbf{I}_{t+1}}(\mathbf{z}/\mathbf{S}_t,\mathbf{I}_t) &= E(\prod_{j=1}^{J} z_j^{I_{t+1}^{(j)}}/\mathbf{S}_t,\mathbf{I}_t) \\ &= E(\prod_{j=1}^{J} z_j^{S_t^{(j)}-S_{t+1}^{(j)}}/\mathbf{S}_t,\mathbf{I}_t) \\ &= \prod_{j=1}^{J} z_j^{S_t^{(j)}} E(\prod_{j=1}^{J}(\frac{1}{z_j})^{S_{t+1}^{(j)}}/\mathbf{S}_t,\mathbf{I}_t) \\ &= \prod_{j=1}^{J} z_j^{S_t^{(j)}} g_{\mathbf{S}_{t+1}}(\frac{1}{z_1},\ldots,\frac{1}{z_J}/\mathbf{S}_t,\mathbf{I}_t) \\ &= \prod_{j=1}^{J} z_j^{S_t^{(j)}} \sum_{k_1=0}^{S_t^{(1)}}\cdots\sum_{k_J=0}^{S_t^{(J)}}\prod_{j=1}^{J}\left(C_{S_t^{(j)}}^{k_j}(\frac{1}{z_j}-1)^{k_j}[q^{(j)}(k_1,\ldots,k_J)]^{I_l^{(j)}}\right) \\ &= \sum_{k_1=0}^{S_t^{(1)}}\cdots\sum_{k_J=0}^{S_t^{(J)}}\prod_{j=1}^{J}\left(C_{S_t^{(j)}}^{k_j}(1-z_j)^{k_j} z_j^{S_t^{(j)}-k_j}\right. \\ &\qquad \left.\times[q^{(j)}(k_1,\ldots,k_J)]^{I_t^{(j)}}\right). \end{aligned} \tag{7}$$

The branching process approximation is based on the following simple heuristic argument. Assume that the numbers n_j are large enough and that the numbers m_j are finite. In the beginning of the epidemic when $S_t^{(j)}\simeq n_j$ and $I_t^{(j)}\simeq i_j$, then we obtain the following approximation:

$$f_{\mathbf{I}_{t+1}}(\mathbf{z}/\mathbf{S}_t,\mathbf{I}_t)\simeq\sum_{k_1=0}^{n_1}\cdots\sum_{k_J=0}^{n_J}\prod_{j=1}^{J}\left(C_{n_j}^{k_j}(1-z_j)^{k_j}z_j^{n_j-k_j}[q^{(j)}(k_1,\ldots,k_J)]^{i_j}\right), \tag{8}$$

consequently,

$$\begin{aligned} f_{\mathbf{I}_{t+1}}(\mathbf{z}/\mathbf{S}_t,\mathbf{I}_t) &\simeq \sum_{k_1=0}^{n_1}\cdots\sum_{k_J=0}^{n_J}\prod_{j=1}^{J} C_{n_j}^{k_j}(1-z_j)^{k_j}z_j^{n_j-k_j} \\ &\quad\times\prod_{j=1}^{J}[\hat{g}^{(j)}(1-\frac{k_1}{M_1},\ldots,1-\frac{k_J}{M_J})]^{i_j}. \end{aligned} \tag{9}$$

Using (9) and the Bernstein theorem [10], we conclude that

$$f_{\mathbf{I}_{t+1}}(\mathbf{z}/\mathbf{S}_t,\mathbf{I}_t)\simeq\prod_{j=1}^{J}\left[\hat{g}^{(j)}(1-\pi_1(1-z_1),\ldots,1-\pi_J(1-z_J))\right]^{I_t^{(j)}}. \tag{10}$$

The statement in (10) implies that $\mathbf{I}_{t+1}$ is approximately similarly distributed as the sum $I_t^{(1)}+\ldots+I_t^{(J)}$ of independent random vectors. $I_t^{(j)}, j=1,\ldots,J$ have the generating function $\hat{g}^{(j)}(1-\pi_1(1-z_1),\ldots,1-\pi_J(1-z_J))$ which is the generating function of new infected individuals caused by a simple infection of an individual from group j. In other words, $(\mathbf{I}_0,\ldots,\mathbf{I}_t)$ is approximately distributed as a multitype branching process, where each individual of group j, $j=1,\ldots,J$, has descendants of type l according to a probability distribution having $\hat{g}^{(j)}(1,\ldots,1,1-\pi_l(1-z_l),1,\ldots,1)$ as generating function and mean $\pi_l\dfrac{\partial\hat{g}^{(j)}}{\partial z_l}(1,\ldots,1)$.

Lemma 3.1 (Kissami, 1993)
Given any function $f \in \mathscr{C}(H=[0,1]^J, IR)$ suppose that, for $n=(n_1,...,n_J)$ the H-sequences $d_n=(d_n^1,...,d_n^J)$ and $c_n=(c_n^1,...,c_n^J)$ converge, respectively, to $d=(d_1,...,d_J)$ and $c=(c_1,...,c_J)$ then the polynomial

$$B_n(f,x)=\sum_{k_J=0}^{n_J}\prod_{j=1}^{J}C_{n_j}^{k_j}(1-x_j)^{k_j}x_j^{n_j-k_j}f(d_n^1+c_n^1\frac{k_1}{n_1},...,d_n^J+c_n^J\frac{k_J}{n_J})$$

converges uniformly to $f(d_1+c_1x_1,...,d_J+c_Jx_J)$ on H as $\min\limits_{i=1,..,j} n_i \to +\infty$.

Proof The following proof is concise than that given by Kissami (1993), it is motivated by Markov's inequality for random vectors. We begin by setting the following arguments : Let $x=(x_1,...,x_J)$ on H, the diagonal matrices $C_n=\mathrm{diag}(c_n)$ and $C=\mathrm{diag}(c)$ and a vector of independent random variables $S_n=(\frac{S_{n_1}}{n_1},...,\frac{S_{n_J}}{n_J})$ where S_{n_i} is binomial random $\mathscr{B}(n_i,x_i)$. Since f is continuous it follows that for $\beta_n=d_n+C_nS_n$, $\beta=d+Cx$ on H and a fixed $\varepsilon>0$, there exists $\delta>0$ such that $\|\beta_n-\beta\|<\delta$ implies $|f(\beta_n)-f(\beta)<\varepsilon$, here $\|.\|$ denotes the Euclidean norm of a vector. Consider the event $A_n:=\{\|\beta_n-\beta\|<\delta\}$, we have

$$\begin{aligned}
|B_n(f,x)-f(\beta)| &= |E(f(\beta_n)-f(\beta))| \\
&\le E(|f(\beta_n)-f(\beta)|) \\
&= E(1\!\!1_{A_n}|f(\beta_n)-f(\beta)|) \\
&\qquad +E(1\!\!1_{A_n^c}|f(\beta_n)-f(\beta)|) \\
&\le \varepsilon\mathbf{P}(A_n)+2\mathbf{P}(A_n^c)\sup_{x\in H}|f(x)| \\
&\le \varepsilon+2\mathbf{P}(\|\beta_n-\beta\|\ge\delta)\sup_{x\in H}|f(x)|.
\end{aligned}$$

Applying Markov's inequality and facts that $E(\frac{S_{n_i}}{n_i})=x_i$ and $var(\frac{S_{n_i}}{n_i})=\frac{x_i(1-x_i)}{n_i}$ yield

$$\begin{aligned}
\mathbf{P}(\|\beta_n-\beta\|\ge\delta) &\le \frac{E(\|\beta_n-\beta\|^2)}{\delta^2} \\
&= \frac{\sum_{i=1}^J E(d_n^i+c_n^i\frac{S_{n_i}}{n_i}-(d_i+c_ix_i))^2}{\delta^2} \\
&= \frac{\sum_{i=1}^J E\left(d_n^i-d_i+c_n^i(\frac{S_{n_i}}{n_i}-x_i)+(c_n^i-c)x_i\right)^2}{\delta^2} \\
&= \sum_{i=1}^J\frac{(d_n^i-d_i+(c_n^i-c)x_i)^2}{\delta^2}+\sum_{i=1}^J(c_n^i)^2\frac{E(\frac{S_{n_i}}{n_i}-x_i)^2}{\delta^2} \\
&= \frac{\|d_n+C_nx-d-Cx\|^2}{\delta^2}+\sum_{i=1}^J(c_n^i)^2\frac{x_i(1-x_i)}{n_i\delta^2}.
\end{aligned}$$

As $\sup\limits_{0\le x_i\le 1}(x_i(1-x_i))\le 1/4$ for all $i=1,..,J$ we have

$$\sup_{x\in H}|B_n(f,x)-f(d+Cx)|\le\varepsilon+\left(2\frac{\|d_n+C_nx-d-Cx\|^2}{\delta^2}+\frac{J\|c_n\|^2}{2\delta^2\min_{i=1,..,J}n_i}\right)\sup_{x\in H}|f(x)|$$

Furthermore, $\|d_n+C_nx-d-Cx\|\to 0$ and $\|c_n\|\to\|c\|$, as $\min\limits_{i=1,..,j} n_i\to+\infty$, hence

$$\lim_{\min_{i=1,..,J}}\sup_{n_i\to\infty}\sup_{x\in H}|B_n(f,x)-f(d+C_x)|\le\varepsilon,$$

which complets the proof.

Proposition 3.1 *Let $t \in itIN$ and $\mathbf{I}_0, \mathbf{I}_1, \ldots, \mathbf{I}_t$ fix vectors in $\mathbb{Z}_+^J$. Then*

$$f_{\mathbf{I}_{t+1}}(\mathbf{z}/\mathbf{S}_t, \mathbf{I}_t) \longrightarrow \prod_{j=1}^{J} \left[\hat{g}^{(j)}(1-\pi_1(1-z_1), \ldots, 1-\pi_J(1-z_J))\right]^{I_t^{(j)}}, \tag{11}$$

uniformly on $[0,1]^J$ as $n_j \longrightarrow +\infty$ for $j = 1, \ldots, J$.

Proof Let $G(\mathbf{z}) = \prod_{j=1}^{J} [\hat{g}^{(j)}(1-\pi_1(1-z_1), \ldots, 1-\pi_J(1-z_J)]^{I_t^{(j)}}$ and $B_{\mathbf{S}_t}^{(n)}(G, \mathbf{z})$ the Bernstein polynomial associated with the function $G(\mathbf{z})$. Then,

$$\begin{aligned}
&| f_{\mathbf{I}_{t+1}}(\mathbf{z}/\mathbf{S}_t, \mathbf{I}_t) - B_{\mathbf{S}_t}^{(n)}(G, \mathbf{z})| \\
&= \left| \sum_{k_1=0}^{S_t^{(1)}} \cdots \sum_{k_J=0}^{S_t^{(J)}} \prod_{j=1}^{J} C_{S_t^{(j)}}^{k_j} (1-z_j)^{k_j} z_j^{S_t^{(j)}-k_j} [q^{(j)}(k_1, \ldots, k_J)]^{I_t^{(j)}} - B_{\mathbf{S}_t}^{(n)}(G, \mathbf{z}) \right| \\
&\leq \left| \sum_{k_1=0}^{S_t^{(1)}} \cdots \sum_{k_J=0}^{S_t^{(J)}} \prod_{j=1}^{J} C_{S_t^{(j)}}^{k_j} (1-z_j)^{k_j} z_j^{S_t^{(j)}-k_j} \Big(\prod_{j=1}^{J} [q^{(j)}(k_1, \ldots, k_J)]^{I_t^{(j)}} - \right. \\
&\qquad \left. \prod_{j=1}^{J} [\hat{g}^{(j)}(1-\frac{k_1}{M_1}, \ldots, 1-\frac{k_J}{M_J})]^{I_t^{(j)}} \Big) \right| \\
&+ \left| G(\mathbf{z}) - \sum_{k_1=0}^{S_t^{(1)}} \cdots \sum_{k_J=0}^{S_t^{(J)}} \prod_{j=1}^{J} C_{S_t^{(j)}}^{k_j} (1-z_j)^{k_j} z_j^{S_t^{(j)}-k_j} [\hat{g}^{(j)}(1-\frac{k_1}{M_1}, \ldots, 1-\frac{k_J}{M_J})]^{I_t^{(j)}} \right| \\
&+ \left| B_{\mathbf{S}_t}^{(n)}(G, \mathbf{z}) - G(\mathbf{z}) \right| \\
&= E_1 + E_2 + E_3.
\end{aligned} \tag{12}$$

Using the triangle inequality we conclude that

$$|f_{\mathbf{I}_{t+1}}(\mathbf{z}/\mathbf{S}_t, \mathbf{I}_t) - G(\mathbf{z})| \leq E_1 + E_2 + 2E_3. \tag{13}$$

We now demonstrate that E_1, E_2 and E_3 converge to 0. We have

$$\begin{aligned}
E_1 &\leq \sup_{0 \leq k_i \leq n_i} \left| \prod_{j=1}^{J} [q^{(j)}(k_1, \ldots, k_J)]^{I_t^{(j)}} - \prod_{j=1}^{J} [\hat{g}^{(j)}(1-\frac{k_1}{M_1}, \ldots, 1-\frac{k_J}{M_J})]^{I_t^{(j)}} \right| \\
&\leq \sup_{0 \leq k_i \leq n_i} \sum_{j=1}^{J} I_t^{(j)} \left| q^{(j)}(k_1, \ldots, k_J) - \hat{g}^{(j)}(1-\frac{k_1}{M_1}, \ldots, 1-\frac{k_J}{M_J}) \right| \\
&\leq \sum_{j=1}^{J} I_t^{(j)} \sup_{0 \leq k_i \leq n_i} \left| q^{(j)}(k_1, \ldots, k_J) - \hat{g}^{(j)}(1-\frac{k_1}{M_1}, \ldots, 1-\frac{k_J}{M_J}) \right|,
\end{aligned} \tag{14}$$

combining (14) with the assumption (ii), we see that $E_1 \longrightarrow 0$ as $n_i \longrightarrow +\infty$, $i = 1,\ldots,J$.

$$
\begin{aligned}
E_2 = & \left| G(\mathbf{z}) - \sum_{k_1=0}^{S_t^{(1)}} \ldots \sum_{k_J=0}^{S_t^{(J)}} \prod_{j=1}^{J} C_{S_t^{(j)}}^{k_j} (1-z_j)^{k_j} z_j^{S_t^{(j)}-k_j} \right. \\
& \left. \times [\hat{g}^{(j)}(1-\frac{k_1}{M_1},\ldots,1-\frac{k_J}{M_J})]^{I_t^{(j)}} \right| \\
= & \left| G(\mathbf{z}) - \sum_{k_1=0}^{S_t^{(1)}} \ldots \sum_{k_J=0}^{S_t^{(J)}} \prod_{j=1}^{J} C_{S_t^{(j)}}^{k_j} z_j^{k_j} (1-z_j)^{S_t^{(j)}-k_j} \right. \\
& \left. \times [\hat{g}^{(j)}(\frac{M_1-S_t^{(1)}}{M_1}+\frac{k_1}{M_1},\ldots,\frac{M_J-S_t^{(J)}}{M_J}+\frac{k_J}{M_J})]^{I_t^{(j)}} \right| \qquad (15) \\
= & \left| G(\mathbf{z}) - \sum_{k_1=0}^{S_t^{(1)}} \ldots \sum_{k_J=0}^{S_t^{(J)}} \prod_{j=1}^{J} C_{S_t^{(j)}}^{k_j} (1-z_j)^{k_j} z_j^{S_t^{(j)}-k_j} \right. \\
& \left. \times [\hat{g}^{(j)}(C_n^{(1)}+\theta_n^{(1)}\frac{k_1}{S_t^{(1)}},\ldots,C_n^{(J)}+\theta_n^{(J)}\frac{k_J}{S_t^{(J)}})]^{I_t^{(j)}} \right|,
\end{aligned}
$$

where $\theta_n^{(i)} = \frac{S_t^{(i)}}{M_i}$ and $C_n^{(i)} = \frac{M_i - S_t^{(i)}}{M_i}$.
By combining (5) and the assumption (i) we deduce that

$$\lim_{n_i \to +\infty} C_n^{(i)} = 1 - \pi_i, \qquad (16)$$

and

$$\lim_{n_i \to +\infty} \theta_n^{(i)} = \pi_i, \quad i = 1,\ldots,J, \qquad (17)$$

hence from (15)-(17) and Lemma **3.1**, we see that $E_2 \longrightarrow 0$. In the same manner we can see that $E_3 \longrightarrow 0$, whenever $n_i \longrightarrow +\infty$ for $i = 1,\ldots,J$. This proves the Proposition.
Let Λ be the square matrix (J,J) having entries

$$\Lambda_{lj} = \left(\frac{\partial \hat{g}^{(l)}}{\partial z_j}(1,\ldots,1) \right), \quad j = 1,\ldots,J \text{ and } l = 1,\ldots,J, \qquad (18)$$

and let further Π be the diagonal matrix defined by

$$\Pi = diag(\pi) = \begin{pmatrix} \pi_1 & 0 & \ldots & 0 \\ 0 & \pi_2 & \ldots & 0 \\ . & . & . & . \\ 0 & 0 & \ldots & \pi_J \end{pmatrix}. \qquad (19)$$

Let R denotes the largest eigen-value of $\Lambda\Pi$ which represents the basic reproduction, it is defined as the mean number of infections arising from a single infected individual during his infectious period in a population of susceptibles, and $\mathbf{T}$ the total number of descendants in the multitype Galton-Watson process initiated by m_j individuals in each group j, $j = 1,\ldots,J$, where each individual in the group j, has descendants of type l according to a distribution with generating function $\hat{g}^{(j)}(1,\ldots,1-\pi_l(1-z_l),1,\ldots,1)$.

Proposition 1. $\mathbf{T}_n$ *converges in distribution towards* $\mathbf{T}$. *Moreover,*

(i)*If* $R \leq 1$, *the extinction probability is 1 and* $\sum_{\mathbf{L} \in IN^J} P(\mathbf{T} = \mathbf{L}) = 1$.

(ii)*If* $R > 1$, *extinction in group j takes place with probability* $\rho_j^{m_j}$, *j=1,...,J, and* $\sum_{\mathbf{L} \in IN^J} P(\mathbf{T} = \mathbf{L}) = \rho_1^{m_1}\rho_2^{m_2}\ldots\rho_J^{m_J}$. *Otherwise explosion takes place in group j with probability* $1-\rho_j^{m_j}$, *j=1,...,J, where the vector* $(\rho_1,\ldots,\rho_J)$ *is the root in* $[0,1]^J$ *of the system*

$$z_j = \hat{g}^{(j)}\left(1-\pi_1(1-z_1),\ldots,1-\pi_J(1-z_J)\right), \quad j = 1,\ldots,J. \qquad (20)$$

If, in addition, Λ is irreducible, $\Pi > 0$ and $R > 1$, then the probability of explosion of the process is given by $1 - \rho_1^{m_1}\rho_2^{m_2}\dots\rho_J^{m_J}$.

Proof Let $\mathbf{B} = (b_1,\dots,b_J) \in \mathbb{Z}_+^J$. We have ,

$$P[\mathbf{T}_n = \mathbf{B}] = \sum_{r=1}^{b} \sum_{D_r(\mathbf{B})} P[\mathbf{I}_1 = \mathbf{i}_1, \mathbf{I}_2 = \mathbf{i}_2, \dots, \mathbf{I}_r = \mathbf{i}_r, \mathbf{I}_{r+1} = \mathbf{0}], \tag{21}$$

where $b = \sum_{k=1}^{J} b_k$, $\mathbf{i}_1, \mathbf{i}_2, \dots, \mathbf{i}_r$ are vectors in $\mathbb{Z}_+^J$ and $D_r(\mathbf{B}) = \{(\mathbf{i}_1,\dots,\mathbf{i}_r) \in (\mathbb{Z}_+^J)^r$ such that $\mathbf{i}_1 > \mathbf{0},\dots,\mathbf{i}_r > \mathbf{0}$ and $\mathbf{i}_1 + \dots + \mathbf{i}_r = \mathbf{B}\}$. For each fix element $(\mathbf{i}_1,\dots,\mathbf{i}_r)$ of $D_r(\mathbf{B})$, we have

$$\begin{aligned}
&P[\mathbf{I}_1 = \mathbf{i}_1, \mathbf{I}_1 = \mathbf{i}_2, \dots, \mathbf{I}_\mathbf{r} = \mathbf{i}_r, \mathbf{I}_{r+1} = \mathbf{0}] = \\
&P[\mathbf{I}_1 = \mathbf{i}_1] P[\mathbf{I}_2 = \mathbf{i}_2 / \mathbf{I}_1 = \mathbf{i}_1; \mathbf{S}_1 = \mathbf{n} - \mathbf{i}_1] \times \\
&P[\mathbf{I}_3 = \mathbf{i}_3 / \mathbf{I}_2 = \mathbf{i}_2; \mathbf{S}_2 = \mathbf{n} - (\mathbf{i}_1 + \mathbf{i}_2)] \times \dots \times \\
&P[\mathbf{I}_{r+1} = \mathbf{i}_{r+1} / \mathbf{I}_r = \mathbf{i}_r; \mathbf{S}_r = \mathbf{n} - (\mathbf{i}_1 + \mathbf{i}_2 + \dots + \mathbf{i}_r)].
\end{aligned} \tag{22}$$

Thanks to Lemma **3.2** each probability in (22) converges to its counterpart in the context of the multitype Galton-Watson process where the generating function of new infectives caused by one single infected individual from group j is given by $\hat{g}^{(j)}(\pi_1(1-z_1),\dots,\pi_J(1-z_J))$. We can thus see that the probability of each term in (21) converges towards the corresponding probability in the above mentioned Galton-Watson process. Consequently, $P[\mathbf{T}_n = \mathbf{B}] \longrightarrow P(\mathbf{B})\ \forall \mathbf{B} \in \mathbb{Z}_+^J$, where P(.) is the distribution of the total population in the process. This proves the first statement of the proposition, for the second statement we refer the reader to the established proprieties of branching process published in [4]. Notice that when Λ is irreducible $\Pi > 0$ and $R > 1$, $\Lambda\Pi$ is irreducible as well as the Galton-Watson process is positively regular. Therefore, if $R > 1$, the random vector $\frac{\mathbf{I}_t}{R^t}$ converges almost surely to a random vector $\mathbf{W}$. Moreover, if $\mathbf{W} \neq 0$, the direction of $\mathbf{W}$ coincides almost surely with the left eigen-vector of the matrix $\Lambda\Pi$. Hence, $\mathbf{I}_t \sim R^t\mathbf{W}$ or $\mathbf{I}_{t+1} \sim R\mathbf{I}_t$. Therefore, $\mathbf{I}_t$ increases approximately as a geometric series of powers of R.

4 Examples of standard epidemic models

In what follows we describe some epidemic models that satisfy the condition (ii).

4.1 A multivariate extension of the general epidemic model

A direct extension of the general epidemic [3] consists in assuming that each infected individual in group j, $j = 1,\dots,J$, is infectious during some random period having the same distribution as a random variable D_j and during this period he can contact, independently of the others, any given susceptible from class l, $l = 1,\dots,J$, in an independent manner and at the points of a Poisson process with parameter $\beta_{jl}^{(n_j)}$. All periods of infection are independent from each other and from the contact process. It is thus clear that the resulting model is a special case of the collective model and for each j, $j = 1,\dots,J$,

$$q^{(j)}(k_1,\dots,k_J) = E(exp[-\sum_{i=1}^{J} k_i \beta_{ji}^{(n_j)} D_j]),\ \ k_1 \in [0,n_1],\dots,k_J \in [0,n_J], \tag{23}$$

where $\beta_{ji}^{(n_j)}$ is of the form $\frac{\hat{\beta}_{ji}^{(n_j)}}{n}$ with $\hat{\beta}_{ji}^{(n_j)} \to \beta_{ji}$, whenever $n_j \to +\infty$. We have

$$\begin{aligned}|q^{(j)}(k_1,\ldots,k_J) - E[exp\{-\sum_{i=1}^{J}\frac{k_i}{n}\beta_{ji}D_j\}]| &= |E[exp\{-\sum_{i=1}^{J}\frac{k_i}{n}\hat{\beta}_{ji}^{(n_j)}D_j\}] - \\ &\qquad E[exp\{-\sum_{i=1}^{J}\frac{k_i}{n}\beta_{ji}D_j\}]| \\ &\leq |E[\sum_{i=1}^{J}\frac{k_i}{n}\hat{\beta}_{ji}^{(n_j)}D_j - \sum_{i=1}^{J}\frac{k_i}{n}\beta_{ji}D_j]| \\ &\leq E(D_j)\sum_{i=1}^{J}\frac{k_i}{n}|\hat{\beta}_{ji}^{(n_j)} - \beta_{ji}| \\ &\leq E(D_j)\sum_{i=1}^{J}|\hat{\beta}_{ji}^{(n_j)} - \beta_{ji}| \to 0,\end{aligned}$$

when $n_j \to +\infty$. In this case we have

$$\hat{g}^{(j)}(\mathbf{z}) = E[exp\{-\sum_{i=1}^{J}(1-z_i)\beta_{ji}D_j\}],\ \ \mathbf{z}\in[0,1]^J, j=1,\ldots,J. \tag{24}$$

4.2 The randomised Reed-Frost process

The randomized Reed-Frost was introduced by [14], in the case of a homogeneous population. We modify that model by assuming that the contacts between infecteds and susceptibles follow a Poisson process. Here we assume that the probability of not transmitting the infection is a random variable with known distribution Q independently of everything else. The resulting model can be generalized in the non-homogeneous case in the following way. Every infected individual in group j does not transmit the infection to a susceptible individual from group i with a random probability distributed according to Q_{ji}. This implies the following

$$q^{(j)}(k_1,\ldots,k_J) = E\left[\prod_{i=1}^{J}Q_{ji}^{k_i}\right],\ \ k_1\in[0,n_1],\ldots,k_J\in[0,n_J], \tag{25}$$

which can also be written in the form

$$q^{(j)}(k_1,\ldots,k_J) = E\left[\prod_{i=1}^{J}e^{-k_iY_{ji}^{(n_i)}}\right],\ \ k_1\in[0,n_1],\ldots,k_J\in[0,n_J],$$

where $Y_{ji}^{(n_i)} = -\ln(Q_{ji})$.

We assume that for every $i,\ \ i=1,\ldots,J$, $n_iY_{ji}^{(n_i)}$ converges in mean towards a random variable $\bar{Y}_{ji}$ as $n_i \to +\infty$. Therefore,

$$\begin{aligned}|q^{(j)}(k_1,\ldots,k_J) - E[exp(-\sum_{i=1}^{J}\frac{k_i\bar{Y}_{ji}}{n_i})]| &= |E[exp(-\sum_{i=1}^{J}k_iY_{ji}^{(n_i)})] - \\ &\qquad E[exp(-\sum_{i=1}^{J}\frac{k_i\bar{Y}_{ji}}{n_i})]| \\ &\leq |E[\sum_{i=1}^{J}k_iY_{ji}^{(n_i)} - \sum_{i=1}^{J}\frac{k_i\bar{Y}_{ji}}{n_i}]| \\ &\leq E[\sum_{i=1}^{J}\frac{k_i}{n_i}|n_iY_{ji}^{(n_i)} - \bar{Y}_{ji}|] \\ &\leq E[\sum_{i=1}^{J}|n_iY_{ji}^{(n_i)} - \bar{Y}_{ji}|] \longrightarrow 0,\end{aligned} \tag{26}$$

when $n_i \longrightarrow +\infty$ for $i = 1,\ldots,J$.
As a consequence, we have

$$\hat{g}^{(j)}(\mathbf{z}) = E[exp\{\sum_{i=1}^{J} -(1-z_i)\bar{Y}_{ji}\}],\ \ \mathbf{z} \in [0,1]^J,\ \ j = 1,\ldots,J. \tag{27}$$

The previous model is the special case of the model where $Q_{ji} = exp(-\beta_{ji}D_j)$.

4.3 The model of Lefèvre and Picard

[9] have constructed a larger model by representing the infection process using a sampling process. Here, each infected in group j, $j = 1,\ldots,J$, transmits the infection, independently of the others, by choosing with replacement, from each of the J classes of susceptibles random subsets of respective sizes $R_{j,1},\ldots,R_{j,J}$. Thus for each j, $j = 1,\ldots,J$, we have

$$q^{(j)}(k_1,\ldots,k_J) = E\left[\prod_{i=1}^{J}(1-\frac{k_i}{n_i})^{R_{ji}}\right],\ \ k_1 \in [0,n_1],\ldots,k_J \in [0,n_J], \tag{28}$$

then

$$\hat{g}^{(j)}(\mathbf{z}) = E\left[\prod_{i=1}^{J} z_i^{R_{ji}}\right],\ \ \mathbf{z} \in [0,1]^J,\ \ j = 1,\ldots,J. \tag{29}$$

4.4 The model of H. Andersson

[1] managed to extend the Martin-Löf model [11]. In his model each infected in group j, $j = 1,\ldots,J$, transmits the infection, independently of the others, by choosing without replacement, from each of the J classes of susceptibles one random subset of size in $R_{j,1},\ldots,R_{j,J}$. Accordingly, for each j, $j = 1,\ldots,J$,

$$q^{(j)}(k_1,\ldots,k_J) = E\left[\prod_{i=1}^{J} C_{n_i-k_i}^{R_{ji}}/C_{n_i}^{R_{ji}}\right],\ \ k_1 \in [0,n_1],\ldots,k_J \in [0,n_J], \tag{30}$$

which can also be written as

$$q^{(j)}(k_1,\ldots,k_J) = E\left[\prod_{i=1}^{J}\left(\prod_{s=0}^{R_{ji}-1}(1-\frac{k_i}{n_i-s})\right)\right],\ \ k_1 \in [0,n_1],\ldots,k_J \in [0,n_J].$$

We have

$$\prod_{s=0}^{R_{ji}-1}\left(1-\frac{k_i}{n_i-s}\right) = \prod_{s=0}^{R_{ji}-1}\left[1-\frac{k_i}{n_i}-\frac{k_i}{n_i}\frac{s/n_i}{(1-s/n_i)}\right]. \tag{31}$$

Since the function $x \longmapsto \frac{x}{1-x}$ increasing in $[0,+\infty[$ then for each $s \leq R_{ji} \leq n_i - k_i$, we have

$$\frac{k_i}{n_i}\frac{(s/n_i)}{(1-s/n_i)} \leq \frac{k_i}{n_i}\frac{R_{ji}/n_i}{(1-R_{j,i}/n_i)} \leq \frac{R_{ji}}{n_i}. \tag{32}$$

Hence combining (31) with (32), we obtain

$$\prod_{s=0}^{R_{ji}-1}(1-\frac{k_i}{n_i-s}) \geq \prod_{s=0}^{R_{ji}-1}(1-\frac{k_i}{n_i}-\frac{R_{ji}}{n_i}) = \left(1-\frac{k_i}{n_i}-\frac{R_{ji}}{n_i}\right)^{R_{ji}}. \tag{33}$$

Applying simple facts from analysis on the function $x \longmapsto x^{R_{ji}}$ in the interval $[1-\frac{k_i}{n_i}-\frac{R_{ji}}{n_i}, 1-\frac{k_i}{n_i}]$ we obtain

$$\left(1-\frac{k_i}{n_i}-\frac{R_{ji}}{n_i}\right)^{R_{ji}} = \left(1-\frac{k_i}{n_i}\right)^{R_{ji}} - \frac{R_{ji}^2}{n_i}\theta^{R_{ji}-1},\ \ \theta \in]1-\frac{k_i}{n_i}-\frac{R_{ji}}{n_i}, 1-\frac{k_i}{n_i}[. \tag{34}$$

From (33) and (34), we have

$$\prod_{s=0}^{R_{ji}-1}\left(1-\frac{k_i}{n_i-s}\right)\geq\left(1-\frac{k_i}{n_i}\right)^{R_{ji}}-\frac{R_{ji}^2}{n_i}. \tag{35}$$

Moreover,

$$\prod_{s=0}^{R_{ji}-1}\left(1-\frac{k_i}{n_i-s}\right)\leq\left(1-\frac{k_i}{n_i}\right)^{R_{ji}}. \tag{36}$$

If $E(R_{ji}^2)<+\infty$, combining (35) with (36) and taking the expectation give, for each j, $j=1,\ldots,J$,

$$\begin{aligned}\left|q^{(j)}(k_1,\ldots,k_J)-E\left[\prod_{i=1}^{J}\left(1-\frac{k_i}{n_i}\right)^{R_{ji}}\right]\right| &\leq E\left[\prod_{i=1}^{J}\left(\prod_{s=0}^{R_{ji}-1}(1-\frac{k_i}{n_i-s})\right)-\prod_{i=1}^{J}\left(1-\frac{k_i}{n_i}\right)^{R_{ji}}\right]\\ &\leq E\left[\sum_{i=1}^{J}\left(\prod_{s=0}^{R_{ji}-1}(1-\frac{k_i}{n_i-s})-\left(1-\frac{k_i}{n_i}\right)^{R_{ji}}\right)\right]\\ &\leq E\left[\sum_{i=1}^{J}\frac{R_{ji}^2}{n_i}\right]\longrightarrow 0,\end{aligned}$$

when $n_i\longrightarrow+\infty$, $i=1,\ldots,J$.
It is thus clear that the functions $\hat{g}^{(j)}(\mathbf{z})$, $j=1,\ldots,J$, are given by

$$\hat{g}^{(j)}(\mathbf{z})=E\left[\prod_{i=1}^{J}z_i^{R_{ji}}\right],\ \mathbf{z}\in[0,1]^J,\ j=1,\ldots,J. \tag{37}$$

When comparing (29) and (37), we see that the distribution of the final size of the epidemic is the same for the models in Sections 4.3 and 4.4. This is not a surprise, the reason is that in the two cases the infecteves transmit the infection by sampling random subsets from each of the J classes of susceptibles. For the model presented in Section 4.2 the sampling is performed with replacement whereas in model in Section 4.3 sampling is without replacement. As n grows the two sampling schemes become more and more like each other.

5 Simulation examples

It is well known [4] that in the subcritical case, where the basic reproduction number, R_0, satisfies the condition $R_0<1$, the extinction of the Galton-Watson process takes place with probability 1. This case corresponds to a minor outbreak. In the supercritical case where $R_0>1$, the Galton-Watson process can escape extinction with a positive probability, which corresponds to a major outbreak. In the latter case, the branching approximation is not suitable [7]. To illustrate this fact, we give some numerical results for the general epidemic model presented in the Section 4.1 by considering two interacting populations. In this section we repeatedly simulate, by using the conditional distribution in (3) and the equality (2), the number of infectives in the two populations and compare it to two interconnected Galton-Watson process for different values of R_0 and for large populations sizes.

For the general epidemic, the infectious periods D_1 and D_2 in the first and second populations are supposed to be negative exponential random variables with means μ_1^{-1} and μ_2^{-1} respectively and the parameter β_{ij} is the pairwise rate for a susceptible from population i to be infected by an infective in population j, $i,j=1,2$ which corresponds to the birth and death rates for the two interconnected Galton-Watson process. With these arguments and by supposing that the infection can only be transmitted between the populations ($\beta_{11}=\beta_{22}=0$), (18), (19) and (23) imply that $R_0=\sqrt{R_{012}R_{21}}$ where $R_{0rs}=\frac{\beta_{rs}}{\mu_r}; r\neq s$.
Figure 1 and Figure 2, for various values of n_1 and n_2, show where time (measured in generations) $t\in 0,1,2,....20$ and

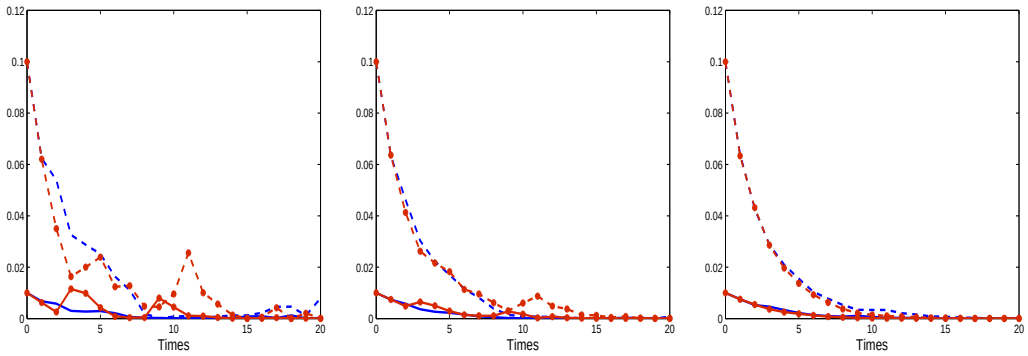

Fig. 1 Single sample of infectives. first population (—) and seconde population (−−) against the single sample of the Galton-Watson process first component (— line dotted) and seconde component (−− dashed line dotted), for $R_0 = 0.4$ and $n_1 = n_2 = 100$, $n_1 = n_2 = 1000$ and $n_1 = n_2 = 10000$.

the fractions of infectives in the two populations against the Galton-Watson approximation fractions. In the first examples (Figure 1), where the parameter values are chosen so that the basic reproduction number satisfies $R_0 < 1$, we see that the numbers of infective individuals in the two populations and the Galton-Watson process die out. In this case, we see that the proximation works well when the initial sizes are large enough. In the second example, where $R_0 \geq 1$, we see that (Figure 2) the epidemic extinction occurs slowly while the branching process continues to grow. In this case the approximation it is not good. In the third example, the frequency histograms show how the joint and marginal approximate distribution of the number of infectives seem to be well approximated by a normal distribution (Figure 3 and Figure 4). The latter fact will be the subject of future research on the generalized Multitype Collective Reed-Frost Model.

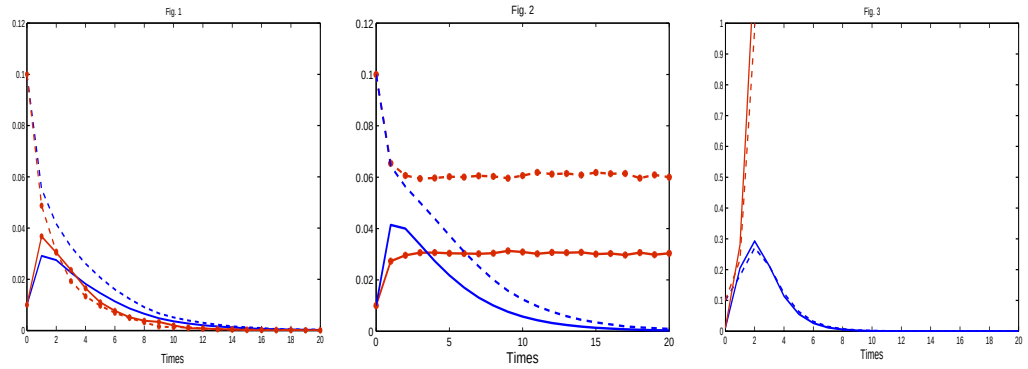

Fig. 2 Single sample of infectives. first population (—) and seconde population (−−) against the single sample of the Galton-Watson process first component (— line dotted) and seconde component (−− dashed line dotted) for $R_0 = 0.85$ (Fig.1), $R_0 = 1$ (Fig.2) and $R_0 = 2.4$ (Fig.3) when $n_1 = n_2 = 10000$.

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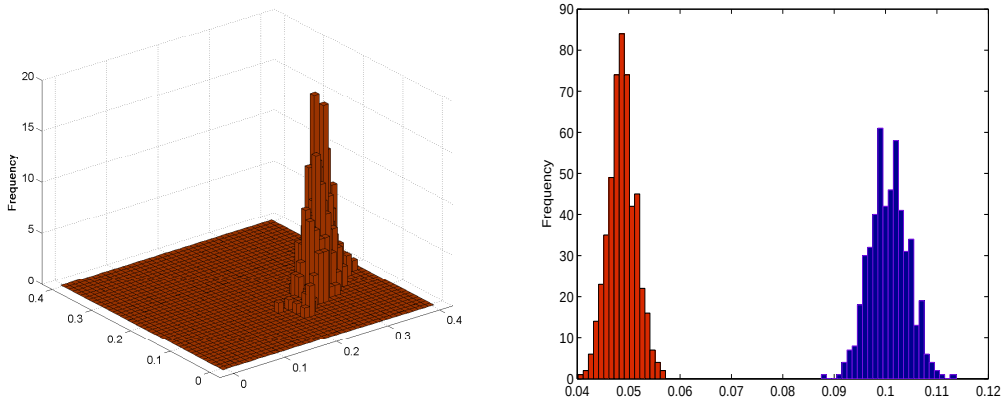

Fig. 3 Frequency histograms based on 10000 simulations for joint (left) and marginal (right) distribution of the numbers of infectives in each population at generation $t = 10$ for $R_0 = 2.4$ when $n_1 = n_2 = 10000$.

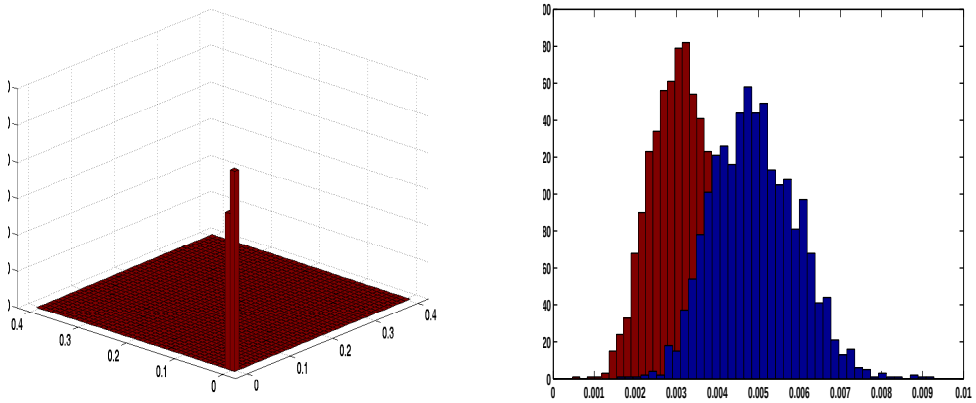

Fig. 4 Frequency histograms based on 10000 simulations for joint (left) and marginal (right) distribution of the numbers of infectives in each population at generation $t = 10$ for $R_0 = 0.85$ when $n_1 = n_2 = 10000$.

References

[1] Andersson, H. A threshold limit theorem for a multitype epidemic model. Math. Biosci., 117: 3-18, (1993).
[2] Bailey, N. T. J. The Mathematical Theory of Infectious Diseases. 2re Edn., Griffin, London, (1975).
[3] Ball F. A unified approach to the distribution of total size and total area under the trajectory of infectives in epidemic models. Adv. Appl. Prob., 18: 289-310, (1986).
[4] Harris, T. E. The theory of Branching Processes. Springer-Verlag, Berlin, (1963).
[5] Isham V. Mathematical modelling of the transmission dynamics of HIV infection and AIDS: a review. J. Roy. Statist. Soc., A 151: 5-30, (1988).
[6] Karim, S.A.A and R. Razali. Aproposed mathematical model of influenza. J. App. Sci., 11: 1457-1460, (2011).
[7] Kissami, A. Problèmes d'urnes et processus épidémiques: modélisation et résultats asymptotiques. Thèse de Docorat. ULB, Bruxelles, (1993).
[8] Kissami, A. and C. Lefèvre and P. Picard. On collective models and partially exchangeable events: a further look motivated by the modelling of epidemic processes. Cahiers de C.E.R.O., 36: 235-250, (1994).
[9] Lefèvre, C. and P. Picard. A non-standard family of polynomials and the final size distribustion of Reed-Frost epidemic processes. Adv. Appl. Prob., 22: 25-48, (1990).

[10] Bernstein polynomials. Chelsea Publishing Company, New York.
[11] Martin-Löf, A. Symetric sampling procedures, general epidemic processes and their threshold limit theorems. J. Appl. Prob., 23: 265-282, (1986).
[12] Picard, P. and C. Lefèvre, 1990. A unified analysis of the final size and severity distribustion in collective Reed-Frost epidemic processes. Adv. Appl. Prob., 22 : 269-294,(1990).
[13] Sani, A. and D. P. Kroese and P. K. Pollett. Stochastic models for the spread of HIV in a mobile heterosexual population. Math. Biosci., 208: 98-124, (2007).

[14] Von Bahr, B. and A. Martin-Löf. Threshold limit theorems for some epidemic processes. Adv. Appl. Prob., 12: 319-349, (1980).