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Commun. Math. Biol. Neurosci. 2022, 2022:18

<https://doi.org/10.28919/cmbn/7098>

ISSN: 2052-2541

THE ASYMPTOTIC BEHAVIOR OF AN SIR EPIDEMIC MODEL: COLLECTIVE REED-FROST PROCESS

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Abstract. The aim of this research is to provide a historical overview of the mathematical theory of epidemics and to study the asymptotic behavior of the final size of a collective Reed-Frost epidemic process with different types of infected people. This model was introduced by Picard and Lefèvre [25] provides an extension of the model of Pettigrew and Weiss [24]. Under certain conditions, we show that when the number of the initial susceptible individuals is large and the number of the initial infected people is finite, the infection process is equivalent to a multitype Galton-Watson process. Our method is simple and based on Bernstein polynomials.

Keywords: Covid-19; epidemiological model; collective Reed-Frost process; final size of the epidemy; Galton-Watson process; Bernstein polynomials.

2010 AMS Subject Classification: 92D30, 65C40.

1. INTRODUCTION

For centuries, epidemics have been an inexorable threat to mankind. They have been a major public health problem in all countries. The impact of these epidemics on the productivity

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Received December 19, 2021

potential of countries and on their socio-economic development is devastating. Mathematical models are one of the important and basic tools that provide some information and predictions that help in understanding and measuring the dynamical behavior of the epidemic.

In December 2019, the world knew the outbreak of a new pandemic, namely, coronavirus disease 2019 (COVID-19). Faced with this complex and serious situation and in the absence of effective vaccines and preventive measures, the interested parties have resorted to mathematical treatments of these plagues in order to help control the epidemic and take measures to prevent its spread, for example: closing borders, awareness, imposing curfews and wearing masks. Many models have been suggested to study the dynamics of COVID-19. For instance, Feng et al. [9] proposed a mathematical model to study the effects of media coverage and quarantine strategy on the COVID-19 infections in the UK. Pang et al. [23] established a model with ordinary differential equations to describe the transmission dynamics of COVID-19. Giordano et al. [11] have proposed a new model that predicts the course of the epidemic to help plan an effective control strategy. The model considers eight stages of infection: susceptible (S), infected (I), diagnosed (D), ailing (A), recognized (R), threatened (T), healed (H) and extinct (E), collectively termed SIDARTHE, this model discriminates between infected individuals depending on whether they have been diagnosed and on the severity of their symptoms. These researchers model possible scenarios of implementation of countermeasures and demonstrate that restrictive social-distancing measures will need to be combined with widespread testing and contact tracing to end the ongoing. Hattaf et al. [14] developed an epidemiological model that incorporates different modes of transmission of COVID-19 and take into account the effect of awareness programs by media on the spread of this dangerous disease.

The history of models implying an epidemic process is quite old. For example, Hammer [15] assumed that the number of new cases of an infectious disease can only depend on the number of existing cases and the number of susceptible individuals in the population. The mathematical theory of epidemics has continued to develop, the most interesting contributions, which have been made, are provided mainly by the following studies: Sellke [28], Ball [3], Gabriel et al. [10], Picard and Lefèvre [25], Kissami [18], Andersson [1], Ball and Clancy [4], Esegir [7], Esegir et al. [8], El Maroufy et al. [5], Hattaf et al. [12, 13].

The epidemic threshold is one of the most important concepts in the study of epidemics, though being a theoretical concept, it allows us to study the behaviour of an epidemic and to control it. This threshold shows that in order to fight effectively against an epidemic danger, it is not necessary, for example, to vaccinate an entire population, it is sufficient to reduce the number of susceptible individuals below the threshold. The original coronavirus required an estimated 67% of the population to be vaccinated to achieve herd immunity, For Delta, those threshold estimates go well over 80% and may be approaching 90%. Also, measles has one of the highest herd immunity thresholds at 95%. Several vaccines with different efficiency and effectiveness are currently being distributed across the world to control the COVID-19 pandemic, hence, policymakers may propose using various combinations of available vaccines to control the pandemic with vaccine-induced herd immunity by vaccinating a fraction of the population. So the herd-immunity threshold must be updated for multi-vaccine strategies and multiple variants [31].

On the other hand, the statistic of interest that has been studied intensively by different authors is the final size of the epidemic: the total number of new cases of infection that have occurred during the process of infection. The central problem for the epidemic processes, is to study, for a given model, the asymptotic behavior of the final size of the epidemic. An important class of epidemic models relates to infectious diseases of the type SIR (Susceptible, Infected, Removed). Their main characteristics are as follows. A closed population is subdivided into three classes, susceptible (healthy individuals but exposed to infection), infected (individuals carrying the infectious germ and who can transmit it) and removed (infected individuals who leave their condition by healing and immunization, or by death). Each infected individual remains infectious for a random period of time called the infection period. During this period, the infected behave independently of each other and may contact the susceptible present, who in this case, in turn become infected. Once an individual's period of infection is over, they are permanently removed from the infection process: depending on the type of the disease, it is healed and is immune, or it dies. For more details, we refer the reader to the book by Bailey [2] and to the volumes edited by Gabriel et al. [10] and Mollison et al. [22].

Many methods and different types of models have been used, in particular, by Von Bahr and

Martin-Löf [29], Scalia-Tomba [27], Picard and Lefèvre [25], Andersson [1], Ball and Clancy [4], to study the asymptotic distribution of the final size of the epidemic when the number of initial susceptible is large enough.

In the present article, we consider an epidemic process with different types of infected. This situation occurs, for example, when the disease is transmitted by both clinically infected people (called infected) and subclinically infected people (called carriers) as in the case of COVID-19. So, we consider a closed and homogeneous population partitioned into three classes, the susceptible, the infected and the removed cases, but now the infected are divided into J types, $J \geq 1$. Ball and Clancy [4], consider the situation where each type infected i , $i = 1, \dots, J$, is infectious for a period distributed as a random variable D_i , and during this period, it can contact, independently of the others, any susceptible at the points of a Poisson process of rate β_i . For this model, these last authors determined the asymptotic distribution of the final size. Our purpose here is to partially extend the results obtained by Ball and Clancy [4] to the case of the general collective Reed-Frost model developed by Picard and Lefèvre [25] and which we will describe below.

2. COLLECTIVE REED-FROST MODEL: CASES OF DIFFERENT TYPES OF INFECTED

Consider a closed population and suppose that the infection is transmitted according to the following rules:

- a) The propagation of the disease is described through the sizes of the successive generations of infected individuals. At each time t , $t \in \mathbb{N}$, the state of the population is given by $\{S_t, I_t^{(j)}, j = 1, \dots, J, t \in \mathbb{N}\}$,
 S_t is the number of susceptible at time t and $I_t^{(j)}$ represents the number of infected type j at time t . Initially, $(S_0, I_0^{(j)}) = (n, m_j)$ and we have

$$S_t = S_{t+1} + \sum_{j=1}^J I_{t+1}^{(j)}, \quad t \in \mathbb{N}. \quad (1)$$

- b) $\{S_t, I_t^{(j)}, j = 1, \dots, J, t \in \mathbb{N}\}$, is a Markov chain with transitions governed by the following rule : Initially the numbers of susceptibles is n , and consider any subset of size k , $k \in [0, n]$ in that class . So, all infected of any generation behave independently.

Moreover, each infected individu of type j , $j = 1, \dots, J$, does not transmit the infection within such a susceptible subset with the (known) probability $q^{(j)}(k)$ which depends only on the type j , and the sizes k and n .

The probabilities $q^{(j)}(k)$ allow us to determine the conditional law of survival by generation. A direct use of formula (3.5) of Kissami [18], gives

$$P \left[S_{t+1} = s / (S_t, I_t^{(j)}, j = 1, \dots, J) \right] = \sum_{k=s}^{S_t} \left(C_{S_t}^k C_k^s (-1)^{k-s} \left[\prod_{j=1}^J q^{(j)}(k) I_t^{(j)} \right] \right), \quad (2)$$

$s \in [0, S_t]$, and $t \in \mathbb{N}$. Subsequently, it is assumed that once a susceptible is contracted it becomes infected of type i with a probability π_i such as $\sum_{i=1}^J \pi_i = 1$.

Moreover, the process $\{S_t, I_t^{(j)}, j = 1, \dots, J, t \in \mathbb{N}\}$, is terminated at the moment

$$K = \inf \left\{ t / I_t^{(j)} = 0, j = 1, \dots, J \right\}. \quad (3)$$

S_K is thus the ultimate number of susceptibles which have avoided contact with all infected. Let T_n^i is the number of initially susceptible individuals who gave birth to infected with type i during the infection process. $\mathbf{T}_n = (T_n^1, \dots, T_n^J)$, denotes the final size of the epidemic.

3. A BRANCHING PROCESS APPROXIMATION

To approximate our process using a branching process, we need the following assumptions

(i) $m_j, j = 1, \dots, J$ is finite .

(ii) For any $j, j = 1, \dots, J$, there exists a continuous function $\hat{g}^{(j)}$ from $[0, 1]$ in $[0, 1]$ such that $|q^{(j)}(k) - \hat{g}^{(j)}(1 - \frac{k}{n})| \rightarrow 0$, whenever $n \rightarrow +\infty$, uniformly on $k, k \in [0, n]$.

Let $\mathbf{I}_t = (I_t^{(1)}, \dots, I_t^{(J)})$, $\mathbf{z} = (z_1, \dots, z_J) \in [0, 1]^J$, $x \in [0, 1]$ and denote by $g_{S_{t+1}}(x/S_t, \mathbf{I}_t)$, (respectively $f_{\mathbf{I}_{t+1}}(\mathbf{z}/S_t, \mathbf{I}_t)$), $t = 0, 1, \dots$, is the conditional generating function \mathbf{S}_{t+1} (respectively of \mathbf{I}_{t+1}). Using the formula (2.7) in Kissami et al. (1995)[19], we obtain

$$g_{S_{t+1}}(x/S_t, \mathbf{I}_t) = E(x^{S_{t+1}}/S_t, \mathbf{I}_t) = \sum_{k=0}^{S_t} C_{S_t}^k (x-1)^k \prod_{j=1}^J [q^{(j)}(k)]^{I_t^{(j)}}. \quad (4)$$

Since,

$$S_{t+1} = S_t - \sum_{j=1}^J I_{t+1}^{(j)}, \quad t \in \mathbb{N}.$$

we have,

$$\begin{aligned}
f_{\mathbf{I}_{t+1}}(\mathbf{z}/S_t, \mathbf{I}_t) &= E\left(\prod_{i=1}^J z_i^{I_t^{(i)}} / S_t, \mathbf{I}_t\right) \\
&= E\left[E\left(\prod_{i=1}^J z_i^{I_t^{(i)}} / S_t, S_{t+1}, \mathbf{I}_t\right) / S_t, \mathbf{I}_t\right] \\
&= E\left[\left(\sum_{i=1}^J \pi_i z_i\right)^{S_t - S_{t+1}} / S_t, \mathbf{I}_t\right] \\
&= \left(\sum_{i=1}^J \pi_i z_i\right)^{S_t} E\left[\left(\frac{1}{\sum_{i=1}^J \pi_i z_i}\right)^{S_{t+1}} / S_t, \mathbf{I}_t\right] \\
&= \left(\sum_{i=1}^J \pi_i z_i\right)^{S_t} g_{S_{t+1}}\left[\frac{1}{\sum_{i=1}^J \pi_i z_i} / S_t, \mathbf{I}_t\right] \\
&= \left(\sum_{i=1}^J \pi_i z_i\right)^{S_t} \sum_{k=0}^{S_t} C_{S_t}^k \left(\frac{1}{\sum_{i=1}^J \pi_i z_i} - 1\right)^k \prod_{j=1}^J [q^{(j)}(k)]^{I_t^{(j)}} \\
&= \sum_{k=0}^{S_t} C_{S_t}^k \left[1 - \sum_{i=1}^J \pi_i z_i\right]^k \left(\sum_{i=1}^J \pi_i z_i\right)^{S_t - k} \prod_{j=1}^J [q^{(j)}(k)]^{I_t^{(j)}}.
\end{aligned} \tag{5}$$

The branching process approximation is based on the following simple heuristic argument. Assume that the number n is large enough and that the numbers m_j are finite. In the beginning of the epidemics when $S_t \simeq n$, we have the approximation,

$$f_{\mathbf{I}_{t+1}}(\mathbf{z}/S_t, \mathbf{I}_t) \simeq \sum_{k=0}^n C_n^k \left[1 - \sum_{i=1}^J \pi_i z_i\right]^k \left(\sum_{i=1}^J \pi_i z_i\right)^{n-k} \prod_{j=1}^J [q^{(j)}(k)]^{I_t^{(j)}}. \tag{6}$$

Consequently,

$$f_{\mathbf{I}_{t+1}}(\mathbf{z}/S_t, \mathbf{I}_t) \simeq \sum_{k=0}^n C_n^k \left[1 - \sum_{i=1}^J \pi_i z_i\right]^k \left(\sum_{i=1}^J \pi_i z_i\right)^{n-k} \prod_{j=1}^J [\hat{g}^{(j)}\left(1 - \frac{k}{n}\right)]^{I_t^{(j)}}. \tag{7}$$

Using (7) and the Bernstein theorem [cf. e.g. Lorentz (1986)[20]], we conclude that

$$f_{\mathbf{I}_{t+1}}(\mathbf{z}/S_t, \mathbf{I}_t) \simeq \prod_{j=1}^J \left[\hat{g}^{(j)}\left(\sum_{i=1}^J \pi_i z_i\right) \right]^{I_t^{(j)}}. \tag{8}$$

This implies that, \mathbf{I}_{t+1} is approximately similarly distributed as the sum $I_t^{(1)} + \dots + I_t^{(J)}$ of independent random vectors. $I_t^{(j)}$, $j = 1, \dots, J$, of these have the generating function $\hat{g}^{(j)}\left(\sum_{i=1}^J \pi_i z_i\right)$, where $\hat{g}^{(j)}\left(\sum_{i=1}^J \pi_i z_i\right)$ is the generating function of new infected individuals due

to simple infection of an individual from group j . In other words, $(\mathbf{I}_0, \dots, \mathbf{I}_t)$ is approximately distributed as a multitype branching process, where each individual of group j , $j = 1, \dots, J$, has descendants of type l according to a probability law having as generating function $\hat{g}^{(j)}(1 - \pi_l(1 - z_l))$ and mean $\pi_l[\hat{g}^{(j)}]'(1)$.

Lemma 3.1.

Let $t \in \mathbb{N}$ et $\mathbf{I}_0, \mathbf{I}_1, \dots, \mathbf{I}_t$ fix vectors in \mathbb{N}^J . Then

$$f_{\mathbf{I}_{t+1}}(\mathbf{z}/S_t, \mathbf{I}_t) \longrightarrow \prod_{j=1}^J \left[\hat{g}^{(j)} \left(\sum_{i=1}^J \pi_i z_i \right) \right]^{I_t^{(j)}}, \quad (9)$$

uniformly on $[0, 1]^J$, as $n \longrightarrow +\infty$.

Proof

Note first that for all t , $t \in \mathbb{N}^*$, $S_t = n - (\sum_{j=1}^J I_1^{(j)} + \dots + \sum_{j=1}^J I_t^{(j)})$. Therefore, when $n \longrightarrow +\infty$, $S_t \longrightarrow +\infty$ et $\frac{S_t}{n} \longrightarrow 1$.

On the other hand, let

$G(\mathbf{z}) = \prod_{j=1}^J \left[\hat{g}^{(j)} \left(\sum_{i=1}^J \pi_i z_i \right) \right]^{I_t^{(j)}}$ et $B_{S_t}^{(n)}(G, \mathbf{z})$ the Bernstein polynomial associated with the function $G(\mathbf{z})$. Then,

$$\begin{aligned} & |f_{\mathbf{I}_{t+1}}(\mathbf{z}/S_t, \mathbf{I}_t) - B_{S_t}^{(n)}(G, \mathbf{z})| \\ &= \left| \sum_{k=0}^{S_t} C_{S_t}^k \left[1 - \sum_{i=1}^J \pi_i z_i \right]^k \left(\sum_{i=1}^J \pi_i z_i \right)^{S_t-k} \prod_{j=1}^J [q^{(j)}(k)]^{I_t^{(j)}} - B_{S_t}^{(n)}(G, \mathbf{z}) \right| \\ &\leq \left| \sum_{k=0}^{S_t} C_{S_t}^k \left[1 - \sum_{i=1}^J \pi_i z_i \right]^k \left(\sum_{i=1}^J \pi_i z_i \right)^{S_t-k} \prod_{j=1}^J [q^{(j)}(k)]^{I_t^{(j)}} - \prod_{j=1}^J [\hat{g}^{(j)}(1 - \frac{k}{n})]^{I_t^{(j)}} \right| \quad (10) \\ &+ \left| G(\mathbf{z}) - \sum_{k=0}^{S_t} C_{S_t}^k \left[1 - \sum_{i=1}^J \pi_i z_i \right]^k \left(\sum_{i=1}^J \pi_i z_i \right)^{S_t-k} \prod_{j=1}^J [\hat{g}^{(j)}(1 - \frac{k}{n})]^{I_t^{(j)}} \right| \\ &+ |B_{S_t}^{(n)}(G, \mathbf{z}) - G(\mathbf{z})| \\ &= E_1 + E_2 + E_3. \end{aligned}$$

Using the triangle inequality we conclude that

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

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

$$\begin{aligned}
E_1 &= \left| \sum_{k=0}^{S_t} C_{S_t}^k [1 - \sum_{i=1}^J \pi_i z_i]^k (\sum_{i=1}^J \pi_i z_i)^{S_t-k} \prod_{j=1}^J [q^{(j)}(k)]^{I_t^{(j)}} - \prod_{j=1}^J [\hat{g}^{(j)}(1 - \frac{k}{n})]^{I_t^{(j)}} \right| \\
&\leq \sup_{0 \leq k \leq n} \left| \prod_{j=1}^J [q^{(j)}(k)]^{I_t^{(j)}} - \prod_{j=1}^J [\hat{g}^{(j)}(1 - \frac{k}{n})]^{I_t^{(j)}} \right| \\
&\leq \sup_{0 \leq k \leq n} \sum_{j=1}^J I_t^{(j)} |q^{(j)}(k) - \hat{g}^{(j)}(1 - \frac{k}{n})| \\
&\leq \sum_{j=1}^J I_t^{(j)} \sup_{0 \leq k \leq n} |q^{(j)}(k) - \hat{g}^{(j)}(1 - \frac{k}{n})|.
\end{aligned} \tag{11}$$

Using (11) and the hypothesis (ii), we see that $E_1 \rightarrow 0$, as $n \rightarrow +\infty$.

$$\begin{aligned}
E_2 &= \left| G(\mathbf{z}) - \sum_{k=0}^{S_t} C_{S_t}^k [1 - \sum_{i=1}^J \pi_i z_i]^k (\sum_{i=1}^J \pi_i z_i)^{S_t-k} \prod_{j=1}^J [\hat{g}^{(j)}(1 - \frac{k}{n})]^{I_t^{(j)}} \right| \\
&= \left| G(\mathbf{z}) - \sum_{k=0}^{S_t} C_{S_t}^k [1 - \sum_{i=1}^J \pi_i z_i]^{S_t-k} (\sum_{i=1}^J \pi_i z_i)^k \prod_{j=1}^J [\hat{g}^{(j)}(1 - \frac{S_t-k}{n})]^{I_t^{(j)}} \right| \\
&= \left| G(\mathbf{z}) - \sum_{k=0}^{S_t} C_{S_t}^k [1 - \sum_{i=1}^J \pi_i z_i]^{S_t-k} (\sum_{i=1}^J \pi_i z_i)^k \prod_{j=1}^J [\hat{g}^{(j)}(\theta_{S_t} + \frac{k}{S_t} C_{S_t})]^{I_t^{(j)}} \right|
\end{aligned} \tag{12}$$

where, $\theta_{S_t} = \frac{n-S_t}{n} \rightarrow 0$ and $C_{S_t} = \frac{S_t}{n} \rightarrow 1$, when $n \rightarrow +\infty$.

According to the Bernstein's theorem [pro.2.3.1.2 Kissami [18]], $E_2 \rightarrow 0$ uniformly on $[0,1]$.

Finally, by applying the proposition 2.3.1.1 of Kissami [18], we show that $E_3 \rightarrow 0$ uniformly on $[0,1]$, This proves the lemma. \square

Let $\lambda = \sum_{j=1}^J \pi_j \hat{g}^{(j)'}(1)$ and denote \mathbf{T} the total number of descendants in the multitype Galton-Watson process initiated by m_j individual of type j where each individual of type j , $j = 1, \dots, J$, has descendants of type l according to a distribution with generating function $\hat{g}^{(j)}(1 - \pi_l(1 - z_l))$.

proposition 3.1.

\mathbf{T}_n converges in distribution towards \mathbf{T} . Moreover,

i) if $\lambda \leq 1$ the extinction probability is 1 and

ii) if $\lambda > 1$ extinction takes place with probability $\prod_{j=1}^J \rho_j^{m_j}$, and explosion with a probability $1 - \prod_{j=1}^J \rho_j^{m_j}$, where the vector ρ

is the smallest solution in $[0, 1]^J$ of the system of equations

$$z_j = \hat{g}^{(j)}\left(\sum_{i=1}^J \pi_i z_i\right), \quad j = 1, \dots, J. \quad (13)$$

iii) The generating function of \mathbf{T} is given by $\prod_{j=1}^J [\psi_j(\mathbf{z})]^{m_j}$, where $\mathbf{z} \in [0, 1]^J$ and the functions ψ_j , $j = 1, \dots, J$ are determined by the system of equations

$$\psi_j(\mathbf{z}) = \hat{g}^{(j)}\left[z_j \sum_{i=1}^J \pi_i \psi_i(\mathbf{z})\right], \quad j = 1, \dots, J. \quad (14)$$

Proof

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

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

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

$D_r(\mathbf{b}) = \{(\mathbf{i}_1, \dots, \mathbf{i}_r) \in (\mathbb{Z}_+^J)^r \text{ such that } \mathbf{i}_1 > \mathbf{0}, \dots, \mathbf{i}_r > \mathbf{0} \text{ and } \mathbf{i}_1 + \dots + \mathbf{i}_r = \mathbf{b}\}$. For each fix element $(\mathbf{i}_1, \dots, \mathbf{i}_r)$ of $D_r(\mathbf{b})$, we have

$$\begin{aligned} P[\mathbf{I}_1 = \mathbf{i}_1, \mathbf{I}_2 = \mathbf{i}_2, \dots, \mathbf{I}_r = \mathbf{i}_r, \mathbf{I}_{r+1} = \mathbf{0}] &= \\ P[\mathbf{I}_1 = \mathbf{i}_1] P[\mathbf{I}_2 = \mathbf{i}_2 / \mathbf{I}_1 = \mathbf{i}_1; S_1] \times & \quad (15) \\ P[\mathbf{I}_3 = \mathbf{i}_3 / \mathbf{I}_2 = \mathbf{i}_2; S_2] \times \dots \times P[\mathbf{I}_{r+1} = \mathbf{i}_{r+1} / \mathbf{I}_r = \mathbf{i}_r; S_r]. & \end{aligned}$$

By virtue of Lemma 3.1, each probability in (15) converges to its counterpart in the context of the multitype Galton-Watson process where the generating function of new infected due to one single infected individual from group j is given by $\hat{g}^{(j)}\left(\sum_{i=1}^J \pi_i z_i\right)$.

We can thus see that the probability of each term in (15) 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 $P(\cdot)$ is the distribution of the total progeny in such a process.

This proves the first statement of the proposition. Referring to the theory of multitype branching processes [see, e.g., Jagers [17]], we deduce the assertions i), ii) and iii). \square

4. RESULTS AND EXAMPLES OF STANDARD EPIDEMIC MODELS

The results showed, under certain conditions, that the final size of our epidemic process converges in distribution to the total number of descendants in a certain Galton-Watson process. We give some examples as special cases of our model.

4.1. The model of Pettigrew and Weiss [24]. The epidemic process of Pettigrew and Weiss (1967) is a continuous time Markov chain whose transitions are governed by the following rule: for all $t \in \mathbb{R}^+$, let $X(t)$, $Y_1(t)$ et $Y_2(t)$ the number of susceptible, carriers (type 1) and infected (type 2), respectively, present at time $t, t \in \mathbb{R}^+$. Initially, $[X(0), Y_1(0), Y_2(0)] = (n, m_1, m_2)$,

and given the state $[X(t), Y_1(t), Y_2(t)]$, four changes can occur during the time interval $(t, t+dt)$,

- the infection of a susceptible susceptible giving birth to a carrier with probability

$$\pi\beta X(t)[Y_1(t) + Y_2(t)]dt + o(dt),$$

- the infection of a susceptible giving birth to an infected with the probability

$$(1-\pi)\beta X(t)[Y_1(t) + Y_2(t)]dt + o(dt),$$

- the elimination of a carrier with the probability $\mu_1 Y_1(t)dt + o(dt)$,

- the elimination of an infected person with probability $\mu_2 Y_2(t)dt + o(dt)$,

where $0 \leq \pi \leq 1$, β , μ_1 et μ_2 are the rates of infection, elimination of a carrier and elimination of an infected respectively.

This process is a special case of our model. In this case, $J = 2$ and the infected of type i , $i = 1, 2$, contact the susceptible ones at the points of a Poisson process of parameter $\beta = \beta_1 = \beta_2$ and infection periods T_i , $i = 1, 2$ of exponential laws of parameters μ_i , $i = 1, 2$, respectively. Therefore

$$q^{(1)}(k) = E[\exp(-k\beta T_1)] = \frac{\mu_1}{\mu_1 + \beta k}, \quad (16)$$

$$q^{(2)}(k) = E[\exp(-k\beta T_2)] = \frac{\mu_2}{\mu_2 + \beta k}, \quad k \in [0, n].$$

The deterministic version of this model was examined by Isham (1988)[16] to describe the early stages of the spread of spread of AIDS. Here, the infected represent the infected persons who develop AIDS, and the carriers represent carriers represent the HIV-positive cases who do not develop AIDS. Downton's (1968)[6] model corresponds to the special case where the infected (type 2) are immediately detected and eliminated i.e. $\mu_2 = +\infty$.

Therefore, the infection is transmitted only by the carriers. Consequently

$$q^{(1)}(k) = E[\exp(-k\beta T_1)] = \frac{\mu_1}{\mu_1 + \beta k}, \quad (17)$$

$$q^{(2)}(k) = 1, \quad k \in [0, n].$$

When $\pi = 0$, the process is reduced to the Weiss (1967) model.

4.2. The model of Ball and Clancy [4]. In this model, each infected person of type j , $j = 1, \dots, J$, remains infectious for a period distributed as a random variable D_j , and during this period it can contact, independently of the others, any susceptible at points of a Poisson process of rate β_j . All these processes are independent; all random variables D_j are independent and independent of the contact process. It is clear that this process is a special case of the collective model. Therefore,

$$q^{(j)}(k) = E[\exp(-k\beta_j D_j)], \quad j = 1, \dots, J, \quad k \in [0, n]. \quad (18)$$

CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

REFERENCES

- [1] H. Andersson, A threshold limit theorem for a multitype epidemic model, Research report no. 165, Institute of Actuarial Mathematics and Mathematical Statistics, Stockholm University, (1993).
- [2] N.T.J. Bailey, The mathematical theory of infectious diseases and its applications, Griffin, London, (1975).
- [3] F. Ball, The threshold behaviour of epidemic models, J. Appl. Probab. 20 (1983), 227–241.
- [4] F. Ball, D. Clancy, The final outcome of an epidemic model with several different types of infective in a large population, J. Appl. Probab. 32 (1995), 579–590.
- [5] H. El Maroufy, D. Kiouach, T. Ziad, Final outcome probabilities for SIR epidemic model, Commun. Stat. - Theory Methods. 45 (2016), 2426–2437.
- [6] F. Downton, The ultimate size of carrier-borne epidemics, Biometrika. 55 (1968), 277–289.
- [7] A. Esegir, Distributions asymptotiques de la taille finale des processus épidémiques multivariés de type Reed-Frost collectif, Doctoral Theses, Université Mohammed premier, Oujda, (2004).
- [8] A. Esegira, A. Kissamia, H. El Maroufyb, T. Ziadc, A branching process approximation of the final size of a multitype collective reed-frost model, J. Stat. Appl. Probab. 2 (2013), 47–59.
- [9] L.-X. Feng, S.-L. Jing, S.-K. Hu, D.-F. Wang, H.-F. Huo, Modelling the effects of media coverage and quarantine on the COVID-19 infections in the UK, Math. Biosci. Eng. 17 (2020), 3618–3636.

- [10] J-P. Gabriel, Cl. Lefèvre and Ph. Picard, eds. Stochastic processes in Epidemic Theory, Lecture Notes in Biomath. 86, Springer, New York, (1990).
- [11] G. Giordano, F. Blanchini, R. Bruno, et al. Modelling the COVID-19 epidemic and implementation of population-wide interventions in Italy, *Nat. Med.* 26 (2020), 855–860.
- [12] A. Mouaouine, A. Boukhouima, K. Hattaf, N. Yousfi, A fractional order SIR epidemic model with nonlinear incidence rate, *Adv. Differ. Equ.* 2018 (2018), 160.
- [13] K. Hattaf, A new generalized definition of fractional derivative with non-singular kernel, *Computation.* 8 (2020), 49.
- [14] K. Hattaf, A.A. Mohsen, J. Harraq, N. Achtaich, Modeling the dynamics of COVID-19 with carrier effect and environmental contamination, *Int. J. Model. Simul. Sci. Comput.* 12 (2021), 2150048.
- [15] W.H. Hamer, Epidemic disease in England, *Lancet.* 1 (1906), 733-739.
- [16] V. Isham, Mathematical modelling of the transmission dynamics of hiv infection and aids: a review, *J. R. Stat. Soc. Ser. A. (Stat. Soc.)* 151 (1988), 5-30+44-49.
- [17] P. Jagers, Branching processes with biological applications, Wiley, Chichester, (1975).
- [18] A. Kissami, Problèmes d’urnes et processus épidémiques: modélisation et résultats asymptotiques. Doctoral Theses, ULB. Bruxelles, (1993).
- [19] A. Kissami, Cl. Lefèvre and Ph. Picard, On the realisation of n (partially) exchangeable events: a further look motivated by the modelling of epidemic processes. *Cahiers de C.E.R.O.* 36 (1995), 235-250.
- [20] G.G. Lorentz, Bernstein polynomials, Chelsea publishing company, New York, (1986).
- [21] A. Martin-Löf, Symmetric sampling procedures, general epidemic processes and their threshold limit theorems, *J. Appl. Probab.* 23 (1986), 265–282.
- [22] D. Mollison, G. Scalia-Tomba, J.A. Jacquez, eds. Spread of epidemics. stochastic modelling and data analysis, *Math. Biosci.* 107 (1991), 149-555.
- [23] L. Pang, S. Liu, X. Zhang, T. Tian, Z. Zhao, Transmission dynamics and control strategies of COVID-19 In Wuhan, China, *J. Biol. Syst.* 28 (2020), 543–560.
- [24] H.M. Pettigrew, G.H. Weiss, Epidemics with carriers: The large population approximation, *J. Appl. Probab.* 4 (1967), 257–263.
- [25] P. Picard, C. Lefevre, A unified analysis of the final size and severity distribution in collective Reed-Frost epidemic processes, *Adv. Appl. Probab.* 22 (1990), 269–294.
- [26] G. Scalia-Tomba, Asymptotic final-size distribution for some chain-binomial processes, *Advances in Applied Probability.* 17 (1985), 477–495.
- [27] G. Scalia-Tomba, Asymptotic final size distribution of the multitype Reed–Frost process, *J. Appl. Probab.* 23 (1986), 563–584.

- [28] T. Sellke, On the asymptotic distribution of the size of a stochastic epidemic, *J. Appl. Probab.* 20 (1983), 390–394.
- [29] B. Von Bahr and B. Von Bahr, A. Martin-Löf, Threshold limit theorems for some epidemic processes, *Adv. Appl. Probab.* 12 (1980), 319–349.
- [30] G.H. Weiss, On the spread of epidemics by carriers, *Biometrics.* 21 (1965), 481–491.
- [31] I. Yadegari, M. Omid, S.R. Smith, The herd-immunity threshold must be updated for multi-vaccine strategies and multiple variants, *Sci. Rep.* 11 (2021), 22970.