Article:
Gomez-Corral, A and Lopez Garcia, M orcid.org/0000-0003-3833-8595 (2017) On SIR epidemic models with generally distributed infectious periods: number of secondary cases and probability of infection. International Journal of Biomathematics, 10 (2). 1750024. ISSN 1793-5245

https://doi.org/10.1142/S1793524517500243

On SIR epidemic models with generally distributed infectious periods: number of secondary cases and probability of infection

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Abstract

Recently, Clancy [6] has shown how SIR epidemics in which individuals' infection periods are not necessarily exponentially distributed may be modeled in terms of a piecewise-deterministic Markov process. In this article, we present a more detailed description of the underlying piecewise-deterministic Markov process, from which we analyze the population transmission number and the infection probability of a certain susceptible individual.

Keywords: number of secondary cases, piecewise-deterministic Markov process, probability of infection, SIR epidemic model

1. Introduction

The SIR-model with general infectious period distribution has been recently revisited by Clancy [6], who introduces a family of martingales that may be used to determine the joint distribution of the number of survivors of the epidemic and the area under the trajectory of infectives. The SIR-model in [6] was previously analyzed using special constructions by Ball [3], and Picard and Lefèvre

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[14], and it is related to the dynamics of disease epidemics in a population where, at time t, individuals are classified into three categories: $S(t)$ susceptibles, $I(t)$ infectives and $R(t)$ removed individuals.

Kermack and McKendrick [11] analyzed in 1927 a general SIR-model for an homogeneous closed population of N individuals, where the infection and recovery rates of a given infective individual depend on the total time that this individual has been infected for. The analytical difficulties of addressing this general model lead in [11] to the consideration of a number of special cases. In particular, Special Case B in [11] addresses the particular situation where infection and recovery rates are constant, becoming the origin of the standard SIR-model; see [11, Equation (29)]. This model assumes that the population is homogeneously mixed, and the only possible events (Table 1) correspond to contacts between an infective and a susceptible, and the removal of an infective. The infection rate function $\lambda_{i,s}$ can be specified in infinitely many ways. For instance, the general stochastic epidemic (Bailey [2, Chapter 6]) is linked to the choice $\lambda_{i,s} = \lambda' is$, and it reflects that each infected individual makes contact with susceptibles according to a Poisson process of rate $\lambda' > 0$, and the contacted individual is chosen uniformly at random from amongst the susceptibles; Models 1 and 2 in Neuts and Li [13] are specified from the respective infection rates $\lambda_{i,s} = \lambda' i^{\alpha} s$ and $\lambda_{i,s} = \lambda' i \min\{s, \epsilon n\}$, where n is the initial number of susceptible individuals, the value $\alpha \in (0, 1)$ quantifies the degree of interaction between susceptibles and infectives, and the parameter ϵ specifies the fraction of susceptible population that is exposed to each infective; although an explicit formula for the contact rate is only obtained in special cases, the paper by Heesterbeek and Metz [10] shows how to derive, by a mechanistic approach, an expression for the saturating contact rate of individual contacts, and it contains applications to sexually transmitted diseases and marriage models; Saunders [16] analyzes the transmission of myxomatosis among rabbits by selecting the rates $\lambda_{i,s} = (i + s)^{-1/2} is$; for other infection rate functions, see [6, Section 3] and references therein.

In modeling infectious periods, the selection $\mu_i = \eta i$ amounts to the assump-

Events	Transitions	Rates
A new infection	$i \to i+1,\ s \to s-1,\ r \to r,$ for $i, s \in \mathbb{N}$	$\lambda_{i,s}$
A removal	$i \to i-1,\ s \to s,\ r \to r+1,$ for $i \in \mathbb{N}, s \in \mathbb{N}_0$	$\mu_i = \eta i,$ with $\eta > 0$

Table 1: Events, stochastic transitions and rates in the standard SIR-model (Kermack and McKendrick [11]) with exponentially distributed infectious periods

tion that, when a susceptible becomes infectious, its infectious period is exponentially distributed with expected value η^{-1}, this period being independent of all infectious periods associated with other currently infected individuals. This distributional assumption is made purely for mathematical convenience since it results in a time-homogeneous continuous-time Markov chain. In a more realistic setting, Clancy [6] shows how SIR-models in which individuals' infectious periods are not necessarily exponentially distributed may be described in terms of a piecewise-deterministic Markov process (PDMP, Davis [8]), which is defined on a general state space rather than a discrete state space. Specifically, Clancy [6] uses the general theory of PDMPs (Davis [8, 9]) as an alternative to the approach of Ball [3], and Picard and Lefèvre [14] in the study of the final outcome of SIR epidemics, including a variant in which the infection rate function $\lambda_{i,s}$ depends in a quite general way upon the current susceptible population size. The aim of this article is to present a description of the PDMP used by Clancy [6], which reflects the dynamics of SIR epidemics more transparently. It is also shown how this description may be appropriately applied to the analysis of the population transmission number and the infection probability of a marked susceptible individual, prior to the first removal.

2. The piecewise-deterministic Markov process framework

The interest is in the SIR-model with general infectious period distribution analyzed by Clancy [6, Sections 2-3], which is related to a closed, homogeneously mixed population decomposed into $S(t)$ susceptibles, $I(t)$ infectives, and $R(t)$

removed individuals, initially consisting of $m \in \mathbb{N}$ infectives and $n \in \mathbb{N}$ susceptible individuals. Individual's infectious periods are assumed to be distributed as any non-negative random variable X with probability distribution function $F(\cdot)$. This means that each infective remains so for a random time identically distributed as X, and it is then removed. At any time instant, infectious periods of all currently infected individuals are assumed to be mutually independent, and they are independent of the contact processes. We let $\lambda_{i,s}$ denote the infection rate function in the case of i infectives and s susceptible individuals.

Under the assumption that X is almost surely finite, Clancy [6] writes the state of the epidemic at time t by using the random variable $(S(t), I(t), \xi(t))$, where $\xi(t) = (\xi_1(t), ..., \xi_{I(t)}(t))$ and $\xi_i(t)$ is the time remaining until the removal of infective i, for $i \in \{1, ..., I(t)\}$. It is then seen that the domain of the extended generator of the resulting PDMP may be characterized from [9, Theorem 26.14], and the extinction times are almost surely finite regardless of the initial numbers of infectives and susceptible individuals. The objective here is to present a more detailed description of the underlying process analyzed in [6], which might be regarded as a more appropriate way to proceed for practical purposes; see [15, Section 2] for an alternative construction of the PDMP $\mathcal{X}$ from independent and identically distributed sequences of uniform random variables and a related simulation solution. For later use, we introduce some notation. In particular, the σ-algebra of Borel sets on the interval $(a, b) \subset (0, \infty)$ is denoted by $\beta(a, b)$, and β'_k is the Borel σ-algebra on the set $E^{(k)} = \{(z_1, ..., z_k) \in (0, \infty)^k : z_1 < ... < z_k\}$, for $k \in \mathbb{N}$. The Kronecker delta is denoted by $\delta_{a,b}$, and the function $1_A(z_1, ..., z_k)$ equals 1 if $(z_1, ..., z_k) \in A$, and 0 otherwise.

To begin with, we reformulate the state at time t in terms of the simplified random variable $(S(t); \xi_1(t), ..., \xi_{I(t)}(t))$, which leads us to a PDMP $\mathcal{X} = \{(S(t); \xi_1(t), ..., \xi_{I(t)}(t)) : t \geq 0\}$ defined on the state space

$$\mathcal{S}(m, n) \quad = \quad \mathcal{C}_0(n) \cup \mathcal{C}(m, n) \cup \partial\mathcal{C}(m, n),$$

where states in $\mathcal{C}_0(n) = \{s : 0 \leq s \leq n\}$ correspond to the ultimate extinction of the epidemic, and the sets $\mathcal{C}(m, n)$ and $\partial\mathcal{C}(m, n)$ are given by $\cup_{i=1}^{m+n} l(i; m, n)$

and $\bigcup_{i=1}^{m+n} \partial l(i;m,n)$, respectively, with $l(i;m,n) = \{(s;x_1,...,x_i) : 0 \leq s \leq \min\{n, m+n-i\}, 0 < x_1 < ... < x_i\}$ and $\partial l(i;m,n) = \{(s;0,x_2,...,x_i) : 0 \leq s \leq \min\{n, m+n-i\}, 0 < x_2 < ... < x_i\}$, for $1 \leq i \leq m+n$.

Differing from Davis [8], we characterize the dynamics of $\mathcal{X}$ by means of two transition measures $K_1(y;\cdot)$ and $K_2(y;\cdot)$ that govern transitions associated with the contact processes (states $y \in \mathcal{C}(m,n)$) and the removal of an infective (states $y \in \partial\mathcal{C}(m,n)$), respectively, and a flow function $\Phi_t(\cdot)$. To be concrete, the PDMP $\mathcal{X}$ changes deterministically according to a flow function $\Phi_t(\cdot)$ between two successive *basic transition*[1] instants, with $\Phi_t(y) = (s;x_1-t,...,x_i-t)$, for states $y \in \mathcal{C}(m,n)$ with $y = (s;x_1,...,x_i)$, and time instants $0 \leq t \leq x_1$. For states $y \in \mathcal{C}(m,n)$ with $y = (s;x_1,...,x_i)$, the transition measure $K_1(y;\cdot)$ is specified as follows:

(i) For sets $A \in \beta(0,x_1)$ and $B \in \beta'_i$,

$$K_1(y;\{s-1\} \times A \times B) \quad = \quad P_F(A)1_B(x_1,...,x_i).$$

(ii) For $1 \leq k \leq i-1$ and sets $A \in \beta'_k$, $B \in \beta(x_k,x_{k+1})$ and $C \in \beta'_{i-k}$,

$$K_1(y;\{s-1\} \times A \times B \times C) \quad = \quad 1_A(x_1,...,x_k)P_F(B)1_C(x_{k+1},...,x_i).$$

(iii) For sets $A \in \beta'_i$ and $B \in \beta(x_i,\infty)$,

$$K_1(y;\{s-1\} \times A \times B) \quad = \quad 1_A(x_1,...,x_i)P_F(B).$$

The transition measure $K_1(y;\cdot)$ captures the transition $y \to y'$, with $y' = (s';x'_1,...,x'_{i+1})$ and $s' = s-1$, and it is thus related to a new infection resulting in a new infectious period of length x –drawn from $F(\cdot)$–, which has to be added to the vector $(x_1,...,x_i)$ of remaining infectious times at the appropriate position to obtain a vector $(x'_1,...,x'_{i+1})$ with ordered entries.

[1] A transition of $\mathcal{X}$ is said to be *basic* as either the number $I(t)$ of infectives or the number $S(t)$ of susceptible individuals are appropriately modified.

In a similar manner, for $y \in \partial\mathcal{C}(m,n)$ with $y = (s; 0, x_2, ..., x_i)$ and sets $A \in \beta'_{i-1}$, the transition measure $K_2(y;\cdot)$ has the form

$$K_2(y; \{s\} \times A) \quad = \quad 1_A(x_2, ..., x_i),$$

thus capturing the transition $y \to y'$, with $y' = (s'; x'_1, ..., x'_{i-1})$, $s' = s$ and $(x'_1, ..., x'_{i-1}) = (x_2, ..., x_i)$.

To emphasize the relevance of the above description of $\mathcal{X}$, we next comment on its practical limitation. To simplify the discussion, we focus on the transient analogue to the final outcome of the epidemic (Clancy [6, Section 2]), and we assume that $F(\cdot)$ is a continuous function. For the initial state $y = (n; x_1, ..., x_m)$ with $0 < x_1 < ... < x_m$, let us define the time-dependent probabilities

$$P(t; y, \{s\}) \quad = \quad P\left(I(t) = 0, S(t) = s \,\middle|\, (S(0); \xi_1(0), ..., \xi_{I(0)}(0)) = y\right),$$

for values $0 \le s \le n$, which correspond to the event that the epidemic will die out before time t and the number of survivors will be equal to s. The reader is alerted to the fact that, although we omit the pair (m,n) by notational convenience, the probabilities $P(t; y, \{s\})$ depend on the initial numbers (m,n) of infective and susceptible individuals.

Theorem 1 *The transient probability $P(t; y, \{s\})$ is given by $P(t; y, \{s\}) = 0$ if $t < x_m$, and it can be evaluated iteratively as*

$$P(t; y, \{s\}) \quad = \quad P^{(n-s)}(t; y, \{s\}), \quad x_m \le t, \tag{1}$$

with $P^{(0)}(t; y', \{n'\}) = \exp\left\{-\sum_{k=1}^{m'} \lambda_{m'+1-k,n'} \left(x'_k - x'_{k-1}\right)\right\}$ as start values for states $y' = (n'; x'_1, ..., x'_{m'}) \in \mathcal{C}(m,n)$ if $x'_{m'} \le t$, and iterating by

$$\begin{aligned} & P^{(r)}(t; y', \{n' - r\}) \\ & \quad = \int_0^{x'_1} \lambda_{m',n'} e^{-\lambda_{m',n'} u} \int_{E^{(m'+1)}} P^{(r-1)}(t-u; y'', \{n' - r\}) K_1(y'; dy'') du \\ & \qquad + (1 - \delta_{1,m'}) e^{-\lambda_{m',n'} x'_1} \int_{E^{(m'-1)}} P^{(r)}(t - x'_1; y'', \{n' - r\}) K_2(y'; dy''), \end{aligned} \tag{2}$$

for integers $1 \le r \le n'$.

The proof of (1)-(2) is based on the use of the final size $n-s$ of the epidemic, in such a way that the next event specifies how to update the dynamics of the PDMP $\mathcal{X}$ in terms of the flow function $\Phi_t(\cdot)$ and the transition measures $K_1(y;\cdot)$ and $K_2(y;\cdot)$. More particularly, the value $P^{(r)}(t;y',\{n'-r\})$ in (2) corresponds to the conditional probability that, given that the initial state is $y'=(n';x'_1,...,x'_{m'})\in\mathcal{C}(m,n)$ with $x'_{m'}\leq t$, the epidemic dies out before time t with $n'-r$ susceptible individuals ($I(t)=0$, $S(t)=n'-r$) and the number of infections taking place during $(0,t]$ equals r. This means that a new infection occurring at an arbitrary time $u\in(0,x'_1)$ implies that $r-1$ infections have to be recorded in the residual interval $(u,t]$, and the removal of an infective at time x'_1 before any infection taking place implies that r infections have to be registered during $(x'_1,t]$. Then, the use of $\Phi_t(\cdot)$ and $K_1(y;\cdot)$ yields

$$\begin{aligned}
&\int_{E^{(m'+1)}} P^{(r-1)}(t-u;y'',\{n'-r\})K_1(y';dy'') \\
&\quad = \int_0^{x'_1-u} P^{(r-1)}(t-u;(n'-1;v,x'_1-u,...,x'_{m'}-u),\{n'-r\})F(dv) \\
&\qquad + \int_{x'_1-u}^{x'_2-u} P^{(r-1)}(t-u;(n'-1;x'_1-u,v,x'_2-u,...,x'_{m'}-u),\{n'-r\})F(dv) \\
&\qquad + ... \\
&\qquad + \int_{x'_{m'-1}-u}^{x'_{m'}-u} P^{(r-1)}(t-u;(n'-1;x'_1-u,...,x'_{m'-1}-u,v,x'_{m'}-u),\{n'-r\})F(dv) \\
&\qquad + \int_{x'_{m'}-u}^{\infty} P^{(r-1)}(t-u;(n'-1;x'_1-u,...,x'_{m'}-u,v),\{n'-r\})F(dv), \qquad (3)
\end{aligned}$$

and, according to $K_2(y;\cdot)$, it is readily seen that

$$\begin{aligned}
&\int_{E^{(m'-1)}} P^{(r)}(t-x'_1;y'',\{n'-r\})K_2(y';dy'') \\
&\qquad = P^{(r)}(t-x'_1;(n';x'_2-x'_1,...,x'_{m'}-x'_1),\{n'-r\}). \qquad (4)
\end{aligned}$$

At first sight, Eqs. (1)-(2) govern the dynamics of $P(t;y,\{s\})$ for the initial state $y=(n;x_1,...,x_m)$ with $0<x_1<...<x_m$ and values $0\leq s\leq n$, and analytical expressions for $P(t;y,\{s\})$ might be naturally derived from them by implementing two steps. Specifically, we should first define $P^{(0)}(t;y,\{s\})$ as a function of time t and remaining infectious periods $x'_1,...,x'_{m'}$, for every state

$y' \in \mathcal{C}(m,n)$ with $y' = (n'; x'_1, ..., x'_{m'})$ and integers $1 \leq m' \leq m + n - n'$ and $0 \leq n' \leq n$; then, by increasing r from 1 to n, we should evaluate analytically the probability $P^{(r)}(t; y, \{s\})$ from (2), jointly with (3)-(4), as a function of t and values $x'_1, ..., x'_{m'}$, for every state $y' \in \mathcal{C}(m,n)$ with $y' = (n'; x'_1, ..., x'_{m'})$ and integers $1 \leq m' \leq m + n - n'$ with $r \leq n' \leq n$. Unfortunately, this approach does not appear to lead us to analytical expressions in the case of concrete specifications of $F(\cdot)$, such as uniform, exponential, Erlang and gamma laws, among others. Instead one may try to derive a numerical solution of (1)-(4) by using numerical integration, but it is seen that general-purpose numerical integration procedures do not perform well with regard to both accuracy and speed, with the exception of small values of $m+n$. There are two reasons for this. First, the number of function evaluations needed to compute the iterated integrals in (2) increases as the 2nd power of the number needed to evaluate a one-dimensional integral. Second, the upper and lower limits of the one-dimensional integrals in (3) depend on the remaining infections times $(x'_1, ..., x'_{m'})$ and the integration variable u. Therefore, the underlying combinatorial explosion in (2)-(4) and storage requirements turn the numerical integration problem into intractable for practical use.

It is evident that, in solving (1)-(4), the computational load is inherently related to the definition of $P(t; y, \{s\})$, which at time t forces us to record the times $\xi_1(t), ..., \xi_{I(t)}(t)$ remaining until the removal of all currently infected individuals, and update appropriately these values between successive basic transition instants (flow function $\Phi_t(\cdot)$), and according to either the contacts between an infective and a susceptible (transition measure $K_1(y; \cdot)$, for states $y \in \mathcal{C}(m,n)$), or the removal of an infective (transition measure $K_2(y; \cdot)$, for states $y \in \partial\mathcal{C}(m,n)$). In this sense, it is important to point out that numerical integration is however of particular interest when, in studying a specific descriptor, the underlying arguments require the use of a single remaining infectious period $\xi_{i^*}(t)$, for a suitably chosen index $i^* \in \{1, ..., I(t)\}$; in Section 3, this is related to the next removal of an infective, which is linked to the case $i^* = 1$.

3. Number of secondary cases and probability of infection

The population transmission number R_p, as discussed in [1, Section 4], is defined as the number of secondary cases produced by all currently infectives prior to the first removal. An important feature of R_p is that, unlike the basic reproduction number $\mathcal{R}_0$ which is related to the *time of invasion* (i.e., $(I(0), S(0)) = (1, N-1)$ and $R(0) = 0$ for a community of N individuals), the descriptor R_p can be appropriately evaluated at every time instant and, more importantly, it is defined as a random variable instead of an expected value.

The population transmission number plays an important role in the design of control strategies, both *preventive* and *responsive*, in order to limit the spread of an epidemic. In those situations in which healthcare decision makers become aware of the epidemic after the first removal occurs (for example, once the first death takes place), the analysis of R_p allows one to measure how fast the disease propagates until its first detection. More concretely, let us consider an invasion time and define $T > 0$ as the time until the first removal occurs. Then, responsive strategies can be put in place from time T, affecting the spread dynamics represented by $(I(t), S(t), R(t))$ for time instants $t \in [T, \infty)$, with initial conditions given by $I(T) = R_p$, $S(T) = N - R_p - 1$ and $R(T) = 1$. Large values of R_p correspond to situations where responsive strategies can be implemented only once a large number of individuals have been infected. In these situations, preventive strategies that do not require for detection of the disease should prevail; see, for example, the paper by López-García [12] where the efficacy of preventive (room configuration design of the unit) and responsive (isolation of patients) strategies is analyzed by means of a SIR-model on an heterogeneous population for the spread of antibiotic resistant bacteria in an intensive care unit. We refer the reader to the paper [4], where the efficacy of responsive strategies (vaccination and isolation of individuals after the first removal occurs) is analyzed for a SEIR-model in a population partitioned into households; this analysis was extended by Ball et al. [5] by including imperfect vaccination, latent individuals being also vaccine-sensitive and both

constant and exponential infectious and latent periods. It is also worth to mention the work carried out in [7] where a SIR-model is considered under the assumption that the infection transmission rate decreases after three days of the first removal, representing the effect of the implementation of a responsive strategy.

It is clear that the probability distribution of R_p depends on the initial state $y = (n; x_1, ..., x_m)$ with $m, n \in \mathbb{N}$ and $0 < x_1 < ... < x_m$, but only in terms of the numbers m and n of infectives and susceptibles, and the smallest value x_1 amongst remaining infectious periods. To simplify notation, we thus reformulate states $y = (n; x_1, ..., x_m)$ of the PDMP $\mathcal{X}$ in the form $\hat{y} = (m, n; x_1)$.

Theorem 2 *The conditional probabilities*

$$P_r(m, n; x_1) \quad = \quad P(R_p = r|(I(0), S(0); \xi_1(0)) = (m, n; x_1))$$

can be expressed as $P_0(m, n; x_1) = e^{-\lambda_{m,n} x_1}$ *and*

$$\begin{aligned} P_r(m, n; x_1) \quad = \quad & \int_0^{x_1} \lambda_{m,n} e^{-\lambda_{m,n} u} \Bigg(\int_0^{x_1 - u} P_{r-1}(m+1, n-1; v) F(dv) \\ & + (1 - F(x_1 - u)) P_{r-1}(m+1, n-1; x_1 - u) \Bigg) du, \end{aligned} \tag{5}$$

for integers $1 \leq r \leq n$. *Moreover, under the assumption that initial sizes are given by* $(I(0), S(0)) = (m, n)$ *with initial remaining infectious periods* $0 < x_1 < ... < x_m$, *the conditional probability* $Q(m, n; x_1)$ *that a marked susceptible becomes infective prior to the first removal satisfies*

$$Q(m, n; x_1) \quad = \quad \frac{1}{n} E[R_p|(m, n; x_1)], \tag{6}$$

where $E[R_p|(m, n; x_1)] = \sum_{r=1}^{n} r P_r(m, n; x_1)$ *is the expectation of* R_p, *conditioned on* $(S(0); \xi_1(0), ..., \xi_{I(0)}(0)) = (n; x_1, ..., x_m)$.

Proof It is first noted that, provided that a new infection occurs at time u with $u \in (0, x_1)$, the first term in (5) corresponds to an infectious period v for the new infective that is less than $x_1 - u$, which means that the smallest remaining infectious period $x_1 - u$ has to be replaced by v in our further arguments.

The second term captures the event that the infectious period v is greater than the time instant $x_1 - u$ of the next removal. Eq. (6) can be analytically derived by decomposing the infection rate function $\lambda_{i,s}$ into two contributions $s^{-1}\lambda_{i,s} + (1 - s^{-1})\lambda_{i,s}$, where the former amounts to the infection of the marked susceptible, and the latter is related to the infection of another susceptible individual. Similarly to (5), it is seen that, starting with $Q(m, 1; x_1) = 1 - e^{-\lambda_{m,1} x_1}$, the probabilities $Q(m, n; x_1)$ with $n \geq 2$ can be evaluated iteratively from

$$\begin{aligned} Q(m,n;x_1) &= \frac{1}{n}\left(1 - e^{-\lambda_{m,n}x_1}\right) \\ &\quad + \left(1 - \frac{1}{n}\right)\left(\int_0^{x_1}\left(1 - e^{-\lambda_{m,n}(x_1 - v)}\right) Q(m+1, n-1; v) F(dv)\right. \\ &\quad \left. + \int_0^{x_1} \lambda_{m,n} e^{-\lambda_{m,n} u} Q(m+1, n-1; x_1 - u)(1 - F(x_1 - u))du\right). \end{aligned} \tag{7}$$

Then, Eq. (6) is readily obtained by multiplying (5) by r and summing over the integer $r \in \{1, ..., n\}$, since $Q(m, 1; x_1) = E[R_p|(m, 1; x_1)]$ and the resulting expressions for $n^{-1}E[R_p|(m, n; x_1)]$, for $n \geq 2$, are identical to the iterative scheme in (7). $\square$

Probability $Q(m, n; x_1)$ is the individual counterpart of the population descriptor R_p, and it represents the risk for an initially marked susceptible individual to become infected until the first removal occurs. In those scenarios in which the first removal amounts to the detection of the disease, leading to the potential implementation of responsive strategies, probability $1 - Q(m, n; x_1)$ needs to be interpreted as the probability of the marked individual being susceptible once these responsive strategies are put in place, becoming an individual measure of the risk of infection until detection of the epidemic.

For illustrative purposes, we next focus on SIR-models at an invasion time (i.e., $I(0) = 1$) with $S(0) = 20$ and $x_1 = E[X]$, and we assume the infection rate function $\lambda_{i,s} = \lambda' is$, for states $(i, s) \in \mathcal{C}(1, 20)$. We consider three scenarios defined by infectious periods distributed according to an Erlang law (with two phases), an exponential law, and a gamma law (its shape parameter equals 0.5),

with expected values $E[X] = 1.0$. In our examples, the exponential assumption for infectious periods yields standard SIR-models, and it is a natural boundary between the Erlang case (with squared coefficient of variation $c_X^2 = 0.5$) and the gamma case ($c_X^2 = 2.0$), which are commonly considered *low-variance* and *high-variance*, respectively.

Figure 1 illustrates the effect of the infectious period distribution on the mass function of the population transmission number R_p for (from top to bottom) per capita contact rates $\lambda' \in \{0.05, 0.1, 0.2\}$. It is seen that distinct infectious period distributions result in identical values of the probability $P_0(1, 20; x_1)$, since this probability amounts to the probability of no infections occurring before x_1 units of time. It is also observed that, regardless of λ' and concrete specifications for $F(\cdot)$, the mass function of R_p always exhibits a *unimodal* shape, but magnitudes are noticeably distinct when λ' increases. More particularly, the gamma case always leads us to probability distributions of R_p concentrated within smaller values of r, whereas the distribution of R_p becomes heavy-tailed in the Erlang case. This means that low- and high-variance assumptions for infectious periods yield epidemics that, in comparison with standard SIR-models, spread faster and slower, respectively; by (6), it is clear that Figure 2 corroborates this assertion in terms of mean values.

Under a situation where detection of the disease amounts to the occurrence of the first removal, our numerical results suggest that, for the parameter values under consideration, the Erlang distribution for the recovery times leads to higher values of R_p, followed by the exponential and the gamma distributions; note that $E[R_p] = 1.2946$, 1.1631 and 1.0230 for the Erlang, exponential and gamma distributions, respectively, in the case $\lambda' = 0.05$ (Figure 1, *top*). This translates into the fact that, if recovery times follow the Erlang distribution analyzed in Figure 1, responsive strategies would be implemented once, in average, 1.2946 individuals have already become infected, while this number reduces to 1.0230 under the gamma distribution. Thus, one should expect responsive strategies to be more effective in the second case. Differences in $E[R_p]$ significantly increase with λ', where $E[R_p] = 6.9182$, 4.7467 and 3.1020 for the Erlang,

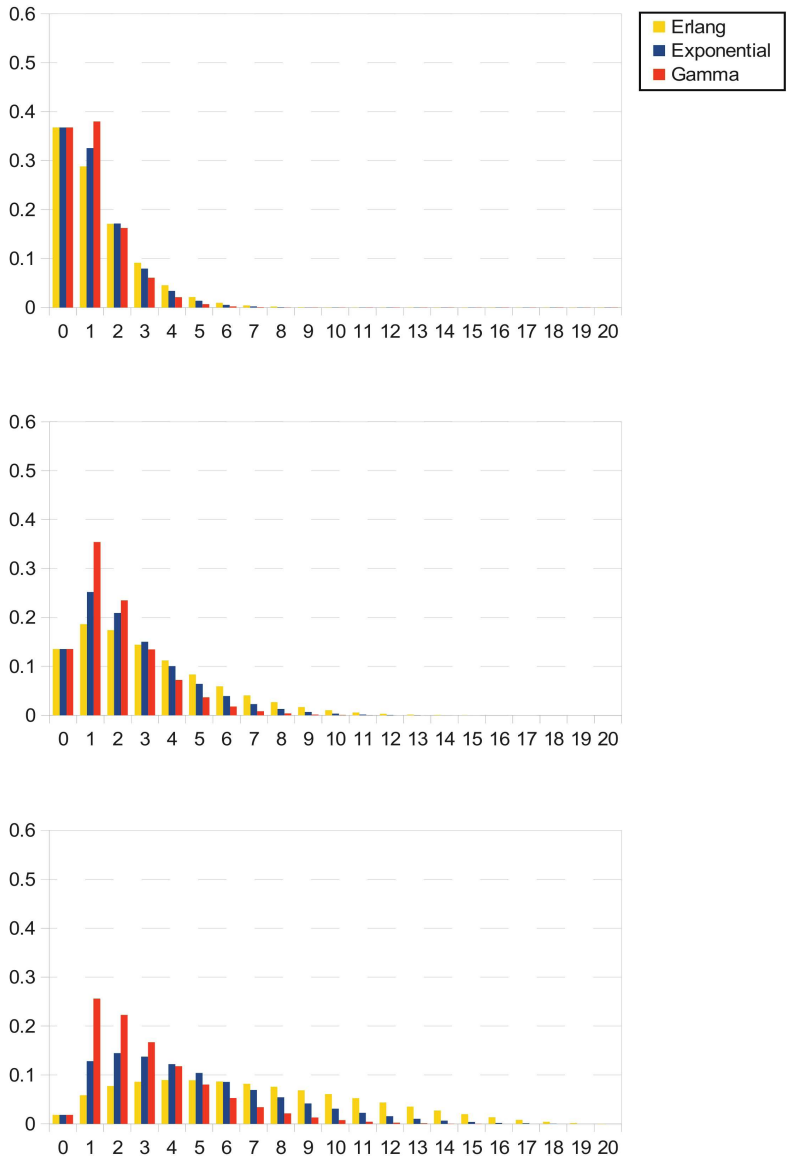

Figure 1: The mass function of R_p at an invasion time, for SIR-models with infection rate function $\lambda_{i,s} = \lambda' is$ and (from *top* to *bottom*) per capita contact rate $\lambda' = 0.05$, 0.1 and 0.2.

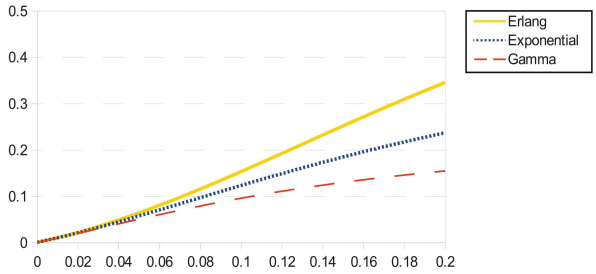

Figure 2: The conditional probability $Q(m, n; x_1)$ that a marked susceptible becomes infected prior the first removal, at an invasion time versus the per capita contact rate λ', for SIR-models with infection rate function $\lambda_{i,s} = \lambda' is$.

exponential and gamma distributions, respectively, for $\lambda' = 0.2$ (Figure 1, *bottom*). Thus, while responsive strategies might be effective under the gamma distribution (these could be implemented once 3.1020 individuals have already become infected in average), preventive strategies might be needed under the Erlang case, where detection of the disease occurs once 6.9182 individuals have already become infected in average.

Similar comments regarding the interpretation of our results in relation to the time until the first detection of the disease could be made for descriptor $Q(m, n; x_1)$ in Figure 2. Note that, in Figure 2, the interest is in the conditional probability that a marked susceptible becomes infected prior to the first removal which, as intuition tells us, behaves as an increasing function of λ'; for every fixed λ', its smallest and highest values are associated with the gamma and Erlang cases, respectively. In terms of the coefficient of variation (Figure 3), the variability of R_p in the Erlang and gamma cases is not essentially different from standard SIR-models, regardless of λ'. In Figure 3, it is observed that the variability of R_p increases as λ' tends to zero, which is associated with the situation when the number of contacts between an infective and a susceptible, taking place before the smallest remaining infectious period expires, becomes negligible.

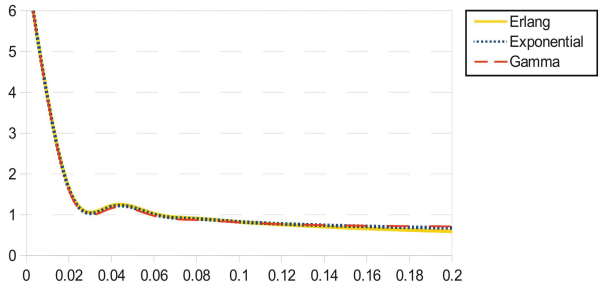

Figure 3: The variation coefficient of R_p at an invasion time versus the per capita contact rate λ', for SIR-models with infection rate function $\lambda_{i,s} = \lambda' is$.

4. Conclusion

In this article, we have relaxed the standard assumption of exponentially distributed infectious periods in SIR-models and, similarly to Clancy [6], we have translated the resulting SIR-model into a PDMP $\mathcal{X}$. The key elements (Section 2) have been a flow function describing how $\mathcal{X}$ changes deterministically between basic transition instants, and two transition measures updating states of the process $\mathcal{X}$ when either an infective makes contact with a susceptible individual or the removal of an infective occurs. An advantage of these elements lies in their straightforward use when, in studying a descriptor, a single infectious period is required from an analytical perspective, which is the case of the population transmission number R_p and the infection probability of a marked susceptible, prior to the first removal (Section 3).

Descriptors R_p and $Q(m, n; x_1)$ represent population and individual alternatives, respectively, for measuring the propagation potential of the epidemic. A particular feature of R_p is that it is defined as a random variable, instead of the usual definition of the basic reproduction number, R_0, as an average value; see the paper [1]. In the particular situation in which detection of the disease occurs after the first removal [4, 5], the analysis of R_p carried out here is crucial in order to identify the number of infectives present in the population once responsive strategies can be put in place, while $Q(m, n; x_1)$ is an individual

measure of the risk of a susceptible individual to become infected before these responsive strategies can be implemented.

In a more general setting (specifically, when computing time-dependent probabilities in Theorem 1), the effectiveness of our approach has been shown to be limited as a result of the amount of latent information imputed in the underlying analysis; in showing this practical drawback, we have focused on a transient version of the final outcome of the epidemic, whose analytical treatment needs all remaining infectious periods. Some aspects of this intricate problem have been outlined in the present work, but arguably more research is required in this area.

Acknowledgements

This work was supported by the Government of Spain (Ministry of Economy and Competitiveness) and the European Commission, project MTM2011-23864. The authors are members of the "Stochastic Modelling Group, UCM-910211", which was supported by the Complutense University of Madrid and Banco de Santander, call 2014-GR3/14. M. López-García is also supported by The Leverhulme Trust, project RPG-2012-772.

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