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The early stage behaviour of a stochastic SIR epidemic with term-time forcing

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Abstract

The general stochastic SIR epidemic in a closed population under the influence of a term-time forced environment is considered. An “environment” is in this context any external factor that influences the contact rate between individuals in the population, but is itself unaffected by the population. Here “term-time forcing” refers to that the changes in contact rate are discontinuous but cyclic. The inclusion of such an environment into the model is done by replacing a single contact rate λ with a cyclically alternating renewal process with k different states denoted $\{\Lambda(t)\}_{t \geq 0}$. Threshold conditions in terms of R_* are obtained, such that $R_* > 1$ implies that π , the probability of a large outbreak, is strictly positive. Examples when it is possible to compute π are given and from these examples the impact of the distribution of the time periods that $\Lambda(t)$ spends in its different states is clearly seen.

KEY WORDS: Stochastic epidemic; Branching process in seasonal environment; Seasonal forcing; Term-time forcing; Threshold conditions.

MSC2000: 92D30, 60J80

1 Introduction

In the present paper we focus on infectious diseases of SIR type, where SIR is an abbreviation for susceptible, infectious and recovered (and immune). These are consequently the only possible states that an individual can belong to when we discuss SIR type diseases, and the possible transitions between these states follows $S \rightarrow I \rightarrow R$.

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In the classical general stochastic SIR epidemic, see e.g. [3, Ch. 2.3, p. 14], many unrealistic assumptions are made. For example one assumes that the population is closed, that all individuals mix homogeneously, that the contact rate is constant etc. As a consequence of this, many modifications of this model have been introduced and analysed in the literature, where different assumptions have been relaxed, see e.g. [3] and references therein. In the present paper we relax the assumption that the rate of contact between individuals is constant over time, and we are particularly interested in the situation where the contact rate changes following some cyclic pattern. An example of this is how school children interact during school terms and holidays respectively. This effect on the contact rate is quantified as a seasonally varying environment. The term “environment” in this context is any external factor that influences the rate with which individuals make contact in the population, but is itself independent of the population. For many viral and bacterial diseases seasonal incidence patterns have been observed, and measles is a striking example where the seasonal incidence patterns seems to be highly correlated with seasonal contact patterns, see e.g. [2, pp. 44, 46, 136-137]. The topic of seasonality in connection to epidemics have been addressed in several papers, where some of the more recent are [19] and [13], and an earlier reference is [22]. These papers are concerned with endemic diseases. In [21] they consider a closed population SIR model in a seasonally varying environment using a deterministic approach and give numerical examples. Related issues can also be found in the area of epizootics, see e.g. [1] and references therein. In common for all these references are that they consider seasons following some oscillating pattern, i.e. modelling the seasonality using sinusoids (so-called “seasonal forcing”, see e.g. [18]). Another type of seasonality that has been considered is the so-called “term-time forcing”, see e.g. [11], [18], [23] and references therein. In this situation the contact rate changes discontinuously, but cyclically, similar to what is natural to believe that happens in the holiday/term example for schoolchildren from above. In the present paper we focus on a situation similar to term-time forcing. That is, we let the environment change cyclically corresponding to that the contact intensities make jumps at (non-)random time points.

The aim of the present paper is to get a better understanding for how term-time forced environments affects a newly introduced disease’s ability to take off. We make a simple modification of the general stochastic SIR epidemic that takes environmental effects into account, and derive threshold conditions for this model. These conditions are expressed in terms of R_* , loosely defined as the expected number of individuals that are infectious at the time point when an entire environmental cycle is completed, given that the epidemic was started by a single infective. The quantity R_* relates to R_0 , the so-called basic reproduction number. The basic reproduction number is defined as the average number of susceptible individuals that a typical infectious individual infects in an otherwise disease-free population during its infectious period. In more detail: A large outbreak can only occur when $R_0 > 1$, see e.g. [3, Thm. 3.1, Ch. 3.3, p. 25]. From our main result, Theorem 3.1, we show that R_* possess the same threshold property.

We consider environments that change cyclically between k different states, and where

the time that the environment spends in any state may be either random or non-random. For the special case when the time periods that the environment spends in its different states are exponentially distributed, we show how this system can be represented as a multi-type epidemic, and show for $k = 2$ that R_0 and R_* are equivalent in terms of threshold behaviour. Moreover, we show that if this kind of system is approximated by a general stochastic SIR epidemic with a single contact rate equal to the time-mean environmental effect on the rate of contacts between individuals, the distributions of the time that the environmental process spends in its different states are of importance. More precisely, we show that R_* of the actual process with environment included is always greater than the corresponding R_* of the approximated system, except when the time periods that the environment spends in its different states are non-random. For this situation the two thresholds are equal. This is quantified in Corollary 3.2. The implication is hence somewhat disappointing from an applied point of view where one rather would see that this kind of approximation would yield an epidemic which takes off easier than the actual one. In fact, in connection with the statement of Theorem 3.1 in Section 3 we give an example where R_* of the approximated system is strictly smaller than 1 whereas R_* of the actual process is strictly greater than 1.

A short outline of our analysis can be formulated as follows: First a coupling between a suitable branching process with a varying environment and the epidemic process with a varying environment is established. Then, by using that the approximating process analysed at the time points when entire environmental cycles are completed behaves as a Galton-Watson branching process, the desired results follows from standard results from branching process theory.

The disposition of this paper is as follows: In Section 2 and 3 we define the model and state and discuss our main results. Section 4 is devoted to the coupling between the number of infectious individuals in our epidemic process and the approximating branching process and the proof of Theorem 3.1. Section 5 is concerned with some important examples, and we conclude with a discussion in Section 6.

2 The general stochastic SIR epidemic in a term-time forced environment

In the general stochastic SIR epidemic, see [3, Ch. 2.3, pp. 14-15], a closed homogeneous population is considered, where all infectious contacts are made according to a Poisson process with intensity λ , and where infectious individuals recovers, independently of everything else, at rate γ . Denote this process by $\{(S^{(n)}(t; \lambda, \gamma), I^{(n)}(t; \lambda, \gamma))\}_{t \geq 0}$, where n refers to the size of the initially susceptible population. Note that $R(t; \lambda, \gamma)$, the number of recovered (and immune) individuals at t , is superfluous since the population is closed, i.e. $S^{(n)}(t; \lambda, \gamma) + I^{(n)}(t; \lambda, \gamma) + R(t; \lambda, \gamma) = n + 1$. Unless otherwise stated we will throughout assume $(S^{(n)}(0; \lambda, \gamma), I^{(n)}(0; \lambda, \gamma)) = (n, 1)$ for notational convenience. As described in

| From | To | Rate |
|----------|------------------|---------------------------|
| (s, i) | $(s - 1, i + 1)$ | $\frac{\Lambda(t)}{n} si$ |
| (s, i) | $(s, i - 1)$ | γi |

Table 1: The general stochastic SIR epidemic in a term-time forced environment. Here $S^{(n)}(t; \Lambda(t), \gamma) = s$ and $I^{(n)}(t; \Lambda(t), \gamma) = i$ denote the number of susceptible and infectious individuals at time t respectively, and $\Lambda(t)$, $\Lambda \in \{\lambda_1, \dots, \lambda_k\}$, is the intensity process that changes state depending on the environment. If not stated otherwise we assume that $(S^{(n)}(0; \Lambda(0), \gamma), I^{(n)}(0; \Lambda(0), \gamma)) = (n, 1)$ and $\Lambda(0) = \lambda_1$.

the introduction, the environment only affects the intensity with which individuals make contact, and the environmental changes are cyclic. We can hence describe our model as follows: When the environment is in state i the rate at which individuals makes contact will be λ_i . Further, the time periods that the environment spends in state i are distributed as the (non-)random variable T_i , and then changes to state $i + 1$. Due to the cyclic behaviour of the environmental process, this will correspond to that the contact rates will change according to $\lambda_1 \rightarrow \lambda_2 \rightarrow \dots \lambda_k \rightarrow \lambda_1 \rightarrow \dots$, when there are k different environmental states. Thus, by replacing a single λ by a cyclically varying intensity process $\{\Lambda(t)\}_{t \geq 0}$ ($\Lambda(0) = \lambda_1$), where $\Lambda(t) = \lambda_i$ if the environmental process is in state i ($i = 1, \dots, k$) at t , the general stochastic SIR epidemic can account for environmental effects, see Table 1. That is, our new epidemic process is $\{(S^{(n)}(t; \Lambda(t), \gamma), I^{(n)}(t; \Lambda(t), \gamma))\}_{t \geq 0}$. Henceforth, we will make no distinction between the environmental process and the intensity process $\Lambda(t)$. If we summarise our restrictions on the environmental process, we will throughout assume that

- (a) it is piece-wise constant, i.e. $\Lambda(t) \in \{\lambda_1, \dots, \lambda_k\}$.
- (b) it changes cyclically between its states.
- (c) the time periods that $\Lambda(t)$ spends in a state i are i.i.d. and distributed as the random variables T_i .
- (d) the time periods that $\Lambda(t)$ spends in different states are independent.

In the next section we state and discuss our main result which describes how a term-time forced environment affects the possibility of a large outbreak. The notion of large outbreak is here equivalent to that the number of ultimately infected individuals will tend to infinity when passing to the limit in n .

3 Results

We now define a number of quantities needed in the formulation of our main result, Theorem 3.1. These quantities will be explained in more detail in Section 4.

Recall from the previous section that T_i is a stochastic variable distributed as the typical time that the environmental process spends in state i . If we denote the moment generating function of T_i (T_i is non-negative) by

$$\Psi_i(s) := \mathbb{E} \left[e^{sT_i} \right] = \int_0^\infty e^{st} f_{T_i}(t) dt, \quad (3.1)$$

which is convergent if there exists some $h > 0$ such that $s < h$ (otherwise $\Psi_i(s) := +\infty$). Note that the integral in (3.1) always is convergent for $s < 0$, but that $\Psi_i(s)$ then loses its interpretation as a moment generating function. We will throughout use statements with negative s without further comments.

Further, let $\xi_i(s, t)$ denote the generating function of $X(t; \lambda_i, \gamma)$, which is defined as a linear birth and death process with birth rate λ_i and death rate γ . Then it can be shown that

$$\xi_i(s, t) := \mathbb{E}[s^{X(t; \lambda_i, \gamma)}] = \frac{\gamma(s-1) - e^{(\gamma-\lambda_i)t}(\lambda_i s - \gamma)}{\lambda_i(s-1) - e^{(\gamma-\lambda_i)t}(\lambda_i s - \gamma)}, \quad |s| \leq 1, \quad t \geq 0, \quad (3.2)$$

see e.g. [5, Ch. III.5, p. 109]. Further, define

$$\chi(s) := \mathbb{E}[\xi_1(\xi_2(\xi_3(\cdots(\xi_k(s, T_k), T_{k-1}), \cdots), T_3), T_2), T_1)], \quad |s| \leq 1. \quad (3.3)$$

Moreover, let ρ be the smallest non-negative solution to the equation

$$s = \chi(s). \quad (3.4)$$

We can now state our main result:

Theorem 3.1. *Let $\{(S^{(n)}(t; \Lambda(t), \gamma), I^{(n)}(t; \Lambda(t), \gamma))\}_{t \geq 0}$ denote an epidemic process in a term-time forced environment defined by the rates in Table 1 and let everything else be defined as above. Moreover, let $E^{(n)} := n - S^{(n)}(\infty; \Lambda(\infty), \gamma)$. Then R_\star defined by*

$$R_\star = \prod_{i=1}^k \Psi_i(\lambda_i - \gamma), \quad (3.5)$$

works as a threshold, such that $E^{(n)} \rightarrow \infty$ as $n \rightarrow \infty$ with probability $\pi := 1 - \rho$, where $\pi > 0$ if and only if $R_\star > 1$.

The proof of Theorem 3.1 is rather lengthy and is given in Section 4.

Remark 3.1. As will become apparent later, the epidemiological interpretation of R_\star is that it corresponds to the average number of individuals that are *infectious* at the time point when an environmental cycle is completed, given that the epidemic was initiated by a single infectious individual whose infectious period starts at the time point when a new environmental cycle is started. In Remark 4.2 after the proof of Theorem 3.1 it will become clear why this threshold should be expressed in terms of infectious rather than infected individuals. By inspecting the above theorem we see that it is relatively easy to compute R_\star , at least with the aid of a computer. The value of R_\star tells us whether the probability of a large outbreak is strictly greater than zero or not. Unfortunately the actual probability π , is much harder to compute. In Section 5 we discuss two special cases where π is possible to compute. Further, if we only have one environmental state, then $T = +\infty$, and $R_\star = +\infty$ if $\lambda - \gamma > 0$, and $R_\star = 0$ otherwise. For the general stochastic SIR epidemic with a single environment $R_0 = \lambda/\gamma$, see e.g. [3, Ch. 2.1, p. 12], and the threshold property of R_\star is hence preserved since $R_0 > 1$ is equivalent to $R_\star = +\infty$. Moreover, from (3.1) it is clear that $\Psi_i(s) > 1$ if $s > 0$, and $\Psi_i(s) \leq 1$ otherwise. Thus, if $\lambda_i > \gamma$ for $i = 1, \dots, k$, then $R_\star > 1$, and if $\lambda_i \leq \gamma$, then $R_\star \leq 1$. Consequently, the remaining cases are those where $\lambda_i > \gamma$ holds for some i , and $\lambda_i \leq \gamma$ for some other i .

Suppose that one would try to approximate this type of system. One way would be to calculate the long-time average environmental effect, $\tilde{\lambda}$, and use this single rate in the general SIR epidemic. Postponing the calculation of $\tilde{\lambda}$ for a moment, assuming that it is possible to obtain, we get

$$\tilde{R}_0 = \frac{\tilde{\lambda}}{\gamma}. \quad (3.6)$$

As mentioned in Section 2, the intensity process $\Lambda(t)$ is a cyclically varying renewal process. (This type of processes are in the literature often referred to as alternating renewal processes when $k = 2$.) For the case with k environmental states, we get

$$\tilde{\lambda} := \sum_{i=1}^k \frac{\mathbf{E}[T_i]}{\mathbf{E}[T_1] + \dots + \mathbf{E}[T_k]} \lambda_i, \quad (3.7)$$

by using well known facts from renewal theory (i.e. the factors in front of the λ_i 's are the long-time proportion of time that $\Lambda(t)$ spends in state i , which follows from the law of large numbers for renewal processes, see e.g. [10, Ch. 3.4, pp. 202-216]). By using R_\star from Theorem 3.1 together with $\tilde{\lambda}$ from (3.7) we can conclude the following:

Corollary 3.2. *Let \tilde{R}_\star be defined as in Theorem 3.1, but with all λ_i ($i = 1, \dots, k$) replaced with $\tilde{\lambda}$ from (3.7), the mean intensity of the process $\Lambda(t)$, and let R_\star be that of Theorem 3.1. For R_\star we also assume that $\text{Var}(T_i) > 0$ for some i and let $t_i := \mathbf{E}[T_i]$. Further, let R_\star^δ denote R_\star where all $T_i \in \delta(t_i)$, i.e. $T_i \equiv t_i$, with the corresponding \tilde{R}_\star^δ . Then,*

$$(i) \quad R_\star > \tilde{R}_\star,$$

$$(ii) R_\star^\delta = \tilde{R}_\star^\delta,$$

$$(iii) R_\star > R_\star^\delta.$$

Proof of Corollary 3.2. By noting that

$$\Psi_i(s) := \mathbb{E}[e^{sT_i}] > e^{s\mathbb{E}[T_i]},$$

holds by a direct application of Jensen's inequality (except when $T_i \equiv \mathbb{E}[T_i]$ when we have equality), the proof of Corollary 3.2 follows directly from the definition of R_\star from (3.5) combined with the definition of $\tilde{\lambda}$ from (3.7). \square

Recall from Section 1 that a large outbreak in general only can occur when $R_0 > 1$ (or in our situation when $\tilde{R}_0 > 1$). Using this and combining Corollary 3.2, Remark 3.1 and (3.6) it is clear that

$$\tilde{R}_\star \underset{\approx}{\geq} 1 \iff \tilde{R}_0 \underset{\approx}{\geq} 1,$$

which implies that $\tilde{R}_0 \geq 1 \Rightarrow R_\star \geq 1$. To see why the opposite implication does not hold, use the following counter-example: Consider the situation with two environmental states, where T_1 and T_2 are independent $\text{Exp}(\nu_i)$ -distributed ($i = 1, 2$) with corresponding moment generating functions

$$\Psi_i(s) = \frac{1}{1 - \frac{s}{\nu_i}},$$

where $s \in [0, \nu_i)$. Further, due to Remark 3.1, we assume that $\lambda_1 > 1 > \lambda_2$, and R_\star from Theorem 3.1 becomes

$$R_\star = \frac{1}{1 - \frac{\lambda_1 - \gamma}{\nu_1}} \frac{1}{1 - \frac{\lambda_2 - \gamma}{\nu_2}}. \quad (3.8)$$

If we for example set $\lambda_1 = 2.7, \lambda_2 = 0.8, \gamma = 2$ and $\nu_1 = \nu_2 = 1$, then $\tilde{\lambda} = 1.75$ and $\tilde{R}_0 = 0.875 < 1$ while $R_\star \approx 1.52 > 1$. Thus, we have an example where the approximation can not take off while the true epidemic can. This could make health authorities incorrectly feel "safe" when using the approximated process.

In the next section we will prove a coupling between a suitable branching process and our epidemic process and prove Theorem 3.1. Once this coupling has been established, we use that this approximating process observed at the time points when environmental cycles are completed behaves as a Galton-Watson process. Known facts from branching process theory then gives us Theorem 3.1.

4 Branching process approximation

In this section we relate the epidemic process to a suitable branching process in a varying environment and show that the ultimate number of infected in the epidemic process converges in distribution to the ultimate number of born individuals in this approximating process. This is done by using a coupling argument analogous to that used in [6] for the general epidemic. For more on branching processes in varying and random environments we refer the reader to e.g. [14, Ch. 5.10, pp. 145-152] and references therein, and for more mathematical references on branching processes in general see e.g. [15], [5] and [16].

Recall that $\Lambda(t)$ denotes the environmental process defined in Section 2, where $\Lambda(t) \in \{\lambda_1, \dots, \lambda_k\}$. Let $X(t; \Lambda(t), \gamma)$ denote a continuous time branching process in a varying environment with k different states describing the number of individuals alive at time t analogously defined as $X(t; \lambda_i, \gamma)$ from the previous section, but with λ_i replaced with $\Lambda(t)$. For this process individuals give birth at rate λ_i while the environmental process is in state i , and individuals die independently of everything else at rate γ .

To see how we can construct $I^{(n)}(t; \Lambda(t), \gamma)$ using $X(t; \Lambda(t), \gamma)$, we first introduce a sequence of i.i.d. $U(0,1)$ -distributed random variables $\zeta_i, i = 1, 2, \dots$ and label all n initially susceptible individuals in the epidemic process from 1 to n . Then whenever an individual in $X(t; \Lambda(t), \gamma)$ is born an individual in $I^{(n)}(t; \Lambda(t), \gamma)$ becomes infectious. That is, when the i :th individual in $X(t; \Lambda(t), \gamma)$ is born, we pick an individual amongst the n initially susceptible individuals labelled $[\zeta_i n] + 1$ and infect it in $I^{(n)}(t; \Lambda(t), \gamma)$. This infected individual will then have the same characteristics as that of the i :th born individual in $X(t; \Lambda(t), \gamma)$. On the other hand, if the picked individual no longer is susceptible the coupling brakes down and we have encountered what is called a “ghost” following Mollison [20], but until this event occurs the two processes will be identical.

Let $B(t; \Lambda(t), \gamma)$ denote the number of births in $X(t; \Lambda(t), \gamma)$ up to t . If we define $E^{(n)} := n - S^{(n)}(\infty; \Lambda(\infty), \gamma)$, and $E := B(\infty; \Lambda(\infty))$, we can state the main result of this section:

Theorem 4.1. *We have that $E^{(n)} \xrightarrow{d} E$ as $n \rightarrow \infty$.*

Before we proceed with the proof of Theorem 4.1, we need to establish for how long time the coupling between $I^{(n)}(t; \Lambda(t), \gamma)$ and $X(t; \Lambda(t), \gamma)$ holds:

Lemma 4.2. *The processes $I^{(n)}(t; \Lambda(t), \gamma)$ and $X(t; \Lambda(t), \gamma)$ can with a probability tending to one (as n tends to infinity) be coupled up to time $\tau(n) := c \log n$, where $0 < c < (1 - 2\epsilon)/(2(\lambda_{\max} - \gamma))$, $\lambda_{\max} := \max_{i=1, \dots, k} \lambda_i$ ($\Lambda(t) \in \{\lambda_1, \dots, \lambda_k\}$) and $0 < \epsilon < 1/2$.*

Proof of Lemma 4.2. To prove Lemma 4.2 we need the following notation: let $B^{(n)}(t; \Lambda(t), \gamma)$ denote the number of individuals infected up to t in $I^{(n)}(t; \Lambda(t), \gamma)$. Further, let $X(t; \lambda_{\max}, \gamma)$ be a linear birth and death process with birth and death intensity λ_{\max} and γ respectively, and let $B(t; \lambda_{\max}, \gamma)$ denote the number of individuals born up to t in $X(t; \lambda_{\max}, \gamma)$. Note that $B^{(n)}(t; \Lambda(t), \gamma) \leq_d B(t; \Lambda(t), \gamma) \leq_d B(t; \lambda_{\max}, \gamma)$ holds for all $t \geq 0$.

From the classical birthday problem it follows that the coupling will hold up until $n^{1/2-\epsilon}$ individuals have been born with a probability tending to one as n tends to infinity where $0 < \epsilon < 1/2$, see e.g. [12, Ch. II.3, pp. 31]. From the relation $B^{(n)}(t; \Lambda(t), \gamma) \leq_d B(t; \Lambda(t), \gamma) \leq_d B(t; \lambda_{\max}, \gamma)$ it follows that if it takes no more than $\tau(n)$ time units for $B(t; \lambda_{\max}, \gamma)$ to reach $n^{1/2-\epsilon}$ individuals with a high probability, the coupling between $I^{(n)}(t; \Lambda(t), \gamma)$ and $X(t; \Lambda(t), \gamma)$ will hold at least as many time units. Thus, we want to show that $\mathbb{P}(B(\tau(n); \lambda_{\max}, \gamma) > n^{1/2-\epsilon}) \rightarrow 0$ as $n \rightarrow \infty$ for some suitable choice of $\tau(n)$. This we will do by using Markov's inequality:

$$\mathbb{P}(B(t; \lambda_{\max}, \gamma) > n^{1/2-\epsilon}) \leq \frac{\mathbb{E}[B(t; \lambda_{\max}, \gamma)]}{n^{1/2-\epsilon}}. \quad (4.1)$$

Recall that $X(t; \lambda_{\max}, \gamma)$ is a linear birth and death process, and can hence be represented as $X(t; \lambda_{\max}, \gamma) = B(t; \lambda_{\max}, \gamma) - D(t; \gamma) + 1$ (i.e. $X(0; \lambda_{\max}, \gamma) = 1$), where $B(t; \lambda_{\max}, \gamma)$ is a pure birth process counting the number of individuals born up to time t , and $D(t; \lambda_{\max}, \gamma)$ is a pure birth process counting the number of individuals that have died up to t . For an ordinary linear birth and death process $X(t; \lambda, \gamma)$ with probability generating function $\xi(s, t)$ given by (3.2) where $\lambda_i = \lambda$, we get that

$$\mathbb{E}[X(t; \lambda, \gamma)] = \left. \frac{d}{ds} \xi(s, t) \right|_{s=1} = e^{(\lambda-\gamma)t}, \quad t \geq 0. \quad (4.2)$$

In order to determine $\mathbb{E}[B(t; \lambda_{\max}, \gamma)]$ we condition and get

$$\begin{aligned} \mathbb{E}[B(t+h; \lambda_{\max}, \gamma)] &= \mathbb{E}[\mathbb{E}[B(t+h; \lambda_{\max}, \gamma) \mid B(t; \lambda_{\max}, \gamma), X(t; \lambda_{\max}, \gamma)]] \\ &= \lambda \mathbb{E}[X(t; \lambda_{\max}, \gamma) + B(t; \lambda_{\max}, \gamma)]h + o(h), \end{aligned}$$

which can be rewritten as

$$\frac{1}{h} \mathbb{E}[B(t+h; \lambda_{\max}, \gamma) - B(t; \lambda_{\max}, \gamma)] = \lambda \mathbb{E}[X(t; \lambda_{\max}, \gamma)] + \frac{o(h)}{h}.$$

If we let $h \rightarrow 0$ and integrate with respect to time, and use (4.2), we arrive at

$$\mathbb{E}[B(t; \lambda_{\max}, \gamma)] = \frac{\lambda_{\max}}{\lambda_{\max} - \gamma} \left(e^{(\lambda_{\max} - \gamma)t} - 1 \right), \quad t \geq 0.$$

This together with (4.1) gives us that $\mathbb{P}(B(\tau(n); \lambda_{\max}, \gamma) > n^{1/2-\epsilon}) \rightarrow 0$ as $n \rightarrow \infty$ if $\tau(n) := c \log n$, where $c < (1 - 2\epsilon)/(2(\lambda_{\max} - \gamma))$ and $0 < \epsilon < 1/2$, which finishes the proof. \square

Remark 4.1. Note that if $\lambda_{\max} \leq \gamma$ then $\tau(n) = +\infty$.

Proof of Theorem 4.1. First, note that $E^{(n)} := n - S^{(n)}(\infty; \Lambda(\infty), \gamma) = B^{(n)}(\infty; \Lambda(\infty), \gamma)$ and recall that $E := B(\infty; \Lambda(\infty), \gamma)$. Hence, in order to show that $E^{(n)} \xrightarrow{d} E$ as $n \rightarrow \infty$, we need to relate $B^{(n)}(t; \Lambda(t), \gamma)$ to $B(t; \Lambda(t), \gamma)$. As before, let $\tau(n)$ denote the time until the coupling breaks down. From Lemma 4.2 we have that for large enough n it holds that $\mathbb{P}(\tau(n) \leq c \log n) > 1 - \epsilon/2$, where $\epsilon > 0$, and that $\{B^{(n)}(t; \Lambda(t), \gamma) \leq k\} = \{B(t; \Lambda(t), \gamma) \leq k\}$ for any fixed $k \leq n^{1/2}$ and $t \leq \tau(n)$. Following the line of proof of Theorem 3.1 from [9]: Introduce $A_n = \{\tau(n) \leq c \log n\}$. For fixed $t \leq \tau(n)$ and $k \leq \sqrt{n}$ we get that

$$\begin{aligned} \mathbb{P}(B^{(n)}(t; \Lambda(t), \gamma) \leq k) &= \mathbb{P}(\{B^{(n)}(t; \Lambda(t), \gamma) \leq k\} \cap A_n) + \mathbb{P}(\{B^{(n)}(t; \Lambda(t), \gamma) \leq k\} \cap A_n^c) \\ &= \mathbb{P}(\{B(t; \Lambda(t), \gamma) \leq k\} \cap A_n) + \mathbb{P}(\{B^{(n)}(t; \Lambda(t), \gamma) \leq k\} \cap A_n^c) \\ &\leq \mathbb{P}(B(t; \Lambda(t), \gamma) \leq k) + \epsilon/2. \end{aligned}$$

Likewise, we get

$$\begin{aligned} \mathbb{P}(B^{(n)}(t; \Lambda(t), \gamma) \leq k) &= \mathbb{P}(\{B^{(n)}(t; \Lambda(t), \gamma) \leq k\} \cap A_n) + \mathbb{P}(\{B^{(n)}(t; \Lambda(t), \gamma) \leq k\} \cap A_n^c) \\ &\geq \mathbb{P}(\{B(t; \Lambda(t), \gamma) \leq k\} \cap A_n) \\ &= \mathbb{P}(B(t; \Lambda(t), \gamma) \leq k) - \mathbb{P}(\{B(t; \Lambda(t), \gamma) \leq k\} \cap A_n^c) \\ &\geq \mathbb{P}(B(t; \Lambda(t), \gamma) \leq k) - \epsilon/2, \end{aligned}$$

and hence

$$|\mathbb{P}(B^{(n)}(t; \Lambda(t), \gamma) \leq k) - \mathbb{P}(B(t; \Lambda(t), \gamma) \leq k)| < \epsilon$$

holds. But, in the limit as $n \rightarrow \infty$, we have that $k \in \mathbb{N}$, $t \in \mathbb{R}^+$ and $\epsilon > 0$ which where all chosen arbitrarily. Hence it holds that $B^{(n)}(t; \Lambda(t), \gamma) \xrightarrow{d} B(t; \Lambda(t), \gamma)$ as $n \rightarrow \infty$ for all fixed $t \in \mathbb{R}^+$ and in particular it follows that $E^{(n)} \xrightarrow{d} E$ as $n \rightarrow \infty$. \square

Note that even though we know that $E^{(n)} \rightarrow E$ in distribution as $n \rightarrow \infty$ we have not yet established any characteristics concerning the distribution of E . For the purposes of the present paper it is of interest to determine whether E is distributed as a degenerate random variable, with a point mass at infinity or not, and give conditions for when this occurs. This is effectively what is stated in Theorem 3.1. By noting that $X(t; \Lambda(t), \gamma)$ observed at the time points when environmental cycles are completed behaves as a certain Galton-Watson process, Theorem 3.1 follows by applying standard results from branching process theory.

Proof of Theorem 3.1. As before, let T_i be a random variable distributed as the time that $\Lambda(t)$ spends in state i during a typical visit. We will also use the notation T_{ij} which denotes the j :th visit in state i , and we will make no distinction between T_i and T_{i1} . Recall that all $\{T_i\}$ are mutually independent and that, for each $i = 1, \dots, k$, all T_{i1}, T_{i2}, \dots are i.i.d.

In addition, let U_i denote the time point of the i :th completed environmental cycle. This gives us that $U_1 = T_1 + \dots + T_k$ and in general we have $U_i = T_1 + \dots + T_k + T_{12} + \dots + T_{ki}$.

Let $\xi_i(s, t)$, $i = 1, \dots, k$, denote the generating function of $X(t; \lambda_i, \gamma)$ given by (3.2). Recall that whenever the environmental process stays constant the process $X(t; \Lambda(t), \gamma)$ will behave like an ordinary linear birth and death process. Thus, by conditioning on the time points $\{U_i\}$ and using that all individuals in $X(t; \Lambda(t), \gamma)$ live for exponentially distributed time periods, we obtain the recursive relation:

$$\begin{aligned} \mathbb{E}[s^{X(U_1; \Lambda(U_1), \gamma)} \mid \{T_1, \dots, T_k; X(T_1 + \dots + T_{k-1}; \Lambda(T_1 + \dots + T_{k-1}), \gamma)\}] &= \\ &= \xi_k(s, T_k)^{X(T_1 + \dots + T_{k-1}; \Lambda(T_1 + \dots + T_{k-1}), \gamma)}. \end{aligned} \quad (4.3)$$

Solving this recursive relation backwards from k to 1 it then follows after some further conditioning that

$$\chi(s) := \mathbb{E}[s^{X(U_1; \Lambda(U_1), \gamma)}] = \mathbb{E}[\xi_1(\xi_2(\xi_3(\dots(\xi_k(s, T_k), T_{k-1}), \dots), T_3), T_2), T_1)],$$

which is the same expression as (3.3).

Moreover, from the definition of $\Lambda(t)$ we know that the sums $T_{1i} + \dots + T_{ki}$, $i = 1, 2, \dots$ are i.i.d., which together with the fact that all individuals live for an exponential life-length in the process $X(t; \Lambda(t), \gamma)$ gives us that the process restarts at the time points $\{U_i\}$. Consequently, if we introduce $\tilde{X}_1, \tilde{X}_2, \dots$ i.i.d. all distributed as $X(U_1; \Lambda(U_1), \gamma)$, we get that

$$\begin{aligned} \mathbb{E}[s^{X(U_2; \Lambda(U_2), \gamma)}] &= \mathbb{E}[\mathbb{E}[s^{X(U_2; \Lambda(U_2), \gamma)} \mid \{T_1, \dots, T_{k2}; X(T_1 + \dots + T_k; \Lambda(T_1 + \dots + T_k), \gamma)\}]] \\ &= \mathbb{E}[\mathbb{E}[s^{X(U_2; \Lambda(U_2), \gamma)} \mid \{T_1, \dots, T_{k2}; X(U_1; \Lambda(U_1), \gamma)\}]] \\ &= \mathbb{E}\left[\mathbb{E}\left[s^{\sum_{i=1}^{X(U_1; \Lambda(U_1), \gamma)} \tilde{X}_i} \mid X(U_1; \Lambda(U_1), \gamma)\right]\right] \\ &= \mathbb{E}[\mathbb{E}[s^{\tilde{X}}]^{X(U_1; \Lambda(U_1), \gamma)}] = \chi(\chi(s)), \end{aligned}$$

and hence $X(t; \Lambda(t), \gamma)$ observed at the time points $\{U_i\}$ behaves as a Galton-Watson process with offspring distribution determined by $\chi(s)$ from (3.3).

Note that if $X(t; \Lambda(t), \gamma)$ observed at the time points $\{U_i\}$ has died out, obviously $X(t; \Lambda(t), \gamma)$ has died out as well. For our purposes the time point when this event occurs is of no interest, and if we introduce $E_d := \sum_{i=0}^{\infty} X(U_i; \Lambda(U_i), \gamma)$, it hence holds that $E_d = +\infty$ if and only if $E = +\infty$. From standard references in branching process theory, see e.g. [16, Ch. 2.11, pp. 39-42], it is known that $\mathbb{P}(E_d = +\infty) > 0$ if and only if $\mathbb{E}[X(U_1; \Lambda(U_1), \gamma)] > 1$. But, from the definition of $\chi(s)$, see (3.3), it follows that $\chi'(1) = \mathbb{E}[X(U_1; \Lambda(U_1), \gamma)]$. Note that (4.3) is a conditional generating function. Hence, we obtain the corresponding conditional expectations by differentiating both sides of (4.3) with respect to s and then setting $s = 1$ and use (4.2). Thus, by solving the recursive relation (4.3) backwards and using that we can extract all the conditional expectations

along the way, we get that $\chi'(1) = R_\star$, where R_\star is from (3.5). Thus, $\mathbb{P}(E_d = +\infty) > 0$ and hence $\pi := \mathbb{P}(E = +\infty) > 0$ if and only if $R_\star > 1$. Moreover, π is given by $\pi := 1 - \rho$, where ρ is the smallest non-negative solution to $s = \chi(s)$, see e.g. [16, Thm. 2.3.1, Ch. 22, p. 22]. Thus, Theorem 3.1 is proved. \square

Remark 4.2. In the proof of Theorem 3.1 we made use of an approximating Galton-Watson process. For this process generations corresponds to completed environmental cycles, and the particles corresponds to infectious individuals. Thus, a natural interpretation of R_\star is as the average number of individuals that are infectious at the time point when the first environmental cycle is completed, given that the epidemic was initiated by a single infectious individual.

5 Examples

In the present section we consider two examples both with $k = 2$ environmental states. The first example is when the time periods spent in different environmental states are exponentially distributed, and the second is when the time periods are non-random. For both of these examples it is possible to compute $\pi := 1 - \rho$ from Theorem 3.1. Before we proceed with the computation of these probabilities, we describe how the situation with exponentially distributed time periods relates to a certain multi-type epidemic.

5.1 On the relation to multi-type epidemics

Consider the situation where the environment may change between two states, corresponding to the intensities λ_1 and λ_2 , and assume that $T_i \in \text{Exp}(\nu_i)$. The situation is now equivalent to that of the counter-example that was treated after that Theorem 3.1 was stated in Section 3, and R_\star is thus given by (3.8). Let $r_1 := \lambda_1/\gamma$ and $r_2 := \lambda_2/\gamma$. The interpretation of r_i is that $r_i > 1$ corresponds to that the epidemic process in its early stages under influence of the environment i behaves like a super-critical branching process, when $r_i < 1$ it behaves like a sub-critical branching process, and when $r_i = 1$ it behaves like a critical branching process. As a consequence of Remark 3.1 the interesting case is when $r_1 > 1 > r_2$. By straightforward algebraic manipulation of (3.8) we get that

$$\frac{r_1 - 1}{\nu_1} \left(\frac{\nu_2}{1 - r_2} + \gamma \right) \begin{matrix} \geq \\ \leq \end{matrix} 1 \iff R_\star \begin{matrix} \geq \\ \leq \end{matrix} 1,$$

and since these expressions are equivalent, we set

$$R_\star := \frac{r_1 - 1}{\nu_1} \left(\frac{\nu_2}{1 - r_2} + \gamma \right). \quad (5.1)$$

By analysing R_\star term-wise, one sees that $(r_1 - 1)/\nu_1$ is the average excess of infectious individuals that is generated during a typical time period when the environment is in

state 1, and $(1 - r_2)/\nu_2$ is the corresponding shortage of infectious individuals. Thus, the heuristic interpretation of this threshold condition is that in order for the epidemic to take off, there must be a sufficient excess of infectious individuals from the time spent in the “favourable” (from the disease’s perspective) state of the environment in order to compensate for the time spent in the “unfavourable” state of the environment.

To relate this system to a two-type epidemic, introduce Z_{ij} , where Z_{ij} denotes the number of type j -individuals that a single type i -individual infects in an otherwise disease free population during its infectious period. Here a type- i individual corresponds to an individual that becomes infectious during a time period where the environment is in state i . Let \tilde{T} denote the infectious period of any individual, $\tilde{T} \in \text{Exp}(\gamma)$, and let T_i denote the time that the environment spends in state i as before. Here $i, j = 1, 2$ and we will for relief of notation henceforth assume that $i \neq j$.

While the environmental process is in state i , infectious contacts are made according to a Poisson process with intensity λ_i . Further, if an individual becomes infectious while the environmental process is in state i , the time until this individual either recovers or that the environment changes state is $\min\{T_i, \tilde{T}\} \in \text{Exp}(\nu_i + \gamma)$. Combining these two facts gives us that the number of i -individuals that a newly infected i -individual will infect up to the time of recovery or until the environment changes state is $\text{Po}(\lambda_i \min\{T_i, \tilde{T}\})$ distributed, conditional on $\min\{T_i, \tilde{T}\}$. Let the corresponding unconditional number of infected i -individuals be denoted by \tilde{Z}_i , and it hence holds that $\tilde{Z}_i \in \text{Geo}(p_i)$, $p_i := (\nu_i + \gamma)/(\nu_i + \gamma + \lambda_i)$, and $\mathbb{E}[\tilde{Z}_i] := (1 - p_i)/p_i = \lambda_i/(\nu_i + \gamma)$. By conditioning on whether an individual recovers before the environment changes state or not, together with that the system is Markovian gives us that

- (i) $[Z_{ii}|T_i > \tilde{T}] =_d \tilde{Z}_i + 0$
- (ii) $[Z_{ii}|T_i \leq \tilde{T}] =_d \tilde{Z}_i + Z_{ji}$
- (iii) $[Z_{ij}|T_i > \tilde{T}] \equiv 0$
- (iv) $[Z_{ij}|T_i \leq \tilde{T}] =_d Z_{jj}$.

Using (i) – (iv) yields

$$\begin{aligned} \mu_{ii} &:= \mathbb{E}[Z_{ii}|T_i > \tilde{T}]\mathbb{P}(T_i > \tilde{T}) + \mathbb{E}[Z_{ii}|T_i \leq \tilde{T}]\mathbb{P}(T_i \leq \tilde{T}) \\ &= \mathbb{E}[\tilde{Z}_i] \frac{\gamma}{\nu_i + \gamma} + \mathbb{E}[\tilde{Z}_i + Z_{ji}] \frac{\nu_i}{\nu_i + \gamma} \\ &= \mathbb{E}[\tilde{Z}_i] + \frac{\nu_i}{\nu_i + \gamma} \mathbb{E}[Z_{ji}] = \frac{\lambda_i}{\nu_i + \gamma} + \frac{\nu_i}{\nu_i + \gamma} \mu_{ji}, \end{aligned}$$

and

$$\mu_{ij} := \frac{\nu_i}{\nu_i + \gamma} \mu_{jj},$$

which gives us

$$\begin{aligned}\mu_{11} &= \frac{\lambda_1}{\nu_1 + \gamma} + \mu_{21} \frac{\nu_1}{\nu_1 + \gamma} \\ \mu_{21} &= \mu_{11} \frac{\nu_2}{\nu_2 + \gamma}\end{aligned}$$

and μ_{22} and μ_{12} follow by symmetry. Combining these relations imply that

$$\begin{aligned}\mu_{ii} &= \frac{\lambda_i(\nu_j + \gamma)}{\gamma(\nu_1 + \nu_2 + \gamma)} \\ \mu_{ij} &= \frac{\lambda_j \nu_i}{\gamma(\nu_1 + \nu_2 + \gamma)}.\end{aligned}$$

The basic reproduction number, R_0 , is then defined as the largest eigenvalue for $\mathbb{M} = \{\mu_{ij}\}_{i,j=1,2}$, i.e. the largest κ such that $\det(\mathbb{M} - \kappa \mathbb{I}) = 0$, see e.g. [7, p. 730]. For this model R_0 becomes

$$R_0 = \frac{\mu_{11} + \mu_{22}}{2} + \sqrt{\left(\frac{\mu_{11} + \mu_{22}}{2}\right)^2 + \mu_{21}\mu_{12} - \mu_{11}\mu_{22}} =: w + \sqrt{w^2 + v}. \quad (5.2)$$

If we consider conditions for $R_0 > 1$, this is equivalent to that $w + \sqrt{w^2 + v} > 1$ which in turn is equivalent to that $2w + v > 1$. Inserting v and w from (5.2) and simplifying then gives us

$$\begin{aligned}\frac{r_1(\nu_2 + \gamma) + r_2(\nu_1 + \gamma) - r_1 r_2 \gamma}{\nu_1 + \nu_2 + \gamma} &> 1 \\ \Rightarrow \frac{r_1 - 1}{\nu_1} \left(\frac{\nu_2}{1 - r_2} + \gamma \right) &> 1,\end{aligned}$$

i.e. $R_\star > 1$, where R_\star is from (5.1). Thus

$$R_\star > 1 \iff R_0 > 1,$$

and consequently the same relation holds for $R_0 \leq 1$ and hence the two thresholds are equivalent. We believe that the conclusions are valid for general k , but it is not clear to us how to prove this.

Above we have described how one can represent an epidemic in an alternating environment with two states, where the time periods that the environment spends in its different states are exponentially distributed, as a two-type epidemic. This could also be done for the case where the time periods follows some arbitrary distribution (with finite expectation). For this situation, however, we must keep track of the *time points* where individuals become infected, since this system is no longer Markovian. In terms of multi-type epidemics this is equivalent to having a continuum of types, where the types now include information about the time point of infection.

5.2 Probability of a large outbreak — Two examples

In this section we give two examples where π , the probability of a large outbreak, is possible to compute. The first example is when the time periods that the environment spends in its different states are exponentially distributed. In this situation we can make use of known facts concerning multi-type epidemics. The second example is when the time periods that the environment spends in its different states are non-random. Then π can be obtained directly as $\pi := 1 - \rho$, where ρ is the smallest non-negative solution to the equation $s = \chi(s)$, where $\chi(s)$ is from (3.3).

5.2.1 Exponentially distributed time periods

In Theorem 3.1 it is stated that the probability of a large outbreak is given as one minus the smallest non-negative solution to the equation $s = \chi(s)$, where $\chi(s)$ is from (3.3). As mentioned before, $\chi(s)$ is the generating function defining a certain Galton-Watson process. In the previous subsection the case when the time periods that the environmental process spends in its different states are exponentially distributed was treated and one could instead represent this situation in terms of a certain multi-type epidemic. For the case with two environmental states, define the generating functions

$$\eta_{(i)}(s_1, s_2) := \mathbf{E}[s_1^{Z_{i1}} s_2^{Z_{i2}}], \quad |s_1|, |s_2| \leq 1, \quad (5.3)$$

where $i = 1, 2$ and where Z_{ij} are defined as in the previous subsection. That is, $\eta_{(i)}(s_1, s_2)$ is the generating function for the number of type 1 and type 2 individuals originating from a single ancestor of type i . The probability of a large outbreak in the multi-type epidemic defined by the generating functions $\eta_{(i)}(s_1, s_2)$, $i = 1, 2$, is obtained as one minus the smallest non-negative solution to the equation system

$$\begin{cases} s_1 &= \eta_{(1)}(s_1, s_2) \\ s_2 &= \eta_{(2)}(s_1, s_2) \end{cases}, \quad (5.4)$$

see e.g. [7, p. 730]. Let $\pi_i := 1 - \rho_i$, $i = 1, 2$, where ρ_i is the smallest non-negative solution to (5.4). Hence, π from Theorem 3.1, i.e. the probability of a large outbreak given that the epidemic was initiated by a single infectious individual infected at the time point when a new environmental cycle was started, is given by $\pi := \pi_1$.

By using (5.3) together with the relations (i) – (iv) from the previous subsection we

can determine $\eta_{(i)}(s_1, s_2)$:

$$\begin{aligned}
\eta_{(1)}(s_1, s_2) &= \mathbf{E}[s_1^{Z_{11}} s_2^{Z_{12}} | T_1 > \tilde{T}] \mathbf{P}(T_1 > \tilde{T}) + \mathbf{E}[s_1^{Z_{11}} s_2^{Z_{12}} | T_1 \leq \tilde{T}] \mathbf{P}(T_1 \leq \tilde{T}) \\
&= \mathbf{E}[s_1^{Z_{11}} | T_1 > \tilde{T}] \frac{\gamma}{\nu_1 + \gamma} + \mathbf{E}[s_1^{Z_{11}} s_2^{Z_{12}} | T_1 \leq \tilde{T}] \frac{\nu_1}{\nu_1 + \gamma} \\
&= \mathbf{E}[s_1^{\tilde{Z}_1}] \frac{\gamma}{\nu_1 + \gamma} + \mathbf{E}[s_1^{\tilde{Z}_1 + Z_{21}} s_2^{Z_{22}}] \frac{\nu_1}{\nu_1 + \gamma} \\
\{\text{independence}\} &= \mathbf{E}[s_1^{\tilde{Z}_1}] \left(\frac{\gamma}{\nu_1 + \gamma} + \mathbf{E}[s_1^{Z_{21}} s_2^{Z_{22}}] \frac{\nu_1}{\nu_1 + \gamma} \right) \\
\{\text{def.}\} &= \tilde{\eta}_1(s_1) \left(\frac{\gamma}{\nu_1 + \gamma} + \eta_{(2)}(s_1, s_2) \frac{\nu_1}{\nu_1 + \gamma} \right), \tag{5.5}
\end{aligned}$$

where

$$\tilde{\eta}_i(s_i) = \mathbf{E}[s_i^{\tilde{Z}_i}] = \frac{p_i}{1 - (1 - p_i)s_i}, \quad i = 1, 2,$$

and where $p_i = (\nu_i + \gamma)/(\nu_i + \gamma + \lambda_i)$. By symmetry it follows that

$$\eta_{(2)}(s_1, s_2) = \tilde{\eta}_2(s_2) \left(\frac{\gamma}{\nu_2 + \gamma} + \eta_{(1)}(s_1, s_2) \frac{\nu_2}{\nu_2 + \gamma} \right). \tag{5.6}$$

Combining (5.5) and (5.6) yields

$$\eta_{(i)}(s_1, s_2) = \frac{\gamma \tilde{\eta}_i(s_i) (\nu_j + \gamma + \nu_i \tilde{\eta}_j(s_j))}{(\nu_1 + \gamma)(\nu_2 + \gamma) - \nu_1 \nu_2 \tilde{\eta}_1(s_1) \tilde{\eta}_2(s_2)}, \tag{5.7}$$

where $i = 1, 2$. Note that from the definition we get

$$\mu_{ij} = \left. \frac{d}{ds_j} \eta_{(i)}(s_1, s_2) \right|_{s_1=s_2=1}, \tag{5.8}$$

and one can check that the calculations agree. Returning to the issue of calculating the probability of a large outbreak we must solve the equation system (5.4). This is unfortunately not possible to do analytically, but the system is easily solved numerically.

In order to compare π , the probability of a large outbreak, for different parameter settings, we first keep $\nu_1 = \nu_2 = 1$ and set $\gamma = 1$ for simplicity, and vary λ_1 and λ_2 in such a way that R_\star is kept fixed. One such parametrisation assuming $\lambda_1 > 1 > \lambda_2$ is

$$\begin{cases} \lambda_1 &= 1 + \delta \\ \lambda_2 &= 2 - \frac{1}{R_\star(1-\delta)} \end{cases}, \quad 1 - \frac{1}{R_\star} < \delta < 1 - \frac{1}{2R_\star}, \tag{5.9}$$

where R_\star is from (3.8). For a numerical illustration of how π varies with λ_1 and λ_2 , see Figure 1, where it is seen that the probability of a large outbreak decreases as δ increases.

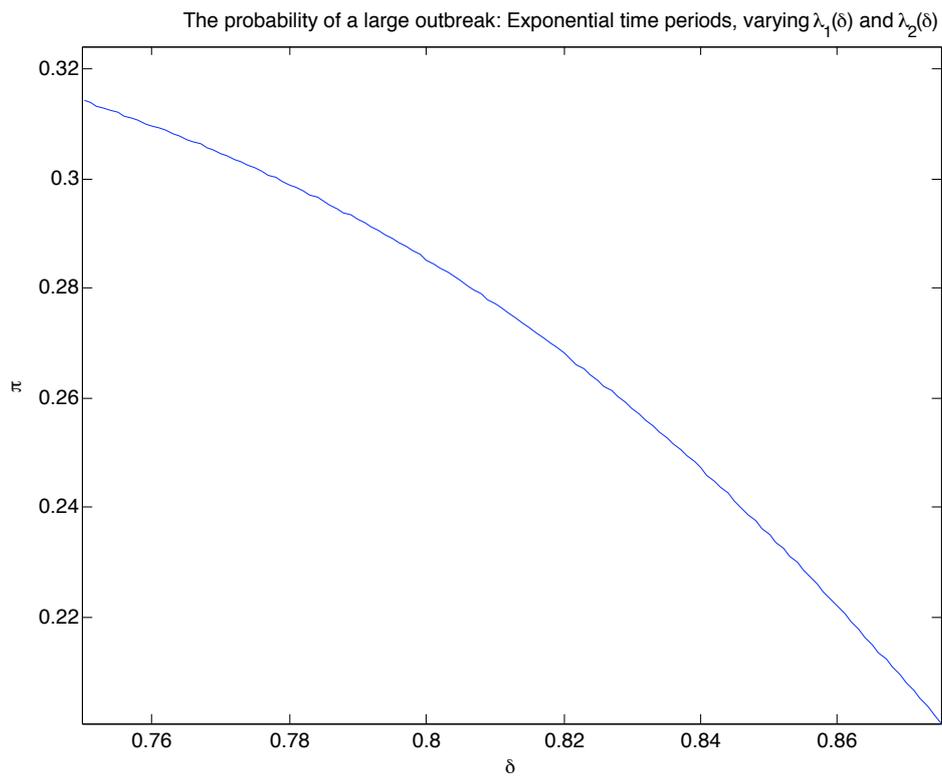


Figure 1: Exponentially distributed time periods: A numerical illustration of how π from Theorem 3.1 varies when $\lambda_1(\delta)$ and $\lambda_2(\delta)$ from (5.9) varies. Here $\gamma = 1$, $\nu_1 = \nu_2 = 1$ and $R_* = 4$. On the y-axis we have π , and on the x-axis we have δ from (5.9).

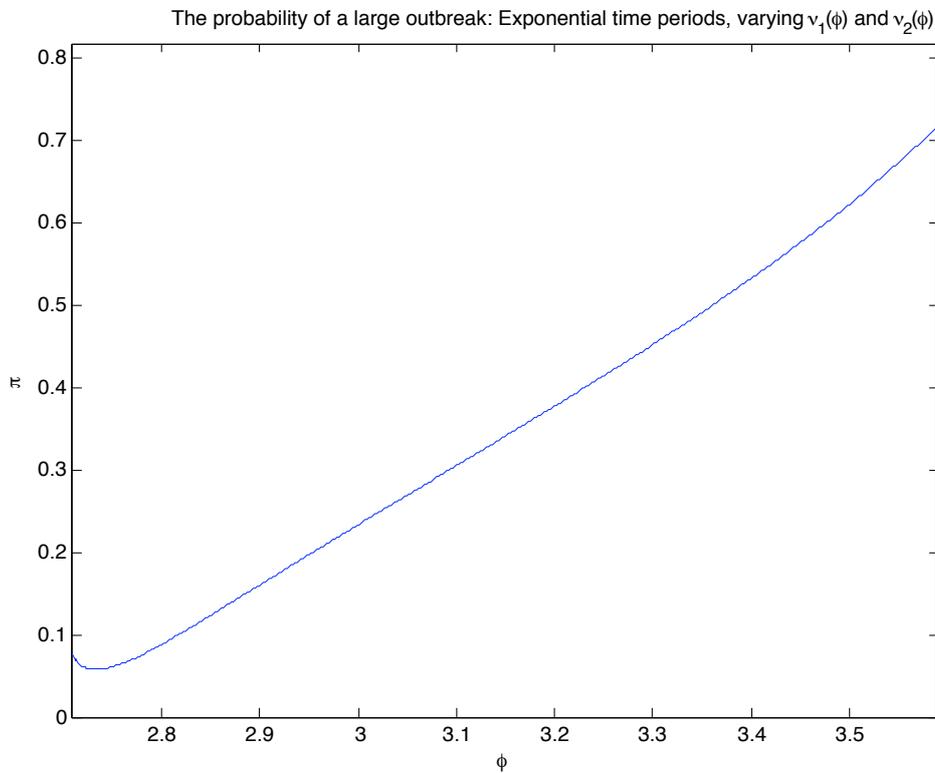


Figure 2: Exponentially distributed time periods: A numerical illustration of how π from Theorem 3.1 varies when $\nu_1(\phi)$ and $\nu_2(\phi)$ from (5.10) varies. Here $\gamma = 1$, $\nu = 1$, $\lambda_1 = 3.7$, $\lambda_2 = .4$ and $R_\star = 4$. On the y-axis we have π , and on the x-axis we have ϕ from (5.10).

On the other hand, one can instead fix λ_1 and λ_2 and set $\gamma = 1$, and vary ν_1 and ν_2 in such a way that R_\star is kept fixed. One such parametrisation assuming $\lambda_1 > 1 > \lambda_2$ is

$$\begin{cases} \nu_1 = \nu\phi \\ \nu_2 = \frac{(1-\lambda_2)R_\star(1-\frac{\lambda_1-1}{\nu\phi})}{1-R_\star(1-\frac{\lambda_1-1}{\nu\phi})} \end{cases}, \quad \frac{\lambda_1-1}{\nu} < \phi < \frac{\lambda_1-1}{\nu(1-\frac{1}{R_\star})}. \quad (5.10)$$

A numerical illustration is given in Figure 2 from which it is seen that the behaviour of π is not necessarily monotonically increasing with increasing difference between ν_1 and ν_2 .

5.2.2 Non-random time periods

For the situation when the time periods that the environmental process spends in its different states are non-random, the probability π , is obtained by directly solving the equation $s = \chi(s)$, where $\chi(s)$ is from (3.3). This is because the outer expectation in

the definition of $\chi(s)$ vanishes. As for the situation with exponentially distributed time periods, it is not possible to solve this equation analytically.

As in the previous subsection we want to vary first λ_1 and λ_2 while all other parameters are fix, including R_\star , and then vary ν_1 and ν_2 while everything else is kept fix. First, from Theorem 3.1 we get for $k = 2$ that

$$R_\star := \exp \left\{ \frac{\lambda_1 - \gamma}{\nu_1} + \frac{\lambda_2 - \gamma}{\nu_2} \right\}, \quad (5.11)$$

if $T_i \equiv 1/\nu_i$. Using this choice of T_i we have the same expectation of as for the exponential case treated in the previous subsection. As before, set $\nu_1 = \nu_2 = 1$ and $\gamma = 1$, and parametrise $\lambda_1 > 1 > \lambda_2$ according to

$$\begin{cases} \lambda_1 &= 1 + \delta \\ \lambda_2 &= 1 - \delta + \log R_\star \end{cases}, \quad \log R_\star < \delta < 1 + \log R_\star, \quad (5.12)$$

in order to keep R_\star from (5.11) fix. For a numerical illustration of how π varies with varying λ_1 and λ_2 , see Figure 3, where it is seen that π increases with δ . Recall that in Figure 1 π instead was decreasing in δ . Thus, the distribution of the time periods that the environment spends in its different states may have a great impact on the behaviour of π .

If we instead fix $\lambda_1 > 1 > \lambda_2$ and set $\gamma = 1$, one can parametrise ν_1 and ν_2 as

$$\begin{cases} \nu_1 &= \nu\phi \\ \nu_2 &= \frac{1-\lambda_2}{\frac{\lambda_1-1}{\nu\phi} - \log R_\star} \end{cases}, \quad 0 < \phi < \frac{\lambda_1 - 1}{\nu \log R_\star}, \quad (5.13)$$

keeping R_\star from (5.11) fix. In Figure 4 an illustration of how π varies with ν_1 and ν_2 is given, and it seen that π increases monotonically with increasing ϕ . In comparison with Figure 2 where non-monotonic behaviour was observed, we again see significant differences in the behaviour of π depending on the distribution of the time that the environment spends in its different states.

To conclude, comparing Figure 1 and 2 with Figure 3 and 4 respectively, it is seen that there are dramatic differences in how π varies depending on the distribution of the time that the environmental process spends in its different states. As an example, by inspecting Figure 1 and Figure 3 one sees that in the former figure π decreases monotonically, whereas in the latter figure π instead increases monotonically.

It is interesting to note that except for the non-monotonic part of Figure 2, Figure 2 and 4 show similar behaviour. This could be an indication to that changes in contact rate are more vulnerable to different distributions of the time periods than is the case for changes in the rate with which the environment switch state.

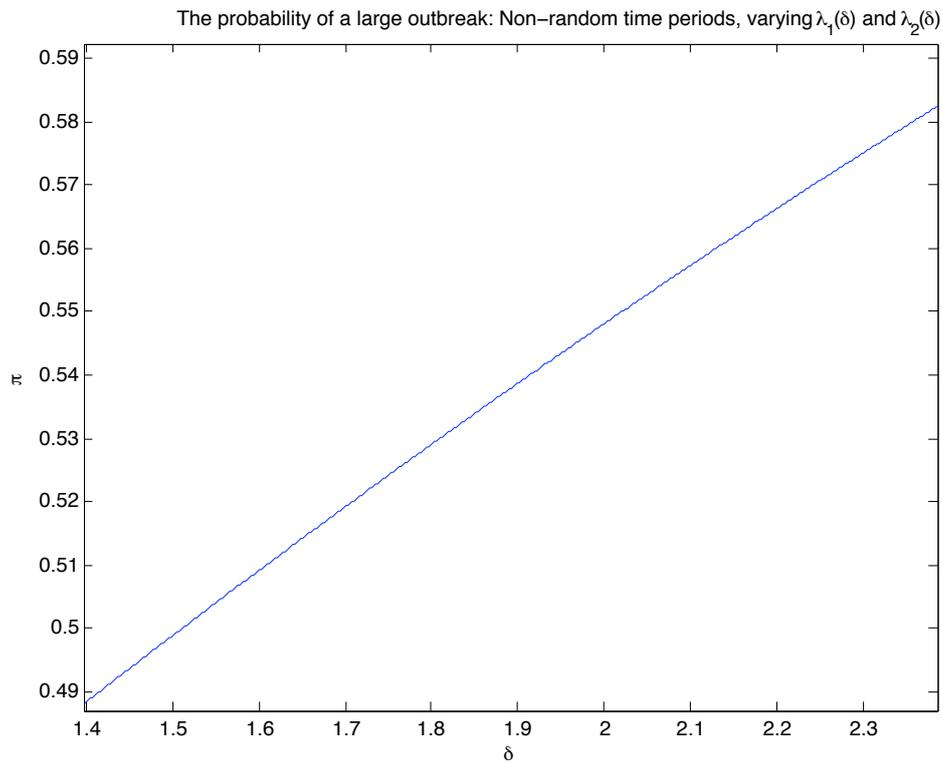


Figure 3: Non-random time periods: A numerical illustration of how π from Theorem 3.1 varies when $\lambda_1(\delta)$ and $\lambda_2(\delta)$ from (5.12) varies. Here $\gamma = 1$, $\nu_1 = \nu_2 = 1$ and $R_\star = 4$. On the y-axis we have π , and on the x-axis we have δ from (5.12).

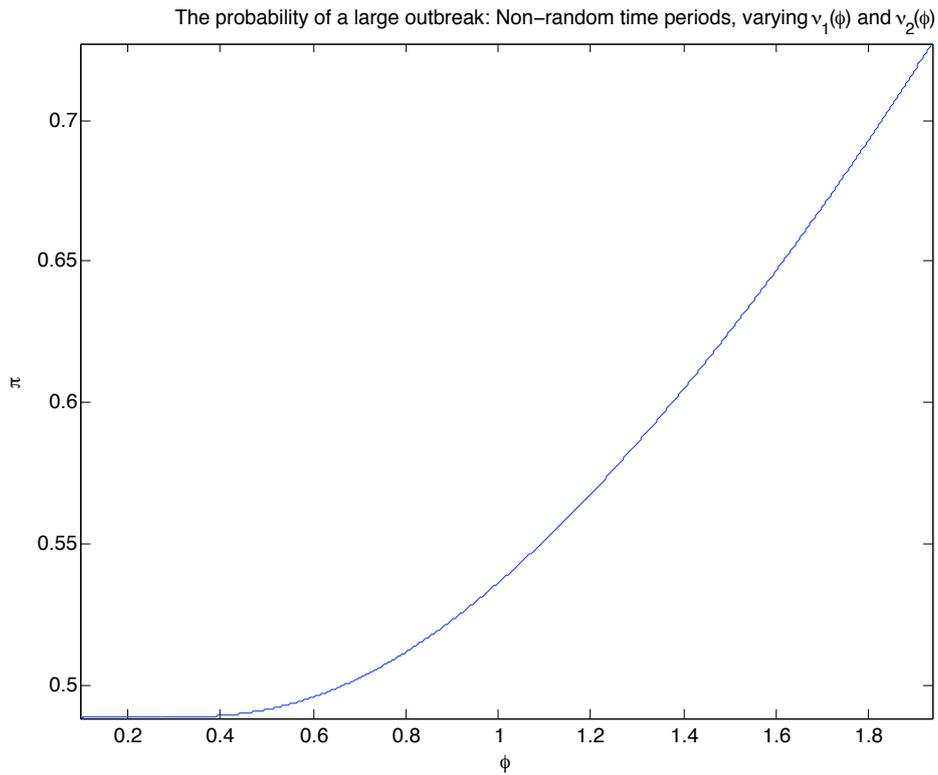


Figure 4: Non-random time periods: A numerical illustration of how π from Theorem 3.1 varies when $\nu_1(\phi)$ and $\nu_2(\phi)$ from (5.10) varies. Here $\gamma = 1$, $\nu = 1$, $\lambda_1 = 3.7$, $\lambda_2 = .4$ and $R_\star = 4$. On the y-axis we have π , and on the x-axis we have ϕ from (5.13).

6 Discussion

The present paper is concerned with the threshold behaviour of a SIR model in a term-time forced environment. An “environment” is in this context any external factor that influences the population, but itself is unaffected of the population. Here “term-time forcing” refers to that the changes in contact rate are discontinuous but cyclic. We describe this kind of system using the general SIR epidemic but where the traditionally single contact rate, λ , is replaced with a cyclically varying renewal process with k states, denoted $\Lambda(t)$. For this model we derive threshold conditions, denoted R_\star , that can be expressed as products of the moment generating functions of the time periods that $\Lambda(t)$ spends in its different states. This is stated more precisely in Theorem 3.1. The quantity R_\star can hence be stated explicitly and works as a threshold, such that a large outbreak can only occur if and only if $R_\star > 1$. Thus, R_\star relates to the so-called basic reproduction number, R_0 , in both of these aspects. The epidemiological interpretation of R_\star is more involved than that of R_0 , but from the derivation of R_\star it is seen that it corresponds to the expected number of individuals that are infectious at the time point when the first environmental cycle is completed, given that the epidemic was initiated by a single infectious individual whose infectious period starts when a new environmental cycle is started.

By checking whether R_\star is greater than one or not, we can easily determine whether a large outbreak can occur or not. That is, R_\star determines whether the probability of a large outbreak, π , is strictly greater than zero or not. The probability π is, however, much harder to compute than R_\star , since it is defined as one minus the smallest non-negative solution to $s = \chi(s)$, where $\chi(s)$ is from (3.3). In Section 5 we give two examples when it is possible to compute π numerically. The first situation is when all time periods that $\Lambda(t)$ spends in its different states are exponentially distributed. This system can then be represented as a multi-type epidemic, and by using known facts concerning multi-type epidemics it is possible to compute π . This is done in detail for the case $k = 2$ when we also show that R_0 and R_\star are equivalent in terms of threshold behaviour. We believe that this is true for general k . The second situation is when the time periods that the environmental process spends in its different states are non-random. For this situation it is possible to directly solve the equation $s = \chi(s)$. In connection with these examples we calculate π numerically, see Figures 1-4, from which it is apparent that the distribution of the time that the environmental process spends in its different states is of importance.

In Corollary 3.2 we give some qualitative relations between the behaviour of the general SIR epidemic in a term-time forced environment and the general SIR epidemic with the single contact rate $\tilde{\lambda}$ from (3.7) corresponding to the long-time average environmental effect. If we denote R_\star of the latter process by \tilde{R}_\star , we show that R_\star of the epidemic with the environment accounted for always will be greater than \tilde{R}_\star , except when all time periods that $\Lambda(t)$ spends in its different states are non-random, when $R_\star = \tilde{R}_\star$ holds.

The derivation of these results rely on the theory of branching processes and the cou-

pling between such processes and epidemic processes. The strongest form of coupling between epidemics and branching process is given in [8, Thm. 2.1, p. 4], but for our purposes it is sufficient to obtain a weaker coupling. More precisely we only need convergence in distribution between the ultimate number of infected individuals in the epidemic process and the ultimate number of born individuals in a certain branching process. This is stated in Theorem 4.1.

Throughout we have made repeated use of the fact that the system is conditionally Markovian in its nature. Consequently, the methods used in the present paper are hard to adapt directly to situations with non-exponential infectious periods, since the Markov property will then be lost. One possible generalisation could, be to allow gamma distributed infectious periods. This could possibly be done by sub-dividing the infectious period into a number of shorter exponentially distributed infectious periods similarly to what is done in [4]. In this way the Markov property of the system would be preserved. For many real-life applications the flexibility of the gamma distribution will suffice from a modelling perspective. But, as soon as the system becomes non-Markovian the approximating branching processes will be of Crump-Mode-Jagers type, and hence depend on the *distribution* of the time points of renewal. It might, however, be possible to derive some threshold conditions using embedding techniques, but it is not clear to us how one would incorporate the environmental effects into such embeddings.

Assuming that the Markov property is fulfilled it is possible to use these methods when the environment depends on the population. The perhaps most simple example corresponds to a naive model for social awareness where the entire population becomes aware of that a disease is present first when a fraction of the population have become infected. In this situation, the process would still be conditionally Markovian, and it is possible to derive closed, but implicit threshold conditions. But, these expressions would instead depend on the distribution of the time it takes until this fraction of the population has become infected, which in general is hard to obtain. In simple situations it might be possible to go via generating functions and relations between such and orthogonal polynomials similar to those used in [17].

Even though we only have treated non-endemic situations in the present paper, we can still determine whether or not a disease may become endemic or not, by using R_* from Theorem 3.1. A natural continuation of the present work would be to analyse endemic diseases under the influence of varying environments in more detail, both in terms of endemic levels, fluctuations, and the time to disease extinction.

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