Basic results for a stochastic model of epidemic spread

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Abstract

A simple "classical", model for epidemic spread in a finite, but large, population is studied. The model is a stochastic version of a deterministic model formulated by Kermack and McKendrick. In the stochastic version the spread starts when one newly infected individual enters a totally susceptible population. The aim of this paper is to describe how an epidemic may develop. We will here collect some well-known results and provide proofs of basic theorems. The presentation is focused on situations where the population in which the spread takes place is large. This makes it possible to use results derived from the study of branching processes. The discussion is illustrated by simple examples.

Keywords: Epidemic model, SEIR-model, Branching Processes.
1 Introduction

We will consider a model for epidemic spread of infections in closed populations. The model is a stochastic version of a deterministic model introduced by Kermack and McKendrick (1927). The stochastic formulation is given in section 2 where basic notation is defined and important, and well-known properties are summarized. We will, in particular, be concerned with situations where the population in which the spread occurs is large.

The model is formulated to account for that an infected person is infectious only during a finite random time interval and after infection is immune to further infections. One fundamental assumption of the model is related to how much infectivity an infected individual spreads in time after being infected. This infectivity is according to another assumption spread randomly over the susceptible part of the population.

The progress of the epidemic can be divided into three phases. These phases can be studied with different mathematical techniques.

As long as there is a small proportion of infected persons in the population the spread can be approximated by a branching process. Section 5 deals with properties of branching processes which are useful in the study of the epidemic process. In section 6 connections between the epidemic process and an approximating branching process are discussed.

When a non-negligible proportion of the population has been infected, if this ever happens, the branching process approximation is no longer valid. In this phase there are many active spreaders and there are sufficiently many susceptible individuals for the epidemic to persist. The process can then be analysed by mass-action tools, e.g. differential equations. This is discussed in section 7.

Finally the epidemic enters a fading off phase and will slowly die out, due to lack of susceptible persons, see section 8.

In section 9 some simple examples are studied and illustrated.

2 The model

We will assume that the population has \( n \) members, who initially are all susceptible to infection. It is assumed that at time \( t = 0 \), one recently infected person enters into a finite totally susceptible population. This may start a chain of infections that eventually causes an epidemic.

We will focus on results that are asymptotically valid when \( n \) is large. The progress of the epidemic is followed by counting the number of infected individuals. The counting process, \( N_n(t) \), tells how many individuals have
been infected up till time $t$. The sub index refers to the size of the population.

The spread, both as regards how large proportion of the population that is infected and the speed at which the epidemic grows, will depend on the infectiousness of the infected individuals. After being infected a person is assumed to have infectious contacts with randomly chosen members of the population according to a Poisson process with a random intensity $h(s)$. We will refer to these intensity functions as infectivity functions and assume that there exists a probability distribution over possible infectivity functions. The individual infectivity function, of the $i$'th infected is denoted $h_i$, and are assumed to be stochastically independent of other infectivity functions. A contact between an infected and a susceptible individual will always result in a secondary infection.

In order to avoid technical difficulties in the proofs we will make some, unnecessarily, restrictive assumptions. We assume that the random functions $h$ are non-lattice and have a common upper bound. We will also assume that the random variable $H(\infty) = \int_0^\infty h(s) ds$ has finite mean and variance.

A formal description of the epidemic model can be done through the intensity of the counting process $N_n$. The intensity is

$$\tilde{\lambda}(t) = \left(1 - \frac{N_n(t-)}{n}\right) \left[\int_0^t h_{N_n(s)}(t-s) dN_n(s) + h_0(t)\right]. \quad (2.1)$$

Looking at this representation we see that the process, $N_n$, can be viewed as a generalized pure death process where the death intensity at time $t$ depends on the history of previous deaths. This observation will be fruitful when we consider how large proportion of the population will stay uninfected during the entire epidemic.

We can also observe that as long as $N_n(t-)/n \approx 0$ we may approximate the process with a pure birth process where the birth intensity depends on the history of previous births. This approximation will be used to find out how fast the epidemic grows initially.

### 3 Notations and relations

In this section we will introduce parameters and properties of the model that has been proven important in previous studies. Theoretical results are in this section cited without proofs. Some proofs will be given or indicated in later sections.
The following notations are already introduced:

- \( n \): the size of the population.
- \( N_n(t) \): the number of infected up till time \( t \).
- \( h(s) \): the individual random infectivity function. The infectivity up till time \( s \) after infection is \( H(s) = \int_0^s h(s) ds \). The realized infectivity function of the \( i \)'th infected is denoted \( h_i \).

### 3.1 The basic reproduction number

The most important parameter deciding the strength of an infection is

- \( R_0 \): the basic reproduction number

\[
R_0 = \mathbb{E}(H(\infty)).
\]  

\( R_0 \) is often, somewhat loosely, referred to as the mean number of infections caused by one infected person in a totally susceptible population.

### 3.2 The final size

An important outcome of an epidemic is the final size, i.e., the proportion of the population that has been infected when the epidemic comes to an end. This is obviously a random quantity. It can be expressed as \( \pi_n = N_n(\infty)/n \).

Heuristically, it is clear that the final size, and its asymptotic properties, will not depend on how infectivity of the infected are distributed in time. This means that not the entire random function, \( h(s) \) has to be considered but only the (random) total infectivity, \( H(\infty) \).

We are particularly interested in large populations. The asymptotic final size is a mass action property and will only depend on mean properties of the model. It turns out that as \( n \to \infty \), the distribution of \( \pi_n \) tends to a two-point distribution. Either the epidemic dies out in an early stage and \( \pi_n \) is close to 0 or the epidemic grows large and ends with a positive proportion of the population infected. We define:

- \( \pi \): the asymptotic final size of the epidemic, i.e.,

\[
\pi = \lim_{n \to \infty} N_n(\infty)/n.
\]  

(3.2)
The final size will solve the equation
\[- \ln(1 - \pi) = R_0 \pi, \tag{3.3}\]
or equivalently
\[\pi = 1 - e^{-R_0 \pi} \tag{3.4}\]
If \(R_0 \leq 1\) this equation has the only solution \(\pi = 0\). If \(R_0 > 1\) there also exists a positive solution.

### 3.3 The probability for a large epidemic

There is a positive probability that the infected individual entering the population will not start a large outbreak. It is thus of interest to find
- \(p\): the asymptotic probability that the epidemic grows large, i.e.

\[p = \lim_{n \to \infty} P(N_n(\infty)/n > 0). \tag{3.5}\]

Also for this property it is clear that it is not important how the individual infectivity is distributed in time. This means that the infectivity functions, \(h\), only influences \(p\) through the total infectivity \(H(\infty)\). The epidemic dies out early causing only a small number of infected if the first infected are poor spreaders. Using the same argument as when proving that a branching process grows large (see Ball and Donnelly (1995)) we can prove that \(p\) is the positive solution of the equation

\[1 - p = L_{H(\infty)}(p), \tag{3.6}\]

where \(L_{H(\infty)}\) is the Laplace transform of the random variable \(H(\infty)\).

This equation has a positive solution if and only if

\[R_0 = E(H(\infty)) > 1. \tag{3.7}\]

### 3.4 The generation time density

To investigate time related properties of the epidemic spread we define
- \(g\): the generation time density. This function is the mean infectivity of an infected at time \(t\) after infection, normed to have total mass 1, see Svensson (2007) and Tomba et al. (2010). Thus

\[g(t) = \frac{E(h(t))}{R_0}. \tag{3.8}\]
The corresponding distribution function is

\[ G(t) = \int_0^t g(t) dt, \quad (3.9) \]

and the Laplace transform

\[ L^g(s) = \int_0^\infty e^{-st} g(t) dt. \quad (3.10) \]

We will also need the related function

\[ \tilde{L}^g(s) = \int_0^\infty te^{-st} g(t) dt = -\frac{dL^g(s)}{ds}. \quad (3.11) \]

### 3.5 The Malthus parameter

The generation time density is related to the speed at which an epidemic grows in a large population. When the epidemic is asymptotically large it grows, in the beginning, at an exponential rate equal to the Malthusian parameter.

- \( \alpha \); the Malthusian parameter defined as the solution of the equation

\[ 1 = R_0 \int_0^\infty \exp(-\alpha t)g(t) dt = R_0 L^g(\alpha) = E\left( \int_0^\infty e^{-\alpha t} h(t) dt \right). \quad (3.12) \]

There will exist a (unique) positive solution to this equation if and only if \( R_0 > 1 \).

The Malthus parameter in branching processes is discussed in section 5.1. That it is essential also for the progress of the epidemic is made clear in section 6.

### 3.6 The basic mean

Another important number is

\[ M_0 = \frac{1}{\alpha R_0 L^g(\alpha)} = -\frac{1}{\alpha R_0 \frac{d}{\alpha} L^g(\alpha)}. \quad (3.13) \]

The basic mean is together with the Malthus parameter an important parameter. In section 6 we prove that the epidemic process in the start is approximated by a branching process, \( B \). For this process we can prove that

\[ E(B(t)e^{-\alpha t}) \to M_0 \quad (3.14) \]

as \( t \to \infty \).
4 The phases of the epidemic

If the population in which the epidemic takes place is large we can divide the progress of the epidemic into three phases. Each phase has to be analysed with different methods.

In the first phase, when \( \frac{N_n(t)}{n} \leq \epsilon \) where \( \epsilon \) is a small number, the probability that an infected individual will have contact with an already infected and immune person is small. The progress is not slowed down by the presence of immunity and the epidemic process can be approximated by a branching process. Such processes have been studied in great detail. In section 5 we give a short account of results that are relevant for the present study.

In the next phase \( \epsilon < \frac{N_n(t-)}{n} < \pi - \epsilon \). The epidemic has then reached a level were a non-negligible proportion of the population is infected but there will also be a large number of active infectors. The process is then in a mass-action phase where the progress can be analysed using differential equations. We will call this the epidemic phase. It is discussed in section 7.

Finally there is a fading of phase, cf section 8, when the epidemic has almost reached it final state, i.e. \( \frac{N_n(t-)}{n} \geq \pi - \epsilon \). In this phase those still infectious has a small chance of contacting a susceptible individual. The spread will slowly fade out.

5 The branching process phase

In the start, i.e., before the facts that the population is finite and that the infection causes immunity influences the spread, we can approximate the epidemic process with a branching process. In this section we will study a branching process closely related to the epidemic process defined by 2.1. The connection between the two processes will be discussed in the next section.

We will define the branching process as a counting process, \( B \), with the intensity

\[
\int_0^t h_B(s)(t-s)dB(s) + h_0(t) = \int_0^t h_B(s)dB(s) + h_0(t).
\]

The branching process, \( B \), differs from the epidemic process, \( N_n \), since it is not influenced by immunity i.e., the term \( 1 - \frac{N_n(t-)}{n} \).

The process \( B \) is a special case of a so-called Crump-Mode-Jagers-process (i.e. a CMJ-process), see Crump and Mode (1968), Crump and Mode (1969), and Jagers (1975). It is usually described in a demographic setting as a model
where individuals have offspring according to a (general) point process during a, possible, random life time. It is natural to refer to the events in \( B \) as births and to talk of mothers and offspring or children (instead of infectors and infected). Such a representation causes here linguistic problems since terminology referring to infections and contacts does not fit well. However we will use the demographic terminology in this section and assume that the random infectious functions, \( h \), corresponds to the intensity of giving birth to children.

Crump-Mode-Jagers-processes have been studied in great detail and much is known of their properties. In the following subsection we will relate some useful results.

When this is done we will, in the following section, use the branching process to construct a related process that have the stochastic properties of the epidemic process. This will make it possible to use results for branching processes to derive results for the epidemic process.

5.1 Results for a branching process

Branching process have successfully been studied in great detail and much is known of their stochastic properties (see e.g. Harris (1963), Kimmel and Axelrod (2002), and Haccou et al. (2005)). In this paper we will in particular use two basic theorems. The assumptions made in this paper simplify derivations of results that are valid in more complex models. Proofs related to Crump-Mode-Jagers processes under more general conditions can be found in Doney (1972).

Let

\[
M(t) = E(B(t)).
\]

and

\[
Z(t) = B(t)e^{-\alpha t}
\]

where \( \alpha \) is the Malthus parameter.

The first theorem reveals the importance of the Malthusian parameter. It follows more generally from results in Feller (1971).

**Theorem 5.1**

\[
\lim_{t \to \infty} E(Z(t)) \to \frac{1}{\alpha R_0 L_0(\alpha)}.
\]

**Proof:**

We will use properties of Poisson processes. The development of the process depends when the initial mother gives birth to children. The function
\( h_0(t) \) defines the fertility of the first mother

\[
E(h_0(t)) = R_0g(t), \tag{5.5}
\]

We will first condition on \( h_0 \). With this conditioning the number of children of the initial mother up till time \( t \), denoted by \( \tilde{R}(t) \), is Poisson distributed with mean \( H_0(t) \). Due to properties of a Poisson process births occurs, in the interval \([0, t]\), at \( \tilde{R}(t) \) random times that are independent and distributed according to the density \( h_0(t)/H_0(t) \).

Due to the regenerative properties of the process (i.e. each birth starts a new independent stochastically identical process) we have

\[
E(B(t) | h_0, \tilde{R}(t)) = \tilde{R}(t) \left(1 + \frac{1}{H_0(t)} \int_0^t M(t - u)h_0(u)du\right). \tag{5.6}
\]

The last term equals 0 if \( H_0(t) = 0 \).

Removing the conditioning on \( \tilde{R}(t) \) we obtain:

\[
E(B(t) | h_0) = H_0(t) + \int_0^t M(t - u)h_0(u)du. \tag{5.7}
\]

It follows that

\[
E(B(t)) = M(t) = R_0 \left(G(t) + \int_0^t g(s)M(t - s)ds\right). \tag{5.8}
\]

Thus the mean of the branching process is only a function of \( R_0 \) and the generation time distribution \( g \).

If we multiply equation (5.7) with \( \exp(-ts) \) where \( s > \alpha \) we obtain

\[
\int_0^\infty E(B(t)e^{-st}) dt = \frac{R_0L^g(s)}{s} + R_0L^g(s) \int_0^\infty E(B(t)e^{-st}) dt. \tag{5.9}
\]

Thus

\[
\frac{1 - R_0L^g(s)}{s - \alpha} \int_0^\infty E(B(t)e^{-st}) dt = \frac{R_0L^g(s)}{s}. \tag{5.10}
\]

The right hand side of this equation tends to \( R_0L_g(\alpha)/\alpha = 1/\alpha \) (see 3.12) and

\[
\frac{1 - R_0L^g(s)}{s - \alpha} \to -R_0 \frac{dL^g(\alpha)}{ds}, \tag{5.11}
\]

9
as \( s \to \alpha \). Thus

\[
(s - \alpha) \int_0^\infty E \left( B(t) e^{-(s-\alpha)t} e^{-\alpha t} \right) dt \to -\frac{1}{\alpha R_0 \frac{dL^\alpha}{ds}} \quad (5.12)
\]
as \( s \to \alpha \).

A Tauberian theorem says that \( r \int_0^\infty Q(t) \exp(-rt) \to Q \) as \( r \to 0 \) if and only if \( Q(t) \to Q \) as \( t \to \infty \), This finally proves the theorem.

The above theorem describes how \( M(t) \) grows for large \( t \). We will later need an inequality valid for all \( t > 0 \).

**Corollary 5.1** There exists a constant \( \bar{K} \) such that

\[
M(t) \leq \bar{K} e^{\alpha t} \quad (5.13)
\]

for all \( t \geq 0 \).

The following theorem gives a relation for the Laplace transform of the limit distribution of \( B(t) \exp(-\alpha t) \). The proof again uses the properties of Poisson processes. Observe the similarity to results by Harris (1963) for slightly different models.

**Theorem 5.2**

\[
L_Z(s) = \lim_{t \to \infty} E(e^{-sZ(t)}),
\]
satisfies

\[
L_Z(s) = E \left[ \exp \left( -\int_0^\infty (1 - L_Z(se^{-\alpha u})) h(u) du \right) \right]. \quad (5.14)
\]

Together with (5.4) this defines \( L_Z(s) \) and the limit distribution of \( Z(t) \) as \( t \to \infty \).

**Proof:** First we analyse what happens conditional on \( h_0 \). The first mother has \( \tilde{R} \) children, where \( \tilde{R} \) is Poisson-distributed with mean \( H_0(\infty) \). The births occur at times which are independent and distributed according to the density \( \frac{h_0(t)}{H_0(\infty)} \). Thus

\[
E(e^{-sZ(t)} \mid h_0, \tilde{R}) = e^{-s\tilde{R} e^{-\alpha t}} \left[ \frac{1}{H_0(\infty)} \int_0^\infty E(e^{-sZ(t-u)e^{-\alpha u}}) h_0(u) du \right]^{\tilde{R}}. \quad (5.15)
\]

Removing the conditioning, first on \( \tilde{R} \), and then on \( h_0 \), and finally letting \( t \to \infty \) we derive the expression (5.14).
From equation (5.14) we can derive interesting limit results. E.g.

\[ L_Z(\infty) = q = 1 - p \]  

(5.16)

where \( p \) satisfies

\[ 1 - p = L_{H(\infty)}(p), \]  

(5.17)

This gives the probability, \( q = 1 - p \), that the process stays finite, see (3.6).

We can write

\[ L_Z(s) = q + pK(s) \]  

(5.18)

where \( K \) is the Laplace transform of the limit of \( Z(t) \) given that the process \( B(t) \) grows asymptotically large. Inserting (5.18) in (5.14) we obtain:

\[
q + pK(s) = \mathbb{E} \left[ \exp \left( -p \int_0^\infty (1 - K(se^{-\alpha u})h(u)du) \right) \right].
\]  

(5.19)

The asymptotic mean of \( Z(t) \) as \( t \to \infty \) is given by theorem 5.2. The second moment given that the process grows large, can be derived from the equation:

\[
K''(0) \left( 1 - R_0 L^g(2\alpha) \right) =
\]

\[
p(K'(0))^2 \mathbb{E} \left( \int_0^\infty e^{-\alpha u}h(u)du \right)^2.
\]  

(5.20)

Here

\[
\mathbb{E}(Z \mid \text{the process grows large}) = K'(0)
\]  

(5.21)

\[
\mathbb{E}(Z^2 \mid \text{the process grows large}) = K''(0)
\]  

(5.22)

### 5.2 Remark

It is, in general, difficult to derive an explicit solution of equation (5.19). However, it is possible in the special case where an infected person is infected with constant infectivity for random time, \( X \), i.e.

\[ h(t) = \lambda I(t \leq X). \]  

(5.23)

Under these assumptions:

\[ H(\infty) = \lambda X, \]  

(5.24)

\[ g(t) = \frac{\Pr(X > t)}{\mathbb{E}(X)}, \]  

(5.25)

\[ L^g(s) = \frac{1 - L_X(s)}{s\mathbb{E}(X)}. \]  

(5.26)
With the definitions given in section 3 we find that
\[ p\lambda = \alpha, \quad (5.27) \]
and we can verify that the Laplace-transform
\[ K(s) = \frac{1}{1 + \gamma s} \quad (5.28) \]
satisfies the equation (5.14).

The asymptotic expression of the mean of \( Z(t) \) given that it is asymptotically large is
\[ \gamma = \frac{1}{p\alpha R_0 L^*(\alpha)}. \quad (5.29) \]
Thus the limit given that the process grows large is exponential distributed with intensity \( 1/\gamma \).

6 Deriving the epidemic process from the branching process

Two important features distinguishes the epidemic process from the branching process:

- The epidemic takes place in a finite population,
- In the epidemic model the growth of the process is slowed down by immunity.

We will develope an idea from Ball and Donnelly (1995) to generate an epidemic process by combining the branching process with another independent process. In this process we choose random numbers independently and uniformly from \( 1, \ldots, n \). These numbers, of course, correspond to the individuals in the population. We will then delete events in the branching process in such a way that the remaining events corresponds to an epidemic process. In time sequence we will attach the random integers to the non deleted events of the branching process. If the number chosen occurs for the first time we will keep the event. If it has already been chosen before the event is deleted as well as all offspring in the branch starting from this event. This is intended to describe the effect of immunity. The events that finally remains correspond to infections in the epidemic process \( N_n \).

With this construction \( N_n \) only jumps when \( B \) jumps.
Let $C_1^n$ denote the value of $B$ then it first happens that a person is chosen for the second time. If $B(t) \leq C_1^n$ the processes $B$ and $N_n$ coincides and they share properties until this happens.

Note that the problem of finding the probability distribution of $C_1^n$ is analogous to the well-known birthday problem. The problem there is to find the probability that at least two persons in a group of $r$ persons have the same birthday (cf Feller (1971)).

$$P(C_1^n > r) = \prod_{i=0}^{r} (1 - i/n) = \frac{n!}{(n - r)! n^r}$$ (6.1)

Applying Stirlings formula we find that the probability $P(C_1^n > r)$ tends to 1 as $n \to \infty$ if $r = n^a$ where $a < 1/2$. Thus when $N_n(t) = B(t) \leq n^a$ where $a < 1/2$ the epidemic process has asymptotically the same properties as the branching process.

We will later need to be able to use the branching process as an approximation for a longer time. To do this we will approximate how many future births are removed in the procedure described above. Let $I_v^n$ be an indicator that equals 1 if a number is chosen at the $v$’th birth has a number that has been chosen before. Otherwise it will equal 0. The indicators are independent of the branching process. Now let $B_v$ denote the (sub) branching process started by the $v$’th event, then

$$N_n(t) = B(t) - \sum_{v=1}^{B(t)} I_v^n B_v(t - \tau_v).$$ (6.2)

This representation may be used to construct close approximations of the epidemic process. However, we will here only use it to study for how long the branching process is a “good” approximation of the epidemic process.

We start by observing that at the $v$’th event less than $v$ numbers have been chosen. Thus

$$E(I_v^n) \leq \frac{v}{n}.$$ (6.3)

The right-hand size is, for fixed $t$, the sum of a random number of random variables. Using lemma 5.1 we find that for each of the summands

$$e^{-\alpha t} E(I_v^n B_v(t - \tau_v) \mid \tau_v) \leq \frac{\tilde{K}}{n} v e^{\alpha (t - \tau_v)} = \frac{\tilde{K}}{n} v e^{-\alpha \tau_v}.$$ (6.4)

Removing the conditioning on $\tau_v$ we find

$$e^{-\alpha t} E(I_v^n B_v(t - \tau_v)) \leq \frac{\tilde{K}}{n}.$$ (6.5)
for some constant $\tilde{K}$.

Since the event $B(t) \geq v$ only depends on what happens in the branching process before time $\tau_v$ and $B_v(t - \tau_v)$ does not we can apply Wald’s lemma and find

$$e^{-\alpha t}E(B(t) - N_n(t)) \leq \frac{\tilde{K}}{n}E(B(t)). \quad (6.6)$$

Now

$$e^{-\alpha t}(B(t) - N_n(t)) = \left(1 - \frac{B(t)}{B(t)}\right)n. \quad (6.7)$$

It is known that of $B(t)\exp(-\alpha t)$ converges almost surely, as $t \to \infty$, towards the random variable $Z$ which is positive if the branching process grows large (see Cohn (1985)). The difference $B(t) - N_n(t)$ is always non-negative and also increasing in $t$. This means that we can apply the Markov inequality and draw the following conclusions:

- $\frac{N_n(t)}{B(t)} \to 1 \quad (6.8)$
  in probability as $n \to \infty$ for all $t$ such that $B(t) \leq n^b$ where $b < 1$.

- for any $\epsilon > 0$ there exists a $\eta$ such that
  $$\frac{N_n(t)}{B(t)} \geq 1 - \epsilon \quad (6.9)$$
  as $n \to \infty$ for all $t$ such that $B(t) \leq \eta n$.

### 6.1 Time in the branching process phase

Assume that the epidemic grows large and a non-negligible proportion are infected, then at some finite time $\tau_\epsilon$ the number of infected will reach the level $n\epsilon$. If $\epsilon$ is sufficiently small the process $N(t)$ can be well approximated by a branching process up till that time. After that the process has to be studied by other methods.

Using the branching process approximation we find that

$$N(\tau_\epsilon)\exp(-\alpha \tau_\epsilon) = n\epsilon \exp(-\alpha \tau_\epsilon) \approx Z \quad (6.10)$$

where $Z$ is a random variable. This implies that

$$\tau_\epsilon = \frac{\ln(n)}{\alpha} + \tilde{Z}. \quad (6.11)$$

where $\tilde{Z}$ is a finite random number.

More exact results (and more rigid analysis) valid in special models can be found in Barbour (1975) and Svensson (1995).
7 The epidemic phase

In this section we will study the epidemic process after it has reached a level where the branching process is no longer a good approximation.

We start by defining the new counting process \( \bar{N}_n(s) \) that counts the number of infections that take place after \( \tau_n \), i.e., \( \bar{N}_n(s) = N_n(s + \tau^n) - N_n(\tau^n) \). This new counting process has the intensity

\[
\bar{\lambda}(s) = (1 - \epsilon - \frac{\bar{N}_n(s-)}{n}) \int_0^s h_{N_n(v+\tau^n)}(s-v)dN_n(v) + (1 - \epsilon - \frac{\bar{N}_n(s-)}{n}) \left[ \int_0^{\tau^n} h_{N_n(u)}(s + \tau^n - u)dN_n(u) + h_0(s + \tau^n) \right].
\]

(7.1)

The first right-hand term gives the intensities of infections caused by those infected after \( \tau^n \) and the second term the intensity caused by those infected before that time but occurring after \( \tau^n \). It is thus necessary to investigate the effect of the remaining infectivity spread by those infected before \( \tau^n \) after that time.

7.1 Remaining infectivity

We will now consider, using an heuristic argument, how much infectivity has been spread in the population at time \( \tau^n \), when \( \epsilon n \) individuals have been infected and how much infectivity is still remaining to be spread after time \( \tau^n \) by those infected before \( \tau^n \).

The infections occurs according to a Poisson process. Thus we can assume that it requires, in mean, a total of infectiousness \( \epsilon n \) to produce \( \epsilon n \) infections if we can disregard effects of immunity. The first \( \epsilon n \) infected can, in mean, generate the infectiousness \( R_0 \epsilon n \). Thus, at the time \( \epsilon n \) individuals has been infected, if that happens, there still remains, in mean, \( (R_0 - 1)\epsilon n \) infectiousness to be spread from those already infected. We will consider how this infectivity is distributed in time after \( \tau^n \). This, of course, depends on when those infected before \( \tau^n \) are infected.

We will try to obtain a useful expression of

\[
\int_0^{\tau^n} h_{N_n(u)}(s + \tau^n - u)dN_n(u) + h_0(s + \tau^n).
\]

(7.2)
We start by rewriting the expression as

$$R_0 e^{\alpha \tau_n} \left[ \int_0^{\tau_n} g(s + \tau_n - u)e^{-\alpha(\tau_n-u)}e^{-\alpha u}dN_n(u) \right] + h_0(s + \tau_n). \quad (7.3)$$

We will use that for large values of $u$

$$\frac{e^{\alpha \tau_n}}{N_n(\tau_n)} \frac{N_n(u)}{e^{\alpha u}} \approx 1 \quad (7.4)$$

since $Z(u)$ converges almost surely, and

$$dN_n(u) \approx \alpha N_n(u) du. \quad (7.5)$$

Inserting these approximations in (7.3) we find, that the infectivity remaining from those infected before time $\tau_n$ spread out in time is $R_0 e^{\alpha \tau_n}$$\cdot$$Rem(s)$ where

$$Rem(s) \approx \left[ \int_0^{\tau_n} g(s + \tau_n - u)e^{-\alpha(\tau_n-u)}\alpha du \right]. \quad (7.6)$$

Since $\tau_n \to \infty$ as $n \to \infty$ according to (6.11) we can use the approximation

$$Rem(s) = \alpha \int_0^{\infty} g(s + t)e^{-\alpha t} dt \quad (7.7)$$

if $n$ is large.

Integrating the second right-hand term of this equation we get

$$R_0 \int_0^{\infty} Rem(t) dt = R_0 \alpha \int_0^{\infty} (1 - G(t))e^{-\alpha t} dt = R_0 - 1 \quad (7.8)$$

which corresponds to the calculation of the amount of the remaining infectiousness made at the beginning of the section. Also observe that

$$R_0 Rem(0) = \alpha. \quad (7.9)$$

This has to be the case, since the process is assumed to have the Malthus parameter $\alpha$. 

7.2 Differential equation approximation

Now let

\[ x(t) = \frac{\bar{N}(t)}{n}. \]  \hspace{1cm} (7.10)

If we take expectations we find that if \( n \) is large

\[ \frac{x'(t)}{1 - (x(t) + \epsilon)} = R_0 \int_0^t g(t - v)dx(v) + R_0 \epsilon \int_0^\infty g(s + t)e^{-\alpha t}dt. \] \hspace{1cm} (7.11)

If we solve this differential equation and let \( \epsilon \to 0 \) we have an expression for the deterministic trajectory for the progress of the epidemic in the nearly deterministic phase provided the population is large or asymptotically as \( n \to \infty \).

8 The Fading off phase

In the final phase most contacts taken by an infectious individual will be with an immune individual and will not result in further spread of the epidemic. This implies that the process will be essentially random. However we know from (3.3) that finally the epidemic, if it grows large, will end up with the proportion, \( \pi \), members infected.

The fading-off phase will last from the time, \( t = \tau_{\pi - \epsilon} \), when \( N(t) = (\pi - \epsilon)n \), until it stops. Here \( \epsilon \) can be chosen arbitrarily small if \( n \to \infty \).

In the start the epidemic behaves as a branching process, which is a birth-and-death process with, if \( R_0 > 1 \), a strong bias to births (i.e. new infections). The final phase the process behave like a birth-and-death process with more deaths (i.e. individuals becoming non-infectious and immune) than births. Of course, it is of interest to study the final phase of the epidemic, but it will not be done in any detail here.

9 Examples

We will illustrate the theory described above and the calculations necessary by considering different sets of assumptions of the infectivity functions.

In the first two examples the infected individuals are assumed to have a constant infectivity during the infectious period. A consequence of this is, according to section 5.2, that \( N_n(t)e^{-\alpha t} \) is asymptotically exponential distributed. The asymptotic mean of this exponential distribution is \( M_0/p \).
In the third example a random infectious period with constant infectivity starts after a (random) latent period during which infections is not transmitted. Finally an example with non constant infectivity is considered.

9.1 Constant infectious time

We assume that the infection is spread with constant intensity, \( \lambda \), during the non-random time \( k \). Such a process is sometimes referred to as a (continuous time) Reed-Frost process. Thus

\[
h(t) = \lambda I(t < k). \quad (9.1)
\]

We can now calculate:

\[
R_0 = \lambda k, \quad (9.2)
\]

\[
g(t) = \frac{I(t < k)}{k}, \quad (9.3)
\]

\[
L^g(s) = \frac{1 - e^{-sk}}{sk}, \quad (9.4)
\]

and

\[
\tilde{L}^g(s) = \frac{1 - e^{-sk} - sk e^{-sk} - sk}{s^2 k}. \quad (9.5)
\]

The Malthus parameter, \( \alpha \), solves the equation

\[
\lambda (1 - e^{-\alpha k}) = \alpha. \quad (9.6)
\]

Furthermore

\[
p = \frac{\alpha}{\lambda}, \quad (9.7)
\]

\[
\pi = p, \quad (9.8)
\]

and

\[
M_0 = \frac{1}{1 - R_0 + \alpha k} \quad (9.9)
\]

Finally

\[
Rem(s) = \frac{1 - e^{-\alpha(k-s)^+}}{k}. \quad (9.10)
\]
9.2 Exponentially distributed infectious time

The basic assumptions are that the infectivity is constant during an exponential infectious period. Thus

\[ h(t) = \lambda I(t < X) \]  

where \( X \) is an exponential distributed random variable with intensity \( \beta \).

This gives:

\[ R_0 = \frac{\lambda}{\beta}, \]  

\[ g(t) = \beta e^{-\beta t}, \]  

\[ L^g(s) = \frac{\beta}{\beta + s}, \]  

\[ \tilde{L}^g(s) = \frac{\beta}{(\beta + s)^2}, \]  

\[ \alpha = \lambda - \beta. \]  

\[ p = \frac{\alpha}{\lambda} = 1 - \frac{1}{R_0}, \]  

\[ M_0 = \frac{R_0}{R_0 - 1} = \frac{\lambda}{\alpha}. \]  

\[ Rem(s) = \frac{\alpha \beta}{\lambda} e^{-\beta s}. \]

It should be observed that

\[ R_0 Rem(s) \equiv \alpha g(s)/\beta. \]  

This implies that at time \( \tau \), there are \( n e \alpha / \beta = (R_0 - 1)e n \) infectious individuals each being as infective as a newly infected individual. This is, of course, due to the “lack of memory” property that characterizes the exponential distribution.
9.3 Exponentially distributed latent and infectious times

The basic assumptions are that the latent time, $Y$, is exponentially distributed with intensity $\delta$, and the infectious time, $X$, is exponentially distributed with intensity $\beta$. $X$ and $Y$ are assumed to be independent. The infectivity is $\lambda$. This gives:

$$h(t) = \lambda I(Y < t < X + Y).$$ \hspace{1cm} (9.21)

$$R_0 = \frac{\lambda}{\beta}.$$ \hspace{1cm} (9.22)

$$g(t) = \frac{\delta \beta}{\delta - \beta} (e^{-\beta t} - e^{-\delta t}).$$ \hspace{1cm} (9.23)

If $\beta = \delta$ then

$$g(t) = \beta^2 t e^{-\beta t}.$$ \hspace{1cm} (9.24)

$$L^g(s) = \frac{\delta \beta}{(\delta + s)(\beta + s)},$$ \hspace{1cm} (9.25)

$$\tilde{L}^g(s) = \frac{\delta \beta + \beta + 2s}{(\delta + s)^2(\beta + s)^2}.$$ \hspace{1cm} (9.26)

The Malthus parameter, $\alpha$, solves the equation

$$\alpha^2 + (\delta + \beta)\alpha - \delta(\lambda - \beta) = 0.$$ \hspace{1cm} (9.27)

$$p = \frac{\lambda - \beta}{\lambda} = 1 - \frac{1}{R_0}.$$ \hspace{1cm} (9.28)

$$Re^m(s) = \frac{\beta \delta \alpha}{\delta - \beta} \left( e^{-\beta s} - e^{-\delta s} \right).$$ \hspace{1cm} (9.29)

If $\beta = \delta$ then

$$Re^m(s) = \alpha \beta^2 e^{-\beta s} \left( \frac{1}{(\beta + \alpha)^2} + \frac{s}{\beta + \alpha} \right).$$ \hspace{1cm} (9.30)
9.4 Decreasing infectivity

We assume that the infectious period, $X$, is exponentially distributed with intensity $\beta$, and

$$h(t) = \lambda(1 - \frac{t}{X})I(t < X)$$  \hspace{1cm} (9.31)

According to this assumption the infectivity decreases linearly during the infectious period. The following expressions involves exponential integrals:

$$E_n(t) = \int_1^\infty \frac{e^{-tu}}{t^n} dt = s^{n-1} \int_1^\infty \frac{e^{-u}}{u^n} du.$$  \hspace{1cm} (9.32)

For properties of these functions and relations between them see Abramowitz and Stegun (2012) or The Digital Library of Mathematical Functions NIST (2019).

Now

$$R_0 = \frac{\lambda}{2\beta},$$  \hspace{1cm} (9.33)

$$p = 1 - \frac{1}{R_0}.$$  \hspace{1cm} (9.34)

$$g(t) = 2\beta \left[ e^{-\beta t} - \beta t \int_\beta^\infty \frac{e^{-u}}{u} du \right] = 2\beta E_2(\beta t),$$  \hspace{1cm} (9.35)

$$L^a(s) = 2\frac{\beta^2}{s^2} \left[ \frac{s}{\beta} - \ln(1 + \frac{s}{\beta}) \right],$$  \hspace{1cm} (9.36)

and

$$\tilde{L}^a(s) = 2\frac{\beta(2 + s/\beta)}{s^2(1 + s/\beta)} - 4\frac{\beta^2}{s^3} \ln(1 + s/\beta).$$  \hspace{1cm} (9.37)

The Malthus parameter, $\alpha$, solves the equation

$$\frac{\alpha}{\beta} - \ln \left(1 + \frac{\alpha}{\beta} \right) = \frac{\alpha^2}{\beta^2} \frac{1}{2R_0}.$$  \hspace{1cm} (9.38)

Furthermore

$$Rem(s) = 2\beta \left[ E_2(\beta s) - \frac{\beta}{\alpha} E_1(\beta s) + \beta e^{\alpha s} E_1((\beta + \alpha)s) \right]$$  \hspace{1cm} (9.39)
Figure 10.1: \( g(t) \) for example 1-4

10 Illustrated examples

To illustrate the models exemplified above we have chosen parameter values that give the same basic reproduction number and the same Malthus parameter. We have aimed at the values:

\[
R_0 = 2,
\]

and

\[
\alpha = 1,
\]

The final size does only depend on the basic reproduction number. In this case the final size is \( \pi = 0.7968 \) (see 3.3). To obtain these values we have to choose corresponding parameter values in the four different examples.

For each example the generation time densities are illustrated in figure 10.1. The Rem-functions that gives the remaining infectivity to be spread after time \( s \) from those infected before \( s \) is presented in figure 10.2.

Figure 10.3 illustrates simulated epidemic functions based on the four examples. Here the population size, \( n = 10,000 \) and the epidemics presented grows large. This implies that they stop with approximately \( 10000\pi = 7968 \) infected individuals.
The main differences between the curves depends on the randomness in the start of the epidemics. Even if the Malthus parameter is the same in the four cases we know from section that $N_n(t)e^{-at}$ for large $t$ is random with asymptotic mean $M_0/p$. In fact in example 1 and 2 its distribution is approximately exponential. This is not the case for example 3 and 4. According to the expressions in section 5.20 for both this examples the distribution is over-dispersed, i.e., the standard deviation is larger than the mean.

Beside the differences at the start the epidemic curves are quite similar. This is, of course, due to the fact that we have chosen parameter values so that the final sizes, i.e. $\pi = 0.7968$ are the same in the four examples and that the initial exponential growth rates, i.e. the Malthus parameters, also are the same. In figure 10.4 the randomness in the initial phase is taken away. It shows how the epidemics develop after 10% of the population has been infected. In the fading of phase randomness is again considerable.

10.1 Parameter values in the examples

Example 1: In the model with no latent time and constant infectious time we choose $\lambda = 1.255$, and $k = 1.593$. This gives the probability for a large
Figure 10.3: Trajectories for epidemics as described by example 1–4

Figure 10.4: Trajectories for proportion of infected after 10 % of the population has been infected in example 1–4
epidemic

\[ p = 0.7968, \quad (10.1) \]

and

\[ M_0 = 1.6846. \quad (10.2) \]

According to the remark in section 5.2 the limit distribution of the approximating branching process normed by \( e^{-\alpha t} \) is exponential with mean \( M_0/p = 2.114 \).

**Example 2:** In the model with no latent time and exponential distributed infectious times we choose \( \lambda = 2 \) and \( \beta = 1 \).

The probability for a large epidemic is

\[ p = 0.5, \quad (10.3) \]

and

\[ M_0 = 2. \quad (10.4) \]

According to the remark in section 5.2 the limit distribution of the approximating branching process normed by \( e^{-\alpha t} \) is exponential with mean \( M_0/p = 4 \).

**Example 3:** In the model with exponential latent and infectious times we choose first \( \beta = 2, \lambda = 4 \), and \( \delta = 3 \).

The probability for a large epidemic is

\[ p = 0.5, \quad (10.5) \]

and

\[ M_0 = 1.7143. \quad (10.6) \]

However the limit distribution of the approximating branching process normed by \( e^{-\alpha t} \) has mean \( M_0/p = 3.43 \). However, it is not exponential distributed in with these parameter values.

**Example 4:** In this model the parameter values \( \beta = 0.6159 \) and \( \lambda = 2.463 \) gives the desired values of \( R_0 \) and \( \alpha \).

This gives the probability for a large epidemic

\[ p = 0.5, \quad (10.7) \]

and

\[ M_0 = 2.103. \quad (10.8) \]

The limit distribution of the approximating branching process normed by \( e^{-\alpha t} \) has mean \( M_0/p = 4.01 \). However, it is not exponential distributed.
In figure 10.1 the generation time densities for the four examples are illustrated and in figure 10.2 the functions $Rem(s)$.
References


