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Some Approximation Results Concerning Near Critical Epidemics

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Abstract

In this thesis we will look at two epidemic models, the SIR and SIS models, and study their behaviour when we make them near critical. This will be done by comparing asymptotic results for near critical epidemics under a certain scaling, with exact recursively calculated results under the same scaling. We will show graphically that for both models the asymptotic result is useful since the recursive algorithm will converge relatively quickly, i.e. for small populations of susceptibles (≈ 10000).

Sammanfattning

I den här uppsatsen tittar vi på två epidemimodeller, SIR och SIS modellerna, och undersöker vissa egenskaper för dessa modeller då vi gör dem nära-kritiska. Detta görs genom att jämföra asymptotiska resultat under en särskilld kritisk skalning, med exakta rekursiva resultat under samma kritiska skalning. Vi visar grafiskt att för båda modellerna är de asymptotiska resultaten användbara, eftersom de rekursiva algoritmerna konvergerar snabbt, dvs. för små populationer (≈ 10000).

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Contents

1 Some concepts concerning epidemics

When you hear the word epidemic, you probably think of an outbreak of some disease in a population, and how the disease is spreading. Another thing commonly associated with the word epidemic is the question of vaccination. Is it possible to vaccinate? How many people need to be vaccinated to avoid an outbreak? And so on.

When you talk about the outbreak of an epidemic, there are two possible outcomes. One outcome is that there will only be a small outbreak with only a few infected individuals and that the disease only persists in the population for a short time period. The other possible outcome is that there will be a big outbreak, an *explosion*, with a large group of infected individuals and that the disease persists in the population for a relatively long time period.

If we try to figure out how a disease is spreading in a population, there are many factors that play an important role, such as how infectious the disease might be, how the disease is transmitted between individuals, for how long an infected individual might infect others, how individuals make contact in the population etc. This leads to an almost innumerable amount of possible factors that might affect the spread of a disease and it's persistance. If we want to try to model an epidemic, we realize that the reality is too complex and that we need to make some assumptions to simplify our model, but these assumptions will also affect which phenomenons that we can observe.

The first thing we can do is to look at the specific disease that we want to model and identify all different states which an individual in our population can visit, i.e. if the individual is ill and infectious, if the individual is well and susceptible or if the individual is immune or for some other reason not possible to infect. We can now look at this as a stochastic process, where an individual can visit some or all of these states, depending on the nature of the disease, and how she is allowed to move between these states.

An assumption that we will use in our model that will limit the credibility is that the population is closed, i.e. we don't allow individuals to immigrate or emmigrate. But on the other hand this assumption will make it possible for us to examine how large an outbreak is, given that an infected individual will become immune or in some other way not possible to infect again. This is called the *final size* of an epidemic, which is the number of individuals that have been infected. If we would have had the more realistic case, where the population isn't closed (or if individuals can't become immune), there isn't an unambiguous way to define what the final size is, since we could have more susceptible individuals at the end of the epidemic, than we had in the begining.

Another assumption that we will make is that we don't allow any social

structures, i.e. all individuals in the population are mixing homogeneously. This is also an assumption that will make our model loose some credibility, but it will make our calculations easier.

Our last assumption is that we will assume that the time period that a given individual is infected will follow an exponential distribution, so our model will become Markovian. This assumption hasn't any foundation in epidemiology, but it will make our model easier to handle.

In this thesis we will concentrate on two simple models. The first model that we will have a look at is the so called standard SIR epidemic, or simply the SIR epidemic. In this model the individuals can be in any of the states, susceptible (S), infectious (I), recovered or immune and then removed (R). For this model it is possible to both calculate the final size distribution of the epidemic and the duration of the epidemic.

The second model is the SIS model, and here an individual can only be in any of the two states susceptible or infectious. Therefore it's only possible to calculate an expression for the mean time to extinction.

As mentioned in the begining of this chapter, an epidemic can have two different outcomes. Either the epidemic will die out in a short time period and only a few individuals get infected, or it will persist for a longer time period and a large group of individuals get infected. These are two drastically different outcomes, which also will affect our results concerning epidemics drastically. In this thesis we will look at epidemics that are just on the border of these two outcomes. Such an epidemic is called *near critical*, and we will define this state later on, and see how this can be expressed in our models.

For more information about these two models or about stochastic epidemic modeling in general, see Andersson and Britton [1].

2 Some basic facts about diffusion processes and diffusion approximations

To get a feeling for how diffusion approximations work, we here give a brief heuristic treatment of the subject.

Let $X(t)$ be a discrete valued stochastic process in continuous time. If we want to make an asymptotic approximation of our process $X(t)$, it is reasonable to do so with a continuous process $U(t)$, i.e. $U(t)$'s sample paths are continuous functions of t with probability 1. This is done by first rescaling our process $X(t)$ in such a way that the jump sizes decrease and the jump frequency increases, so that when taking limits our process will tend towards continuity (in sample path). But that's not all. We also need to have finite

first and second moments and negligable higher order moments, when taking limits, under our new scaling.

But what is a diffusion? And why does our discrete process converge to a diffusion process?

A diffusion is a stochastic process with continuous paths, i.e. with probability 1, that posseses the strong Markov property.

To see why our discrete process will converge to a diffusion we will look at the first and second moments of the diffusion. These moments are called the infinitesimal *drift* and *variance* coefficients, respectively, and are denoted $\mu(x)$ and $\sigma^2(x)$. This gives us that the class of diffusion processes is quite liberal, in the sense that we for example can let our process be location dependent via it's drift. An example of this is the Ornstein-Uhlenbeck process which we will encounter later on in this thesis. Another example of a diffusion is the standard Wiener process.

When taking limits, the first and second moments of our scaled process $X(t)$ will converge to the infinitesimal drift and variance coefficients, with which we identify our diffusion process that we can use as an asymptotic approximation of our discrete process.

For more elaborate treatment of these topics, see Karlin and Taylor [7].

3 SIR model

In the SIR model we start with a closed population that doesn't have any social structure. Initially we have n susceptible individuals and m infected individuals. Each infected individual stays infectious for an exponentially distributed time period, with mean ι. The case we will have a look at has $\iota = 1$. An infected individual will then make contact with a given individual according to a Poisson process with the intensity λ/n. All infection times and Poisson processes are assumed independent.

We can now look at the SIR model as a two dimensional Markov process where (S_t, I_t) denote the number of susceptible and infected individuals at time t. The reason for this being only a two dimensional process follows from the fact that the population is closed, i.e. $R_t = n + m - S_t - I_t$.

The jumps possible for our process (S, I) to make with respective transition intensities are

$$\begin{array}{ll} (s,i) \to (s-1, i+1) & \text{with intensity } \lambda si/n \\ (s,i) \to (s, i-1) & \text{with intensity } i \end{array}$$

Since the population is closed, we are able to calculate the distribution for the final size, which is the total number of infected individuals Z, but we

can also get the distribution for the time to absorption, i.e. the time it takes until $I_t = 0$ for the first time.

But first we have to look at how we can make our model near critical.

As previously mentioned, our epidemic can either die out fast and only a few individuals get infected or it can persist for a longer time period with a large number of infected. These two outcomes are usually referred to as the *sub critical* and the *super critical* case.

But how can we make our model near critical? To make this a bit clearer we now introduce the quantity R_0 called the *basic reproduction number*. R_0 is defined to be the expected number of individuals that an infected individual infects in a large population, or more formally $R_0 = \lambda\iota$ ($= \lambda$ in our case).

One can show that

$$\frac{Z'_n}{n} \to \tau \text{ when } n \to \infty$$

where $Z'_n = Z_n + m_n$ and $m_n/n \to \mu$, where τ is the solution to the equation

$$1 + \mu - \tau = e^{-\lambda\tau} \ (\heartsuit)$$

This gives us that τ is the proportion of individuals who have been infected, while the left side of ($\heartsuit$) represent the proportion of individuals that have avoided infection. We then have that $e^{-\lambda\tau} \approx$ the probability that a given individual will avoid infection from a given infected individual.

Depending on which values we let the parameter λ take, we can obtain the sub and super critical case, by letting $\lambda < 1$ and $\lambda > 1$ respectively. Therefore it hopefully seems reasonable to get the near critical case when $\lambda \approx 1$, and now we see why λ some times is referred to as the *critical parameter*.

3.1 Asymptotic results and diffusion approximations under a critical scaling

In Greenwood et. al [5] it is shown that there exists an asymptotic distribution for the final size Z of the near critical epidemic under a certain 'critical scaling'. We will now give a very brief resumé of how this is done, using diffusion approximations. For more details, see Greenwood et. al [5], Martin-Löf [8], [9] and [10].

If we start by looking at our transition intensities and possible jumps

$$\begin{array}{ll} (s,i) \to (s-1, i+1) & \text{with intensity } \lambda si/n \\ (s,i) \to (s, i-1) & \text{with intensity } i \end{array}$$

and replace our old time scale t by it, i.e. when $I_t = i$ we get the clock rate idt instead of dt. This gives us

$$\begin{array}{ll} (s,i) \to (s-1, i+1) & \text{with intensity } \lambda s/n \\ (s,i) \to (s, i-1) & \text{with intensity } 1 \end{array}$$

To obtain suitable diffusion approximations for S_t and I_t, we need to have a look at the first and second expected increments moments.

Let $\Delta S = S_{t+\Delta t} - S_t$ and $\Delta I = I_{t+\Delta t} - I_t$. We then get the first expected increments moments

$$\begin{cases} E_{s,i}(\Delta S) &= -\frac{\lambda s}{n}\Delta t, \\ E_{s,i}(\Delta I) &= \left(\frac{\lambda s}{n} - 1\right)\Delta t, \end{cases}$$

and the second expected increments moments

$$\begin{cases} E_{s,i}(\Delta S^2) &= -\frac{\lambda s}{n}\Delta t, \\ E_{s,i}(\Delta I^2) &= \left(\frac{\lambda s}{n} + 1\right)\Delta t, \\ E_{s,i}(\Delta S \Delta I) &= -\frac{\lambda s}{n}\Delta t. \end{cases}$$

Since $\frac{S_t}{n} \approx 1$ when $t \approx 0$, the following approximation of S_t and I_t is proposed:

$$\begin{cases} \frac{S_t}{n} &= 1 + \frac{X_t^n}{n^\alpha} \\ I_t &= n^\beta Y_t^n \end{cases}$$

where

$$\lambda = 1 + \frac{a}{n^\gamma}.$$

It is shown, that by choosing α, β and γ as $\alpha = \beta = \gamma = 1/3$, we can obtain a diffusion approximation to Y^n. By taking the limits as $n \to \infty$, on the time scale $s = t/n^{2/3}$, it can be shown that X_s^n and Y_s^n converge to the diffusions X_s and Y_s.

When we use the proposed values of α, β and γ, and only keep the dominating terms in the expressions for the first and second increments moments of X_t^n and Y_t^n we get

$$\begin{cases} E\Delta X^n &= n^{-2/3}\Delta t \\ E\Delta Y^n &= n^{-2/3}(a-x)\Delta t \end{cases}$$

and

$$\begin{cases} E(\Delta X^n)^2 & = n^{-4/3}\Delta t \\ E(\Delta Y^n)^2 & = 2n^{-2/3}\Delta t \\ E(\Delta X^n \Delta Y^n) & = n^{-1}\Delta t. \end{cases}$$

The stochastic differential equations defining X_s and Y_s are hence

$$\begin{cases} dX_s & = ds \\ dY_s & = (a - X_s)ds + \sqrt{2}dW_s \end{cases}$$

on the time scale $s = t/n^{2/3}$. This gives us that

$$\begin{cases} X_s & = s \\ Y_s & = b + as - s^2/2 + \sqrt{2}W_s, \text{ where } b = lim_{n,m\to\infty} m/n^{1/s} \end{cases}$$

i.e the final size of the epidemic is the value of $X_s = s$ at T, the first time when $Y_s = 0$, which is the first time the process $\sqrt{2}W_s$ hit the parabolic barrier $b + as - s^2/2$.

Hence we have that $Z/n^{2/3} \to T$ as $n \to \infty$.

The density of T valid for a wide class of models has been calculated in Martin-Löf [9].

Using Airy functions this expression can be written as

$$\begin{aligned} -\frac{dP_x(T > t)}{dt} &= \exp\{-w((t-a)^3 + a^3)/6 - ax\} \\ &\times \int_{-\infty}^{\infty} \frac{e^{tu}(B(u)A(u-x) - A(u)B(u-x))}{\pi(A^2(u) + B^2(u))} du \ (\spadesuit) \end{aligned}$$

where $A(u) = Ai(cu), B(u) = Bi(cu), w = \frac{1}{\sigma^2}, c = (\frac{2}{w})^{1/3}$ and $x = wb > 0$.

To get the Markovian model we see from the Itô equation that $\sigma = \sqrt{2}$ which gives us that $w = 1/2$.

In Dhlakama [2] the properties of ($\spadesuit$) are investigated using numerical approximations, looking at it's behavior for different choices of the parameters a and b and $c = 1$, i.e. $w = 2$ (note that this isn't the Markovian model). It turns out that under certain parametrizations, the asymptotic distribution of T is bimodal.

3.2 Description of a recursive algorithm

If we look at our two dimensional Markov process (S_t, I_t), and only look at those time points where the process jumps, we can let $(\Delta S_i, \Delta I_i)$ denote the

ith jump made by (S_t, I_t). By the definition of (S_t, I_t), we get that $(\Delta S_i, \Delta I_i)$ either take the value $(-1, 1)$ or $(0, -1)$. If we introduce the Markov process $U_i = S_0 - S_i = n - S_i, U_0 = 0$, we can express our two dimensional process using only this 0/1 process since

$$\begin{aligned}(\Delta S_i, \Delta I_i) &= (-\Delta U_i, 2\Delta U_i - 1) \\ &= \begin{cases} (-1, 1) & \text{if } \Delta U_i = 1 \\ (0, -1) & \text{if } \Delta U_i = 0. \end{cases}\end{aligned}$$

So what's the point with this rewriting? And how do we connect this relation to the stopping time T?

Remeber that the stopping time T is the first time point when $I_i = 0$, which we can write as

$$\begin{aligned} I_i &= \sum_{j=0}^{i} I_j = I_0 + \sum_{j=1}^{i}(2\Delta U_j - 1) = m + 2U_i - i = 0 \\ &\Longrightarrow \\ U_T &= \frac{T - m}{2}. \end{aligned}$$

But, since U_i is defined to be the number of individuals infected by time step i, and T is the first time point when $I_i = 0$, we have that $U_T = Z$, which is the final size of the epidemic.

Now we see that we can compute the distribution of T, by calculating the time it takes until the process U_i hits the linear barrier $(i - m)/2$. Suppose that we know the distribution of T, then the final size distribution Z is easy to get since

$$p_z(z) = P(Z = z) = P\left(\frac{T - m}{2} = z\right) = P(T = 2z + m) = p_T(2z + m)$$

where $z = 0, 1, \ldots, n$.

To see how these relations can be used to calculate the exact distribution of T, we will now look at a recursive algorithm presented in Greenwood et. al [5].

It can be shown that the transition probabilities of U_i will be

$$\begin{aligned} P(U_i = k + 1 \mid U_{i-1} = k) &= \frac{\lambda(n - k)}{\lambda(n - k) + n} = p_k \\ P(U_i = k - 1 \mid U_{i-1} = k) &= 1 - p_k = q_k \end{aligned}$$

The next step is to calculate the defective distribution $W_i(k) = P(U_i = k, T > i)$, where $W_0(k) = \delta_{k,0}$, and it's computed as

$$W_i(k) = \begin{cases} W_{i-1}(k-1)p_{k-1} + W_{i-1}(k)q_k & \text{when } k > \frac{i-m}{2} \\ 0 & \text{when } k \leq \frac{i-m}{2} \end{cases}$$

If we let $P(T = 0)$ and suppose that we know W_{i-1} and $P(T = j), j < i$, then

$$P(T = i) = \begin{cases} W_{i-1}(\frac{i-m}{2})q_{(i-m)/2} & \text{if } i - m \text{ is even} \\ 0 & \text{otherwise} \end{cases}$$

In other words we compute $P(T = i)$ by first checking that we can reach the barrier in this time step, and if that's possible, we get the wanted probability by multiplicating the probability that we weren't absorbed in the previous time step and the probability that the process U_i stays on the level $(i - m)/2$ since the previous time step, i.e. U_i hits the barrier.

This linear barrier that we use will make it impossible for us to reach it in all discrete time steps, but as we have seen before, this will not be a problem when we want to compute $P(Z = z)$ for all $z = 1, \ldots, n$.

3.3 A comparison between exact and asymptotic results

To be able to compare our exact distribution of Z, with the asymptotic distribution (♠), we need to use the scaling

$$\begin{aligned} m &\approx bn^{1/3} \\ \lambda &\approx 1 + \frac{a}{n^{1/3}} \end{aligned}$$

proposed in Greenwood et. al [5] to make the epidemic near critical, and to plot $p_Z(z) = P(Z = z)$ on the new time scale $s = t/n^{2/3}$. Don't forget that the distribution of Z, computed with the algorithm above, is discrete and that we don't need to adjust our probability mass even though we use a different time scale.

However, to be able to compare these two distributions we need to make an integral approximation of the discrete distribution. This is done by

$$n^{2/3} \sum_{k=0}^{n} p_Z(s_k)(s_k - s_{k-1}) = 1.$$

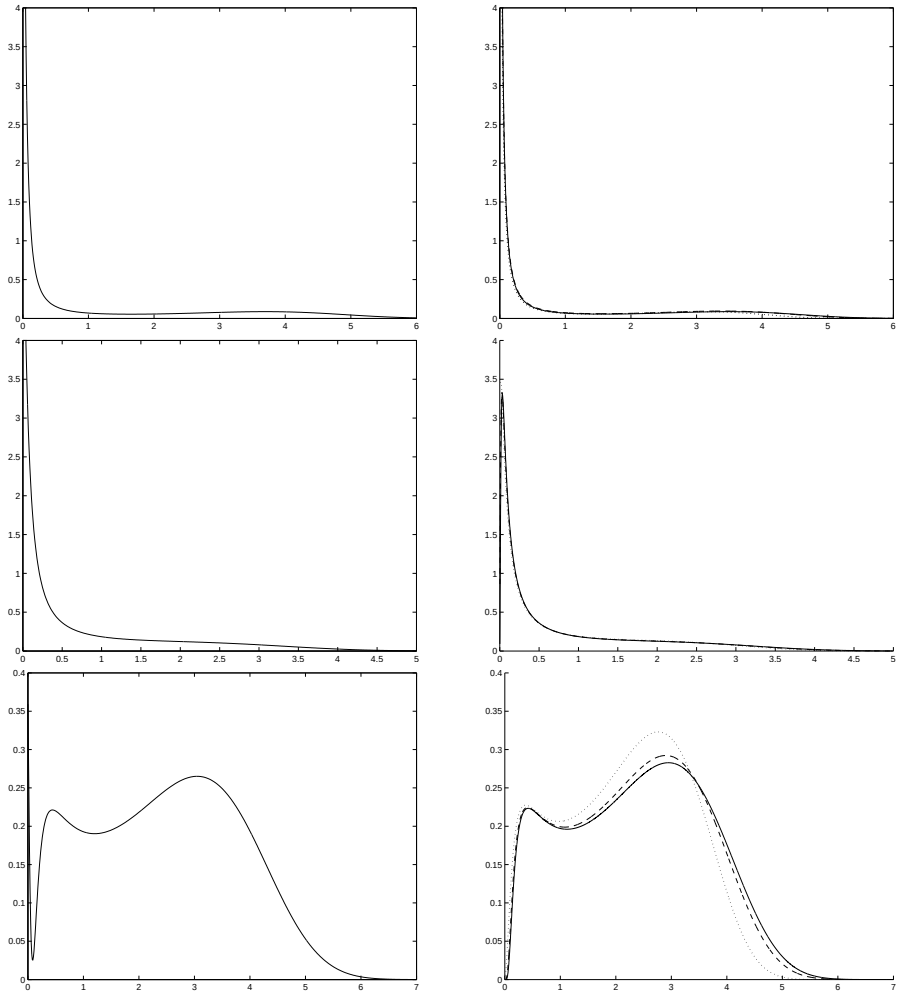

Figure 1: On the left side we have the asymptotic curves and on the right side we have the recursive ones. From the top down we have the following parameter values: $a = 1.5$ and $b = 0.25$ (top), $a = 0.5$ and $b = 0.5$ (middle) and $a = 1.0$ and $b = 1.5$ (bottom). For all cases we have $w = 0.5$ (corresponding to the Markovian assumption). Recursive calculations are carried out for populations (susceptibles) of size 1000 (dotted), 8000 (dashed) and 27000 (solid).

From fig. 1 we can see that the exact result seems to converge towards the asymptotic results relatively quickly, i.e. for moderately large populations (≈ 10000). We also see that there is a precision problem for small t in the analytic curve when $a = 1.0, b = 1.5$ and $w = 0.5$.

As we can see in fig. 1, some choices of the parameters a and b make the distribution for Z bimodal. But this is not as odd as one could think. In the introductury chapter of this thesis it was mentioned that one usually look at sub and super critical epidemics seperately, and that these two cases have very different outcomes. If the epidemic is sub critical the number of infectives decline exponentially fast and we have high probabilities for small final sizes and zero probabilities for large population sizes. For the super critical case we get that the probability mass is divided into two parts, one which has got an exponential behaviour for small final sizes and one part for large population sizes which is approximately Gaussian. With this in mind, our observed bimodal behaviour doesn't seem so strange, since what we have done, is that we in some sense have taken these two different outcomes and merged them togheter into one coherrent distribution under a new scaling. For more about the behaviour of sub and super critical epidemics, see Anderson and Britton [1].

4 SIS model

If we use the same assumptions as for the SIR model regarding infectious periods and so on, and don't allow an individual to become removed, then we have the SIS model. In the same way as we described the SIR epidemic with a two dimensional Markov process, (S_t, I_t), we can now use a one dimensional process instead, since $n + m = S_t + I_t$. For notational convenience, we will from now on let n denote the total number of individuals in our population, instead of the initial number of susceptibles. As mentioned before, we can no longer calculate the final size distribution Z, since we no longer can define what a final size is unambiguously. The aim of this part of the thesis will instead be to compare the asymptotic expression for the mean time to absorption with an exact expression which will be computed recursively using an algorithm.

But first, let's have look at our one dimensional Markov process I_t. This will in fact be a birth and death process in continuous time, with $I_0 = m$, an absorbing state in $I_t = 0$ and a reflecting state in $I_t = n$. Let λ_m be the rate with which the process may take a jump to state $m + 1$ and let μ_m be the rate with which the process may jump to $m - 1$, with $\lambda_0 = 0$ and $\lambda_n = 0$. In Martin-Löf [10] the transition rates are set to

$$\begin{cases} \lambda_i &= \lambda i \left[1 - \frac{i}{n}\right] = n\lambda \left(\frac{i}{n}\right) \left[1 - \frac{i}{n}\right] = n\lambda \left(\frac{i}{n}\right) \\ \mu_i &= i = n \left(\frac{i}{n}\right) = n\mu \left(\frac{i}{n}\right) \end{cases}$$

where

$$\begin{cases} \lambda(i) &= \lambda i(1-i) \\ \mu(i) &= i \end{cases}$$

is called Feller's 'Stochastic Verhulst Process'.

For the sake of completeness we should also mention that the SIS model also has got an expression for the basic reproduction number R_0, which is λ. So we see that for our chosen jump intensities the basic reproduction number is the same for both models.

4.1 Asymptotic results and diffusion approximations under a critical scaling

To get a diffusion approximation and a critical scaling, we must have a look at the expected increments moments which are

$$\begin{cases} E_i(\Delta I) &= n \left[\lambda \left(\frac{i}{n}\right) - \mu \left(\frac{i}{n}\right)\right] \Delta t \\ E_i(\Delta I^2) &= n \left[\lambda \left(\frac{i}{n}\right) + \mu \left(\frac{i}{n}\right)\right] \Delta t. \end{cases} \quad (\diamondsuit)$$

Next we rescale by letting $U_n(t) = \frac{1}{n} I_t$, and we get

$$\begin{cases} E_u(\Delta U) &= (\lambda(u) - \mu(u))\Delta t \\ E_u(\Delta U^2) &= \frac{1}{n}(\lambda(u) + \mu(u))\Delta t \end{cases}$$

which implies that $U_n(t) \to x(t)$ as $n \to \infty$, where $x(t)$ is the solution of

$$\frac{dx}{dt} = \lambda(x) - \mu(x) = \{ \text{ in our case } \} = (\lambda - 1)x - x^2.$$

As before, λ is the critical parameter, and the epidemic is near-critical when $\lambda \approx 1$.

To obtain a critical scaling we have to look at the expected increments moments $(\diamondsuit)$, which can be rewritten as

$$\begin{cases} E_i(\Delta I) &= i \left[(\lambda - 1) - \frac{\lambda i}{n}\right] \Delta t \\ E_i(\Delta I^2) &= i \left[(\lambda + 1) - \frac{\lambda i}{n}\right] \Delta t \end{cases}$$

If we let $\lambda \to 1$ as $n \to \infty$ we get

$$\begin{cases} (\lambda - 1) & = \frac{a}{\sqrt{n}} \\ \frac{I(\sqrt{n}t)}{\sqrt{n}} & = U_n(t). \end{cases}$$

One can now show that

$$\begin{cases} E_u(\Delta U) & = u(a-u)\Delta t \\ E_u(\Delta U^2) & = (2u)\Delta t \end{cases}$$

and as in the SIR case we get that $U_n(t)$ converges to a diffusion process $U(t)$ defined by the Itô equation

$$dU = U(a-U)dt + \sqrt{2U}dW$$

where $W(t)$ is a Wiener process. Unfortunately we can't calculate an analytic expression for the distribution of T, the time until absorbtion, nor the final size distribution, but we can obtain an expression for $T_u^* = E_u(T)$. In Martin-Löf [10] it is shown that T_u^* will be

$$T_u^* = \int_0^u \frac{F(v)}{f(v)} dv \quad (\clubsuit)$$

where

$$F(v) = \int_v^\infty \frac{f(s)}{s} ds \text{ and } f(s) = e^{-\frac{(s-a)^2}{2}}.$$

If we look at the integrand of ($\clubsuit$) we see that it is non negative for all $u > 0$, i.e. our function is monotone. A consequence of this fact is of course that we won't get a bimodal behaviour for T_u^* no matter which values we let the parameter a take.

4.2 Description of a recursive algorithm

In this section we will have a look at a recursive algorithm which make it possible to compute $T_u^* = E_u(T)$ exactly. Then it will be possible to compare the asymptotic result ($\clubsuit$) with the exact one, which we will do in the next section.

First of all, let us look at what we know about the SIS model.

In Martin-Löf [10] it is proposed that

$$\begin{cases} \lambda_k & = \lambda k \left(1 - \frac{k}{n}\right) \\ \mu_k & = k \end{cases}$$

Then we have the following transition probabilities:

$$p_k = \frac{\lambda_k}{\lambda_k + \mu_k} = \frac{\lambda(1-k/n)}{\lambda(1-k/n)+1} = 1 - q_k$$

where p_k is the probability for a jump from k to $k+1$, and q_k is the probability for a jump from k to $k-1$.

As mentioned in the previous chapter we can make our epidemic critical by using $\lambda = 1 + a/\sqrt{n}$. Note that this isn't the same scaling as for the SIR model.

If we let $T_k = E(T \mid I_0 = k)$ we have that

$$\begin{aligned} T_k &= E(\text{length of infectious period } k) + p_k T_{k+1} + q_k T_{k-1} \\ &= \frac{1}{\lambda_k + \mu_k} + \frac{\lambda_k}{\lambda_k + \mu_k} T_{k+1} + \frac{\mu_k}{\lambda_k + \mu_k} T_{k-1} \end{aligned}$$

since our model is Markovian. This can be rewritten as

$$\lambda_k(T_{k+1} - T_k) = \mu_k(T_k - T_{k-1}) - 1$$

and by introducing $D_k = T_{k+1} - T_k$ we get the difference equation

$$D_k = \frac{\mu_k}{\lambda_k} D_{k-1} - \frac{1}{\lambda_k}.$$

We also know that $I_t = 0$ is an absorbing state and that $I_t = n$ is a reflecting barrier. This gives us that $\lambda_0 = \mu_0 = 0$ and that $\lambda_n = 0$.

By solving the difference equation backwards, i.e. starting in D_n, we get that

$$\begin{aligned} 0 \cdot D_n &= \mu_n D_{n-1} - 1 \Rightarrow D_{n-1} = \frac{1}{\mu_n} \\ D_{n-1} &= \frac{\mu_{n-1}}{\lambda_{n-1}} D_{n-2} - \frac{1}{\lambda_{n-1}} \Rightarrow D_{n-2} = \frac{\lambda_{n-1}}{\mu_{n-1}} D_{n-1} + \frac{1}{\mu_{n-1}} \\ D_{n-2} &= \frac{\mu_{n-2}}{\lambda_{n-2}} D_{n-3} + \frac{1}{\lambda_{n-2}} \Rightarrow D_{n-3} = \frac{\lambda_{n-2}}{\mu_{n-2}} D_{n-2} + \frac{1}{\mu_{n-2}} \\ &\vdots \\ D_k &= \frac{\lambda_{k+1}}{\mu_{k+1}} D_{k+1} + \frac{1}{\mu_{k+1}}, \quad \text{for } k = 0, \ldots, n-2 \end{aligned}$$

and one can show by induction that

$$\begin{cases} D_{n-1} &= \frac{1}{\mu_n} \\ D_k &= \frac{1}{\mu_{k+1}} \left[1 + \sum_{i=1}^{n-(k+1)} \prod_{j=k+1}^{n-i} \left(\frac{\lambda_j}{\mu_{j+1}}\right)\right] \quad \text{for } k = 0, \ldots, n-2. \end{cases}$$

Since all D_k can be calculated, we can finally compute our T_k using the following forward equation

$$\begin{aligned} T_0 &= 0 \quad \text{(by definition)} \\ T_1 &= T_1 - T_0 = D_0 \\ T_2 &= (T_2 - T_1) + (T_1 - T_0) = D_1 + D_0 \\ T_3 &= (T_3 - T_2) + (T_2 - T_1) + (T_1 - T_0) = D_2 + D_1 + D_0 \\ &\vdots \\ T_n &= \sum_{k=0}^{n-1} D_k. \end{aligned}$$

For more about difference equations and birth and death processes, see Dynkin and Juschkewitsch [4] or Karlin [6].

4.3 A comparison between exact and asymptotic results

Now it's time to make a similar comparison between our exact and asymptotic results as we have done in the previous section for the SIR model. To do so we first need to look at our critical scaling

$$\begin{aligned} \lambda &\approx 1 + \frac{a}{\sqrt{n}} \\ s &= \frac{t}{\sqrt{n}}. \end{aligned}$$

Notice once again that this is *not* the same critical scaling as used for the SIR model.

We will now plot the asymptotic function for T_u^* and compare it with the results from the recursive algorithm for different population sizes and values of the parameter a. But as mentioned before the expression (♣) is monotone, so we will not get a similar interesting bimodal behaviour as for the distribution of Z for the SIR model.

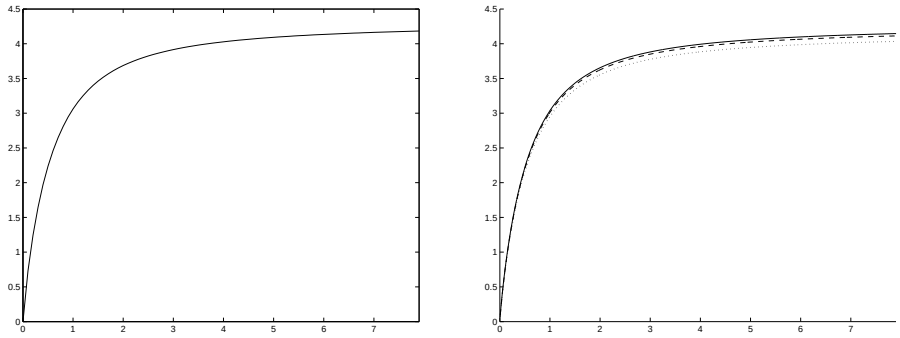

Figure 2: The left side is the asymptotic distribution and the right side is the recursive distribution. Both curves are for $a = 1.5$, and for the recursive curves the population sizes (susceptibles) are 2500 (dotted), 10000 (dashed) and 28900 (solid).

In fig. 2 we see, as for the SIR model, that the recursive algorithm converges quickly, that is for populations of susceptibles around 10 000 individuals.

One also notices that the x-axis is cut off at the value 8, which is due to numerical problems with the expression (♣), but this is not a problem since the most dramatic behaviour of the functions are in the beginning, and in this interval they have already started to level out.

5 A short summary and closing discussion

We have now had a look at two epidemic models, the SIR and the SIS model, and their asymptotic behaviour when they are made near critical. For the SIR model we have studied the behaviour for the final size distribution, and we have seen that under some parametrizations we get a bimodal distribution.

For the SIS model we have looked at the expected time until absorption given that the epidemic has k initially infected individuals, instead of the final size distribution (since the final size doesn't have an unambiguous expression for this model).

We have also compared these two asymptotic results with calculations made with two recursive algorithms, one for each model. Comparing the asymptotic results with the recursively calculated ones showed us that for both models we had what looked like a fast convergence towards the asymptotic result for populations of susceptibles consisting of about 10000 individuals.

If we again look at the concept of near critical epidemics, they are useful

in a vaccination situation, i.e. when we have super critical epidemic and we start a vaccination program, we will after some time reach a state of the epidemic when we can consider it as near critical.

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