

Discrete and continuous SIS epidemic models: a unifying approach

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Abstract

The Susceptible-Infective-Susceptible (SIS) epidemiological scheme is the simplest description of the dynamics of a disease that is contact-transmitted, and that does not lead to immunity. Two by now classical approaches to such a description are: (i) the use of a mass-action compartmental model that leads to a single ordinary differential equation (SIS-ODE); (ii) the use of a discrete-time Markov chain model (SIS-DTMC). While the former can be seen as a mean-field approximation of the latter under certain conditions, it is also known that their dynamics can be significantly different, if the basic reproduction number is greater than one. The goal of this work is to introduce a continuous model, based on a partial differential equation (SIS-PDE), that retains the finite populations effects present in the SIS-DTMC model, and that allows the use of analytical techniques for its study. In particular, it will reduce itself to the SIS-ODE model in many circumstances. This is accomplished by deriving a diffusion-drift approximation to the probability density of the SIS-DTMC model. Such a diffusion is degenerated at the origin, and must conserve probability. These two features then lead to an interesting consequence: the biologically correct solution is a measure solution. We then provide a convenient representation of such a measure solution that allows the use of classical techniques for its computation, and that also provides a tool for obtaining information about several dynamical features of the model. In particular, we show that the SIS-ODE gives the most likely state, conditional on non-absorption. As a further application of

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such representation, we show how to define the disease-outbreak probability in terms of the SIS-PDE model, and show that this definition can be used both for certain and uncertain initial presence of infected individuals. As a final application, we compute an approximation for the extinction time of the disease. In addition, we present many numerical examples that confirm the good approximation of the SIS-DTMC by the SIS-PDE.

Keywords: SIS Epidemiological Models, IBM Modelling, Differential Equations, Diffusive Limits

1. Introduction

Real populations are always finite. This means that stochastic effects will, sooner or later, play a significant role on its development. However, in very large populations the time scale for such stochastic events can be extremely long and, in these cases, the dynamics may be well approximated by a deterministic model. In the realm of epidemiological models, such course led to the development of compartmental mass action models pioneered by MacDonald and Ross (see Ross, 1911; Macdonald, 1957). This in turn sprung into a development of its own, benefited by the powerful analytical results in differential equations and dynamical systems (Anderson and May, 1995; Diekmann et al., 2013).

Nevertheless, there are many cases where stochastic effects cannot be neglected: moderately large populations or low population variability Ross (2011). These situations naturally led to the development of stochastic models for discrete populations with either continuous or discrete time (Allen, 2008; Keeling and Ross, 2008).

As it might be expected, these two paradigms do not, in general, give the same qualitatively dynamics—cf. discussion in §1.1. The very natural question then is how to reconcile such models, or at least to have some criteria for choosing the most appropriate paradigm in a given situation. In general, the answer for this question will be largely dependent on the underlying models.

In this work, we revisit the SIS (Susceptible-Infectious-Susceptible) model, which is one of the most elementary epidemiological dynamics. In this model, each individual in a population can be susceptible (i.e., can be infected) to a certain infectious disease or is in fact infected. Individuals will change from one group to the other: the SI transition (infection) occurs with probabil-

ity proportional to the number of infected and to the time of exposure; the IS transition (recovery) occurs with constant-in-time transition probability. This model is summarised in the following diagram:



The constants α and β in (1) can be interpreted as rates (either discrete or continuous) or as probabilities among many other possible choices. Here, we shall focus on two classical implementations of such dynamics: the first one, based on the mass-action principle and ordinary differential equations (SIS-ODE), and the second one based on discrete time Markov chains (SIS-DTMC). The former model includes the usual mass-action homogeneity and infinity population assumptions, whereas the SIS-DTMC requires only homogeneity — see the review in §1.1. Although the aim of this work is to understand the models just discussed on a unified framework, it is fair to observe that, in many situations, the heterogeneities of the population can be larger than the ones allowed by these classical models. In this case, alternatives approaches are necessary. See Diekmann et al. (2013) for a general discussion. In the deterministic framework, there are extensions to account for such heterogeneities as meta-population and multi-group models (e.g. Hyman and Li (1997); Feng et al. (2005); Guo et al. (2006)) or the consideration of self-awareness—cf. Van Segbroeck et al. (2010). In the stochastic framework, the inner variability of individuals can lead to non-Markovian dynamics as statistically observed in Yang (1972); Becker (1989) and this motivated the development of corresponding non-Markov models Mieghem (2013); Cator et al. (2013).

In connection with these two classical models, our main aim is to introduce a continuous model that will approximate the stochastic model, in the large population regime, but that will also approximate the deterministic model in many circumstances. In particular, it will honour its qualitatively dynamics of the stochastic model, but it will also reduces to the deterministic one when both dynamics are compatible. As a consequence, this continuous model provides a unifying view of both formulations, and it also brings forward the tools of differential equations to contribute for their analysis.

1.1. Classical discrete and continuous views of the SIS model

The SIS-ODE is one of the simplest epidemiological model based on the mass action principle—a particular popular interpretation of (1). It is dis-

cussed in many classical and more recent references (see, e.g, Rass and Radcliffe (2003); Bailey (1975); Dietz (1975); Anderson and May (1995); Diekmann et al. (2013)), and it is given by the following system of ODEs:

$$\begin{aligned}\dot{S} &= -\alpha SI + \beta I \\ \dot{I} &= \alpha SI - \beta I .\end{aligned}$$

Assuming, without loss of generality, that $S(0) + I(0) = 1$, we find

$$I' = \alpha I \left(1 - \frac{\beta}{\alpha} - I \right) . \quad (2)$$

The final value as in $t \rightarrow \infty$ of the solution for any non-trivial initial condition depends on the value $R_0 := \alpha/\beta$. For $R_0 \leq 1$, this limiting value is zero—the so-called disease free equilibrium; otherwise it is a positive constant $I_* = 1 - R_0^{-1}$ —the endemic equilibrium.

The second approach, the SIS-DTMC, consists of a population of N individuals, divided in two subgroups: Nx **I**nfected and $N(1 - x)$ **S**usceptibles, where $x \in \{0, \frac{1}{N}, \frac{2}{N}, \dots, 1\}$ is the fraction of infected. At each time step $\Delta t > 0$ one individual is chosen at random and then

- If it is of type **I**, then it becomes **S** with probability β ;
- If it is of type **S**, then it becomes **I** with probability proportional to the number of infected in the population: αx .

These rules specify a birth-death process, with the corresponding transition probabilities given by:

$$\begin{aligned}T^+(x) &= \alpha x(1 - x) , \\ T^0(x) &= 1 - T^+(x) - T^-(x) , \\ T^-(x) &= x\beta .\end{aligned}$$

Let $P_{(N,\Delta t)}(x, t)$ be the probability to find a fraction x of **I** individuals at time t in a population of size N , evolving in time steps of size Δt . The corresponding master equation is

$$\begin{aligned}P_{(N,\Delta t)}(x, t + \Delta t) &= T^+(x - z)P_{(N,\Delta t)}(x - z, t) + T^0(x)P_{(N,\Delta t)}(x, t) + \\ &T^-(x + z)P_{(N,\Delta t)}(x + z, t) ,\end{aligned} \quad (3)$$

where, for notation convenience, we set $z = N^{-1}$. The state $x = 0$ is an absorbing state, and for any choice of $\beta, \alpha > 0$, the chain is irreducible. Hence absorption is eventually certain, and thus $x = 0$ is the only stationary state (see, e.g, Allen (2008)).

In addition, notice that we always have

$$\sum_{x=0}^1 P_{(N,\Delta t)}(x, t) = \sum_{x=0}^1 P_{(N,\Delta t)}(x, 0) \quad (4)$$

The master equation (3) can be related with a discrete version of (2) as follows: let X_t be the fraction of infected individuals at time t . Let us define the expected fraction of infected individuals:

$$n(t) = \mathbb{E}[X_t] = \sum_{x=0}^1 x P_{(N,\Delta t)}(x, t) ,$$

where the summation shall be understood in the set $\{0, N^{-1}, 2N^{-1}, \dots, 1\}$.

Therefore

$$\begin{aligned}
n(t + \Delta t) &= \sum_{x=0}^1 x\alpha(x-z)(1-x+z)P_{(N,\Delta t)}(x-z,t) \\
&\quad + \sum_{x=0}^1 x(1-\alpha x(1-x)-\beta x)P_{(N,\Delta t)}(x,t) \\
&\quad + \sum_{x=0}^1 x\beta(x+z)P_{(N,\Delta t)}(x+z,t) \\
&= \sum_{x=0}^1 (x+z)\alpha x(1-x)P_{(N,\Delta t)}(x,t) \\
&\quad + \sum_{x=0}^1 x(1-\alpha x(1-x)-\beta x)P_{(N,\Delta t)}(x,t) \\
&\quad + \sum_{x=0}^1 (x-z)\beta xP_{(N,\Delta t)}(x,t) \\
&= \sum_{x=0}^1 xP_{(N,\Delta t)}(x,t) + z(\alpha-\beta)\sum_{x=0}^1 xP_{(N,\Delta t)}(x,t) - z\alpha\sum_{x=0}^1 x^2P(x,t) \\
&= \left[1 + \frac{\alpha}{N}\left(1 - \frac{1}{R_0^*}\right)\right] n(t) - \frac{\alpha}{N}\sum_{x=0}^1 x^2P_{(N,\Delta t)}(x,t) \\
&= n(t) + \frac{\alpha}{N}n(t)\left[\left(1 - \frac{1}{R_0^*}\right) - n(t)\right] - \frac{\alpha}{N}\mathbb{V}[X_t],
\end{aligned}$$

where \mathbb{V} denotes the variance and $R_0^* = \alpha/\beta$. Then, if we let $\alpha/N = \Delta t$, and neglect the variance term, we are left with an Euler discretisation of (2). Notice, however, that neglecting the variance term is not justifiable close to the disease extinction or to the endemic equilibrium. For a comprehensive monograph about the SIS DTMC, see Näsell (2011). See also Bailey (1963); Allen (1994); Allen and Burgin (2000); Allen (2008); McKane and Newman (2004) and references therein for different interpretations of stochastic modelling in epidemiology. See also Allen (1994) for discrete deterministic versions of epidemiological models, and a discussion where chaotic behaviour can appear and depart significantly from the corresponding continuous model.

The previous discussion highlights that, despite the fact that the modelling assumptions on the disease dynamics are similar in both cases, the

results obtained might differ considerably. In particular, as discussed before, for certain choices of parameters, there is a non-trivial stationary solution, which attracts all non-trivial initial conditions of the SIS-ODE model, whereas for the SIS-DTMC model, the only stationary state is the trivial one.

This apparent contradiction is solved by considering the behaviour of the transient states of the discrete process in the limit of large population. Indeed, the SIS-DTMC model is a Markov chain with leading eigenvalue $\lambda = 1$; the associated eigenvector denotes the trivial state, the only stationary state of the process and the absorbing state of any initial condition. The second eigenvalue $\lambda_* \in (0, 1)$ is associated to the transient state and the typical time such that the transient state fades out is directly related to the inverse size of the spectral gap $1 - \lambda_*$. However, when the population is large $1 - \lambda_* \ll 1$, making the transient state a quasi-stationary one. See Näsell (2011). Therefore, in the limit of infinite population (one of the basic assumptions of any modelling by ordinary differential equations) we possibly have a stationary state that is not present in the discrete model. In addition, for initial populations with a small fraction of infected individuals, the stochastic effects can lead to disease extinction in a much faster time scale.

1.2. Unified modelling

The solution of the reconciliation puzzle suggests that the ODE model can be understood as an approximation of the discrete model only for a certain range of time scales and initial conditions. In order to have a continuous model that approximate the discrete model at all time scales, and for all initial conditions, it turns out that we need to introduce partial differential equations (PDEs). In order to grasp both the deterministic effects (highlighted by the ODE model) and the stochastic effects (the eventual absorption in the discrete model), this equation has to be of drift-diffusion type. As the state where all individuals are of S-type (i.e., $x = 0$) is stationary for all populations, we cannot impose boundary conditions at $x = 0$, and the diffusion coefficient will be degenerated at the boundary. The correct solution (in the sense of being an approximation of the discrete process) will be obtained by imposing a boundary condition at $x = 1$, together with the conservation of probability. These constraints will lead to a measure solution for the corresponding PDE—for a similar situation in the context of population genetics, see Chalub and Souza (2009a,b, 2013). After introducing the SIS-PDE model, we derive a representation of the solution to this equation,

in order to obtain information of the solution of the discrete SIS model. Recalling that every population is finite, we have a partial differential equation model that gives information on the final and transient states of the discrete problem. Therefore, it generalises the ODE model to all time scales.

The first goal in this work is to derive the first order correction for the continuous model for finite size population effects. This is done in two steps: in the first step, we perform a formal asymptotic expansion in N of the transition matrix. This can be seen as reminiscent of the Kramers-Moyal expansion (Gardiner et al., 1985) or as a system size expansion (van Kampen, 2001), but we follow a more analytical route, along the lines of Chalub and Souza (2009a). Then, we obtain the following PDE:

$$\partial_t p = -\partial_x \{x [R_0(1-x) - 1] p\} + \frac{1}{2N} \partial_x^2 \{x(R_0(1-x) + 1)p\}, \quad (5)$$

supplemented with the boundary condition

$$\frac{1}{2N} ((1 - R_0)p(1, t) + \partial_x p(1, t)) + p(1, t) = 0,$$

and the conservation law

$$\frac{d}{dt} \int_0^1 p(x, t) dx = 0. \quad (6)$$

When N is equal to infinity, we formally obtain the equation:

$$\partial_t p = -\partial_x \{x [R_0(1-x) - 1] p\}, \quad (7)$$

supplemented by the boundary condition

$$p(1, t) = 0.$$

In this case, the conservation law (6) is always satisfied.

Equation (7) can be shown to be equivalent to (2) by observing that the latter is the equation for the projected characteristics of the former—see Chalub and Souza (2011) for a similar PDE derivation of the SIR model. The next step is to show that equation (5) can be consistently solved in the class of probability measures, and thus that its solution describes an approximation of the probability distribution of the discrete version, while maintaining its main features. In particular, large time limit of any solution will be a unity

Dirac mass at the origin, which means that the disease will be eventually be extinguished.

The second goal of this work is to probe the difference between the deterministic, the stochastic and the diffusion approximation obtained here through numerical simulations of the models. These simulations will confirm that the SIS-PDE is a consistent approximation of the SIS-DTMC and, consequently, to the SIS-ODE when it agrees with the SIS-DTMC. As a by-product, these simulations will also show that the ODE-SIS formulation models the interior mode (i.e., the mode of the probability distribution restricted to $x > 0$) of the probability distribution, and not the mean value. They will also shown the departure of the SIS-ODE model when the number of infected individuals is small, even if the entire population is large.

The third and final goal is to illustrate how the continuous formulation can contribute to the study of the dynamical features of the SIS-DTMC model. Using the information from the representation of the PDE solution, we give a slightly modified definition of outbreak probability, which agrees with the classical ones when considering the discrete counterpart, and show that the usual approximation—as given in (Allen, 2008)—can incur in serious errors for slightly supercritical values of R_0 . In particular, we shall consider the outbreak threshold number, \mathcal{T}_0 , recently introduced by Hartfield and Alizon (2013) and see how their results change in view of these differences. Finally, we provide an asymptotic derivation of the extinction time that complements earlier results by Nåsell (2011) and even earlier results by Doering et al. (2005).

1.3. Outline

The structure of this work is as follows: In section 2, we formally derive the SIS-PDE model. A comprehensive study of the PDE model is performed in section 3. Various numerical examples confirming the approximation quality of the SIS-DTMC by the SIS-PDE model are given in section 4. In section 5, we study the disease-outbreak probability for finite populations and in section 6 we compute an approximation for the extinction time of the discrete model. We conclude in section 7.

2. Formal Derivation of PDE model

2.1. Asymptotic expansion

We proceed with a second order Taylor expansion in x of $P_{(N,\Delta t)}$ and find

$$P_{(N,\Delta t)}(x \pm z, t) = P_{(N,\Delta t)}(x, t) \pm z \partial_x P_{(N,\Delta t)}(x, t) + \frac{z^2}{2} \partial_x^2 P_{(N,\Delta t)}(x, t) + \mathcal{O}(z^3). \quad (8)$$

Using $P = P_{(N,\Delta t)}(x, t)$, we derive the formal expansion for equation (3):

$$\begin{aligned} P_{(N,\Delta t)}(x, t + \Delta t) &= \alpha(x - z)(1 - x + z) \left(P - z \partial_x P + \frac{z^2}{2} \partial_x^2 P \right) \\ &\quad + (1 - \beta x - \alpha x(1 - x)) P + \beta(x + z) \left(P + z \partial_x P + \frac{z^2}{2} \partial_x^2 P \right) \\ &\quad + \mathcal{O}(\beta z^3, \alpha z^3) \\ &= P + z(-\alpha(1 - 2x)P - \alpha x(1 - x)\partial_x P + \beta P + \beta x \partial_x P) \\ &\quad + z^2 \left(-\alpha P + \alpha(1 - 2x)\partial_x P + \frac{\alpha x(1 - x)}{2} \partial_x^2 P + \beta \partial_x P + \frac{\beta x}{2} \partial_x^2 P \right) \\ &\quad + \mathcal{O}(\beta z^3, \alpha z^3) \\ &= P + z \partial_x (\beta x P - \alpha x(1 - x)P) + \frac{z^2}{2} \partial_x^2 (\beta x P + \alpha x(1 - x)P) \\ &\quad + \mathcal{O}(\beta z^3, \alpha z^3) \end{aligned}$$

We introduce the following assumptions:

1. $\beta_{(N,\Delta t)} = \beta_0 (\Delta t)^\gamma (1 + o(\Delta t))$,
2. $\alpha_{(N,\Delta t)} = \alpha_0 (\Delta t)^\gamma (1 + o(\Delta t))$.
3. $N = \kappa (\Delta t)^{\gamma-1}$.

with $0 < \gamma \leq 1/2$.

For a more detailed discussion about the role of scaling in obtaining the diffusive limit, see Chalub and Souza (2009a, 2013)

We also introduce the basic reproductive factor

$$R_0 (1 + o(\Delta t)) , \quad (9)$$

with $R_0 = \alpha_0/\beta_0$, and we rewrite the master equation as:

$$\begin{aligned} \frac{\Delta P}{\Delta t} &= \frac{\beta}{N \Delta t} \partial_x [x(1 - R_0(1 - x))P] + \frac{\beta}{2N^2 \Delta t} \partial_x^2 (x(R_0(1 - x) + 1)P) \\ &\quad + \mathcal{O}(\Delta t, N^{-2}) . \end{aligned}$$

On using the assumptions (and rescaling time $t \rightarrow \kappa t/\beta_0$), we find

$$\partial_t p = -\partial_x \{x [R_0(1-x) - 1] p\} + \frac{1}{2N} \partial_x^2 \{x(R_0(1-x) + 1)p\} + \mathcal{O}(\Delta t) , \quad (10)$$

Remark 2.1. *The fact the equation (10) can be obtained with various time-scale parameters can be used to increase computational efficiency in the simulations. For instance, consider the traditional diffusive scaling $\Delta t = (\Delta x)^2$, and an alternative scaling with $\gamma = 1/4$, hence $\Delta t = (\Delta x)^{4/3}$. The ratio of the latter to the former is $(\Delta x)^{-2/3}$, which for $N = 200$ is approximately 34. This means that a simulation that runs in one hour with the diffusive scaling will run in less than two minutes in the alternative scaling.*

The first approximation of the SIS model, which is size-independent, is given by the hyperbolic equation (7), and it is equivalent to the ODE (2); namely, the characteristics of equation (7) are solutions of (2).

If we consider the first correction due to finite-size effects, we find the parabolic equation (10), setting the $\mathcal{O}(\Delta t)$ term equal to zero.

2.2. Boundary condition and conservation law

Note that we can, in principle, extend equation (3) to values of x larger than 1. The compactness of $P_{(N,\Delta t)}$ is preserved; explicitly, if $P_{(N,\Delta t)}(x, 0) = 0$ for any $x \notin [0, 1]$ then $P_{(N,\Delta t)}(x, t) = 0$ for any $x \notin [0, 1]$, and $t > 0$ (this follows from the fact that $T^-(0) = T^+(1) = 0$). Therefore, in the continuous limit, it is natural to assume that the solution of the PDE is such that $p(x, t) = 0$ for any $x \notin [0, 1]$ and any $t > 0$. From the uniform parabolicity of the PDE in any neighbourhood of $x = 1$, we conclude the continuity of the flow of p around $x = 1$. We initially write the PDE (10) (with null $\mathcal{O}(\Delta t)$ term) in divergence form:

$$\partial_t p = \partial_x \left\{ \frac{\varepsilon}{2} [(R_0(1-x) + 1)p - xR_0p + x(R_0(1-x) + 1)\partial_x p] + x [R_0(1-x) - 1] p \right\} ,$$

where from now on, we will write $\varepsilon = \frac{1}{N}$.

Imposing the continuity of the flow at $x = 1$, we conclude that

$$\begin{aligned}
0 &= \lim_{y \rightarrow 0^+} \left[\frac{\varepsilon}{2} [(1 - R_0)p|_{1+y} + \partial_x p|_{1+y}] + p|_{1+y} \right] \\
&= \lim_{y \rightarrow 0^+} \left[\frac{\varepsilon}{2} [(1 - R_0)p|_{1-y} + \partial_x p|_{1-y}] + p|_{1-y} \right] \\
&= \left[\frac{\varepsilon}{2} [(1 - R_0)p|_1 + \partial_x p|_1] + p|_1 \right].
\end{aligned}$$

Finally, we observe that the conservation law (4) becomes

$$\int_0^1 p(x, t) \, dx = \int_0^1 p(x, 0) \, dx$$

which we will write as

$$\frac{d}{dt} \int_0^1 p(x, t) \, dx = 0. \tag{11}$$

3. Analytical results for the continuous model

We begin with the weak formulation introduced in Chalub and Souza (2009b) :

$$\begin{aligned}
&\int_0^\infty \int_0^1 p(x, t) \partial_t \phi(x, t) \, dx \, dt \\
&+ \frac{\varepsilon}{2} \int_0^\infty \int_0^1 p(x, t) x (R_0(1 - x) + 1) \partial_x^2 \phi(x, t) \, dx \, dt \\
&+ \int_0^\infty \int_0^1 p(x, t) x (R_0(1 - x) - 1) \partial_x \phi(x, t) \, dx \, dt \\
&+ \int_0^1 p(x, 0) \phi(x, 0) \, dx = 0,
\end{aligned} \tag{12}$$

where

$$\phi \in C_c^\infty([0, 1] \times [0, \infty)).$$

Since we are going to be interested in solution to (12) in a measure sense, we recall the following result proved in Chalub and Souza (2009b):

Lemma 3.1. *Let ν be a Radon measure supported in $[0, 1]$. Then we can write $\nu = \nu_0 + \nu_i + \nu_1$, where $\text{sing supp}(\nu_0) \subset \{0\}$, $\text{sing supp}(\nu_i) \in (0, 1)$, and $\text{sing supp}(\nu_1) \subset \{1\}$.*

In what follows, we shall be interested in positive and bounded Radon measures in $[0,1]$, and we shall denote these by $\mathcal{BM}^+([0,1])$. See Chalub and Souza (2009b) for more details about the choices of spaces.

In view of Lemma 3.1, we shall write for $p_0 \in \mathcal{BM}^+([0,1])$:

$$p_0 = a_0\delta_0 + r_0 + b_0\delta_1.$$

We now proceed to study (12) more thoroughly. We begin by observing that (12) is uniformly parabolic on $[\xi, 1]$, for any $0 < \xi < 1$. In particular we have the following

Lemma 3.2. *Let $p \in L^\infty([0, \infty); \mathcal{BM}^+([0, 1]))$ be a solution to (12). Then*

$$p \in C^\infty((0, \infty); C^\infty((0, 1))) .$$

Furthermore, $p(x, t) = a(t)\delta_0 + r(x, t)$, where r satisfies:

$$\begin{aligned} \partial_t r &= -\partial_x \{x [R_0(1-x) - 1] r\} + \frac{\varepsilon}{2} \partial_x^2 \{x(R_0(1-x) + 1)r\}, \\ \frac{\varepsilon}{2} ((1 - R_0)r(1, t) + \partial_x r(1, t)) + r(1, t) &= 0 \\ r(x, 0) &= r_0 + b_0\delta_1 \end{aligned} \tag{13}$$

Moreover,

$$a(t) = \frac{\varepsilon(R_0 + 1)}{2} \int_0^t r(0, s) \, ds + a_0. \tag{14}$$

Proof. Firstly, in any $(a, b) \subset [0, 1]$, (12) is uniformly parabolic, and the local regularity of p follows from standard arguments—see Lieberman (1996).

In view of Lemma 3.1, we write

$$p(t, x) = a(t)\delta_0 + r(x, t) + b(t)\delta_1. \tag{15}$$

Let $\phi(x, t) = \eta(t)\varphi(x)$, with $\eta \in C_c^\infty((0, \infty))$ and $\varphi \in C_c^\infty((0, 1))$. Then ϕ is an appropriate test function, and we have recast (12) in terms of r only. Then the regularity of p implies that $r \in C^\infty((0, \infty); C^\infty((0, 1)))$, and that it satisfies the equation (13) in classical sense. In what follows, we assume that (13) is well-posed, and defer its analysis to Proposition 3.4.

Now, let $\varphi \in C_c^\infty([0, 1])$, then on substituting (15) into (12), and using the regularity of r to integrate by parts, we obtain

$$\begin{aligned} & \int_0^\infty a(t)\eta'(t)\varphi(0) \, dx \, dt + \int_0^\infty b(t)\eta'(t)\varphi(1) \, dx \, dt + \\ & + \int_0^\infty b(t)\eta(t) \left(\frac{\varepsilon}{2}\varphi''(1) - \varphi'(1) \right) \, dx \, dt + \\ & + \frac{\varepsilon(1 + R_0)}{2} \int_0^\infty r(t, 0)\varphi(0) \, dt - \\ & - \int_0^\infty \left(r(1, t) + \frac{\varepsilon}{2}((1 - R_0)r(t, 1) + \partial_x r(1, t)) \right) \varphi(1)\eta(t) \, dt + \\ & + \frac{\varepsilon}{2} \int_0^\infty r(1, t)\varphi'(1)\eta(t) \, dt = 0. \end{aligned}$$

If we choose $\varphi \in C_c^\infty([0, 1])$, with $\varphi(0) = \varphi(1) = \varphi'(1) = 0$ and $\varphi''(1) \neq 0$, we find that

$$\frac{\varepsilon}{2} \int_0^\infty b(t)\eta(t)\varphi''(1) \, dt = 0.$$

Hence $b(t) = 0$ for almost every time.

Now, let us choose φ with $\varphi(0) \neq 0$, and $\varphi(1) = \varphi'(1) = 0$. We then conclude that

$$a(t) = \frac{\varepsilon(R_0 + 1)}{2} \int_0^t r(0, s) \, ds + a_0,$$

which is (14). Now let $\varphi(x) \equiv 1$. Then we are left with

$$\int_0^\infty \left(r(1, t) + \frac{\varepsilon}{2}((1 - R_0)r(t, 1) + \partial_x r(1, t)) \right) \eta(t) \, dt = 0,$$

and hence we obtain again the boundary condition in (13). \square

Remark 3.3. Notice that we are still left with

$$\frac{\varepsilon}{2} \int_0^\infty r(1, t)\varphi'(1)\eta(t) \, dt = 0.$$

This identity is satisfied if we choose φ with $\varphi'(1) = 0$. However, in general, we need to show that $\partial_x r(1, t)$ is uniformly bounded in ε . In this case, the boundary condition in (13) implies that $r(1, t) = \mathcal{O}(\varepsilon)$, and hence the above identity is $\mathcal{O}(\varepsilon^2)$, which can then be consistently neglected at this truncation order.

We now turn back to the classical solution. In the following, we write

$$F(x) = R_0(1 - x) + 1, \quad \Pi(x) = 1 - \frac{2}{F(x)}, \quad H(x) = x + \frac{2}{R_0} \log \left(\frac{F(x)}{F(0)} \right).$$

Proposition 3.4. *There exists a unique solution to (13) with $r(x, 0) = r_0 + b_0\delta_1$. Such a solution can be written as*

$$r(x, t) = \omega(x) \sum_{k=1}^{\infty} \alpha_k e^{\lambda_k t} \varphi_k(x), \quad (16)$$

where

$$\omega(x) = \frac{P(x)}{xF(x)}, \quad P(x) = \exp(2NH(x)),$$

φ satisfies

$$\omega^{-1}(x) \frac{1}{2N} \partial_x (P(x) \partial_x \varphi_k) = \lambda_k \varphi_k, \quad \varphi_k(0) = \partial_x \varphi_k(1) = 0, \quad (17)$$

with $\lambda_k < 0$.

Moreover,

$$\alpha_k = \int_0^1 r(x, 0) \varphi_k(x) \, dx \quad (18)$$

In particular, $r \in C^\infty((0, \infty); C^\infty([0, 1])) \cap C((0, \infty); \mathcal{BM}^+([0, 1]))$, and

$$\lim_{t \rightarrow \infty} \|r\|_\infty = 0.$$

Finally, if $r_0, b_0 \geq 0$, then $r \geq 0$.

Proof. Let

$$r(x, t) = \omega(x) u(x, t)$$

Then equation (13) becomes

$$\begin{aligned} \omega(x) \partial_t u &= \frac{1}{2N} \partial_x (P(x) \partial_x u) \\ u(0, t) &= \partial_x u(1, t) = 0, \\ u(x, 0) &= \omega^{-1}(x) (r_0(x) + b_0 \delta_1). \end{aligned} \quad (19)$$

with equation (17) being the spectral problem of the infinitesimal generator associated to (19). The operator on the left hand side of (17) is symmetric

in the space of C^2 functions f that satisfy $f(0) = f'(1) = 0$, with respect to the inner product

$$(f, g) = \int_0^1 f(x)g(x)\omega(x) dx.$$

Hence, the associated spectral problem is a Sturm-Liouville problem that is singular—of limit-circle type—at the origin. Thus the eigenfunctions yield a Hilbert basis for $L^2([0, 1])$. (cf. Zettl (2005)).

Therefore, the solution to (19) can be represented through the corresponding spectral expansion (see Evans (2010); Taylor (1996)), i.e.

$$u(x, t) = \sum_{k=1}^{\infty} \alpha_k e^{\lambda_k t} \varphi_k(x), \quad \alpha_k = (u(\cdot, 0), \varphi_k), \quad (\varphi_k, \varphi_k) = 1. \quad (20)$$

Thus, the expressions (16) and (18) follows immediately from the relation between r and u .

Finally, if $r_0, b_0 \geq 0$ then we have $u(x, 0) \geq 0$, and since (19) satisfies the strong maximum principle Lieberman (1996), we have that $u \geq 0$, and hence $r \geq 0$. \square

The important point about solutions in the sense of (12) is that they satisfy probability conservation as the next result shows

Theorem 3.5. *Let $p \in L^\infty([0, \infty); \mathcal{BM}^+([0, 1]))$ be a solution to (12). Then*

$$\int_0^1 p(x, t) dx = \int_0^1 p_0(x) dx,$$

for almost every time.

Proof. Consider $\phi(t, x) = \eta(t)$, with $\eta \in C_c([0, \infty))$, with $\eta(0) = 1$. Substituting in (12) yields

$$\int_0^\infty \int_0^1 p(x, t) \eta'(t) dx dt + \int_0^1 p(x, 0) dx = 0$$

and the result follows. \square

Theorem 3.6. *Let $p_0 \in \mathcal{BM}^+([0, 1])$. Then, equation (12) has a unique solution $p \in L^\infty([0, \infty); \mathcal{BM}^+([0, 1]))$. Moreover, we have*

$$p(x, t) = a(t)\delta_0 + r(x, t), \quad a(t) = \frac{\varepsilon(R_0 + 1)}{2} \int_0^t r(0, s) ds + a_0, \quad (21)$$

with $r \in C^\infty([0, 1] \times [0, \infty))$, and

$$\begin{aligned}\lim_{t \rightarrow \infty} \|r(\cdot, t)\|_\infty &= 0 \\ \lim_{t \rightarrow \infty} a(t) &= 1.\end{aligned}$$

Proof. Let r be the solution to (13). Then all the statements about r follow from Proposition 3.4. Let p be given by (21). Then $p(x, 0) = p_0$ and, upon substituting p in (12) and integrating by parts the terms with r , one verifies that p is indeed a solution. The statement about a follows from probability conservation.

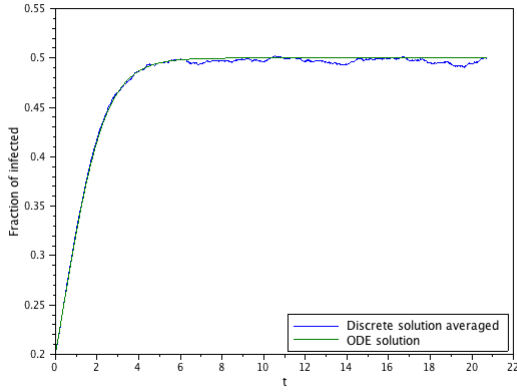
Now, let \tilde{p} be another solution to (12). By Lemma 3.2, we can write $\tilde{p}(x, t) = \tilde{a}(t)\delta_0 + \tilde{r}(x, t)$, and \tilde{r} satisfies (13). By virtue of Proposition 3.4, we have that $r = \tilde{r}$. On substituting \tilde{p} in (12), we find that $\tilde{a} = a$. Hence $p = \tilde{p}$. □

4. Numerical illustrations

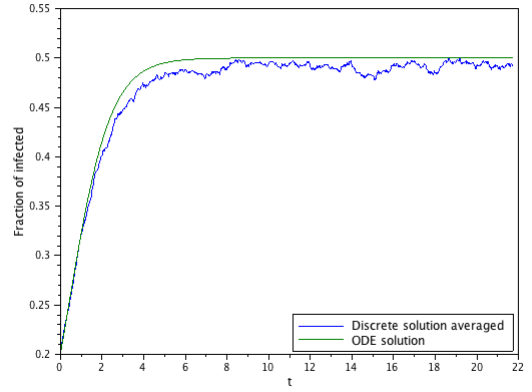
In this section, we provide a number of numerical examples of the three models considered so far. The choice of examples have two goals in mind: (i) to highlight the differences between the finite and infinite populations cases; (ii) to illustrate the performance of the approximation by the SIS-PDE, obtained in Section 2, and the use of the representation derived in Section 3.

4.1. The supercritical case with significant initial infection

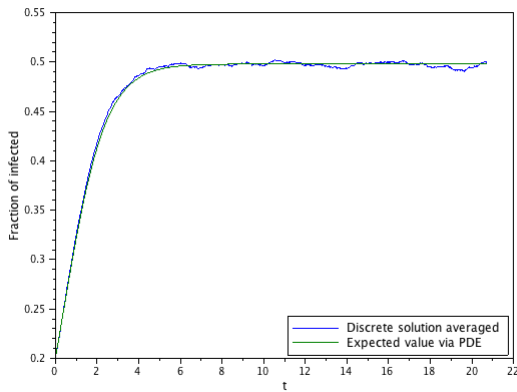
In this comparison, the initial population always have 20% of infected individuals, and $R_0 = 2$. We have included three sets: the first one compares the SIS-ODE and SIS-PDE with SIS-DTMC for two different sizes ($N = 500, 50$); the ODE solution vs the sample mode of $X(t)$ conditional on non absorption (for the same sizes as the previous set) ; the probability density given by the full PDE model (for $N = 500$). The agreement between the three models is quite good, even for a not too large population ($N = 50$). The mode for $X(t)$, albeit oscillatory, is well approximated by the ODE solution. We show also how the PDE solution contains various informations about the DTMC and ODE models.



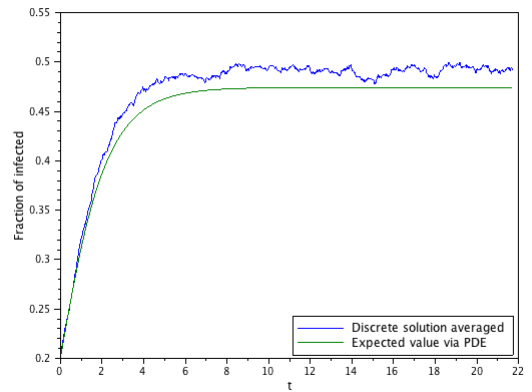
(a)



(b)

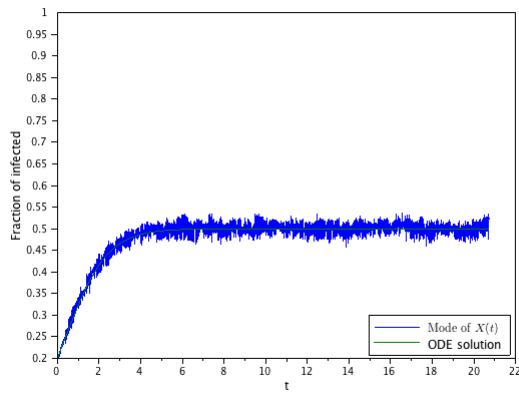


(c)

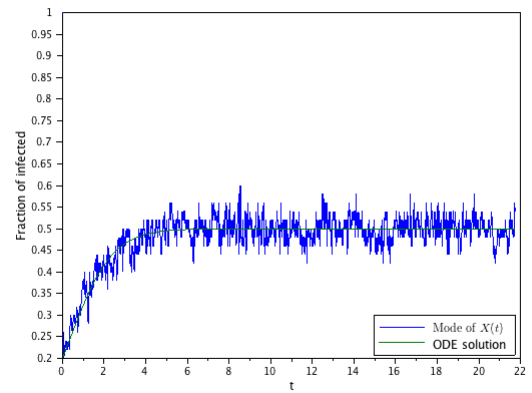


(d)

Figure 1: This set compares the simulations obtained using the Gillespie algorithm (see Gillespie, 1976) with both the ODE and the PDE approximations for two populations sizes: $N = 500, 50$ in the left and right columns, respectively. The initial populations has 20% of infected individuals. From the results, it is apparent that the ODE approximation works well, even for populations that are not extremely large. The PDE approximation also works effectively in both cases. When N is smaller the ODE approximation seems to overestimate the mean, while the PDE seems to underestimate it. Naturally, in this case, this variance is larger.

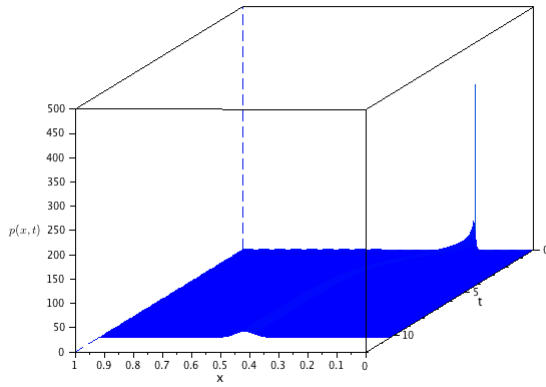


(a)

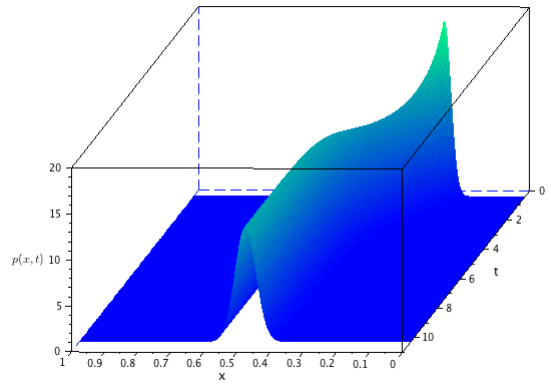


(b)

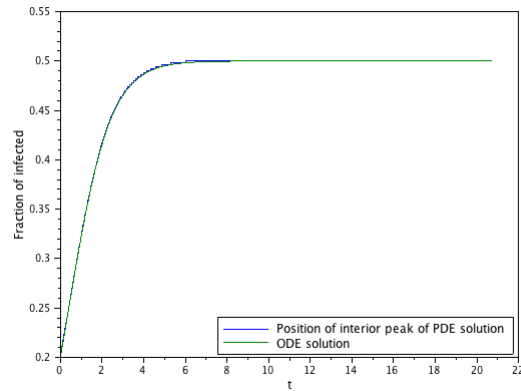
Figure 2: This set shows the ODE approximation versus the mode of the fraction of infected at each time—the mode of the sample of $X(t)$, conditional on not being absorbed. Although this mode is quite oscillatory, it is well approximated by the ODE in both population sizes—again the smaller population displays a larger variance. Leftmost is $N = 500$.



(a)



(b)

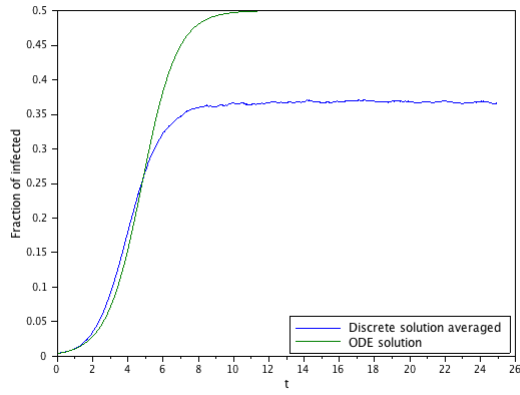


(c)

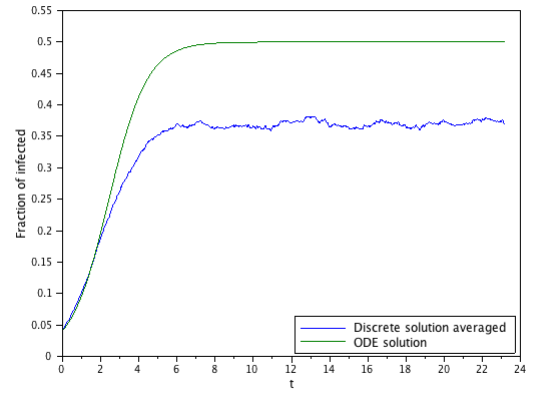
Figure 3: This set shows the probability density obtained from the solutions to the PDE. The initial Dirac at 0.2 diffuses quickly towards the quasi-stationary, with a negligible mass going to the Dirac mass at the origin. Thus one expects very little absorption for this initial condition, and hence a good agreement between the mean of infected and the ODE solution as observed in the previous set. In addition, notice that the the position of the maximum of the probability density does indeed follows the ODE dynamics.

4.2. The supercritical case with a small initial infection

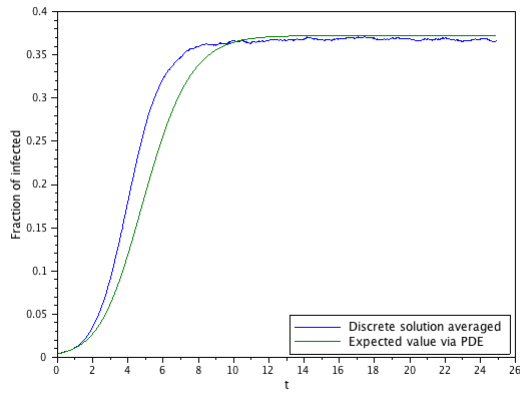
In the following sets, we repeat the experiment in the previous sets but with a very small initial fraction of infected. The results then change dramatically, as it would be expected. The SIS-ODE approximation to the SIS-DTMC breaks down. On the other hand, the SIS-PDE still works fine. Moreover, the sample mode conditional on non absorption of the discrete model continues to be approximated the ODE trajectories. The Dirac mass at the origin is now significant, and yields the extinction disease probability at time t . The peak of the probability distribution will approximately follow the ODE dynamics, after sometime.



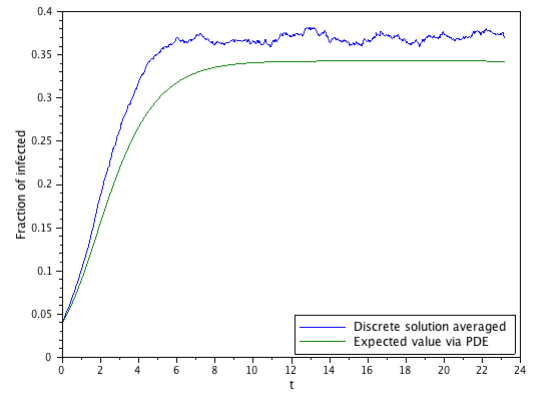
(a)



(b)

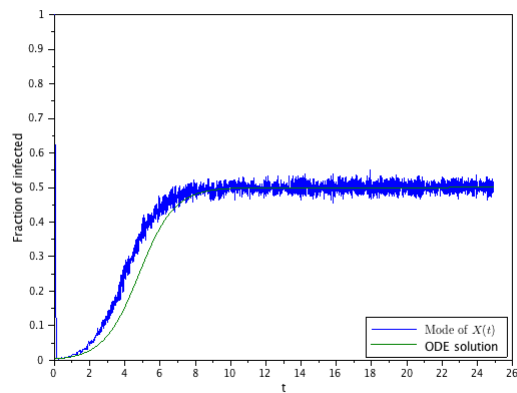


(c)

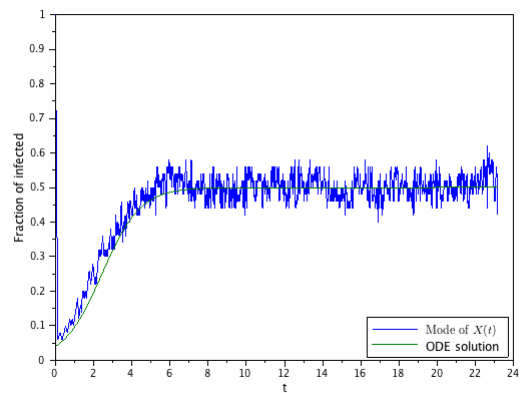


(d)

Figure 4: This set compares is corresponds to the one showed in Figure 1, but with only two individuals initially infected. In this case, the ODE approximations fails, while the PDE approximation continue to work well.

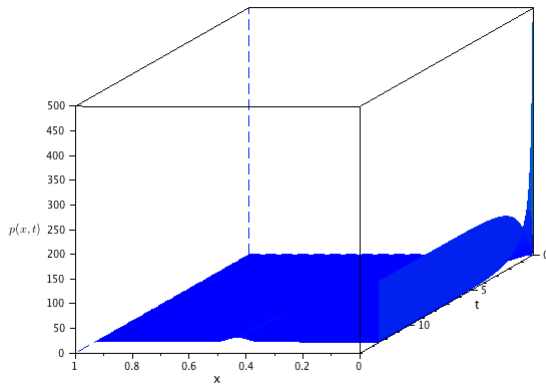


(a)

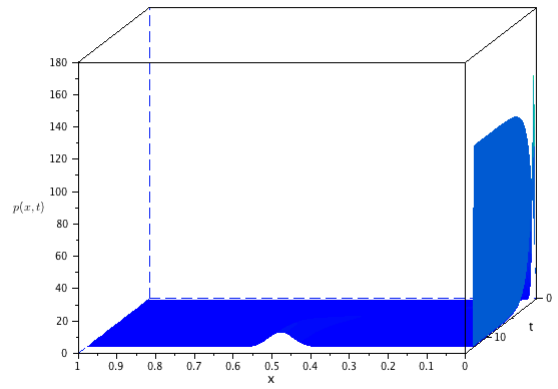


(b)

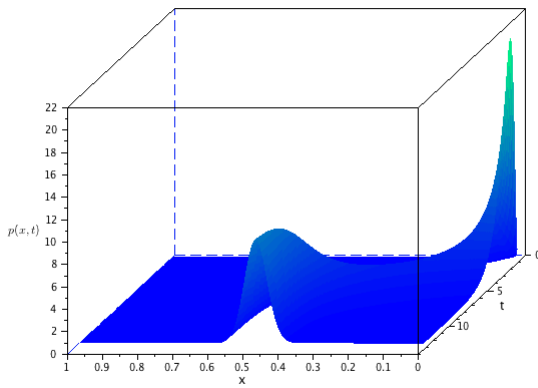
Figure 5: This set shows the ODE approximation versus the mode of the fraction of infected at each time—the mode of the sample of $X(t)$. Once again the mode is quite oscillatory, and it is well approximated by the ODE at both population sizes—with the smaller population displaying a larger variance. Notice that this approximation continues to work, even if the approximation of the mean by the ODE does not hold.



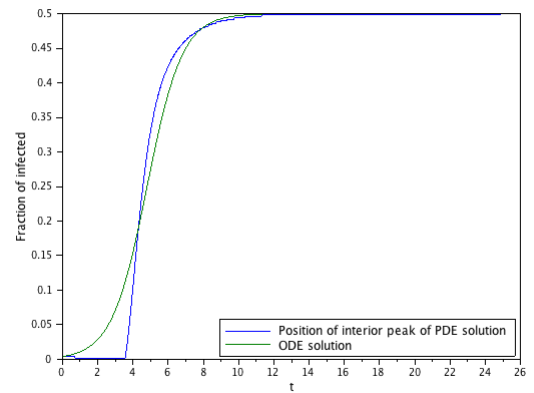
(a)



(b)



(c)



(d)

Figure 6: In this case, there is a significant increase of the Dirac mass at the origin. A closer look at the solution in the interior shows that, qualitatively, it still resembles the one obtained in the previous set. The interior maximum, after some time, is also well approximated by the ODE solution.

5. Disease outbreak in finite populations

In the following, we investigate the outbreak probability in finite, but large populations. In what follows, we assume $R_0 > 1$.

5.1. Rationale

For large N , and $R_0 > 1$, one expects to have $|\lambda_1|$ fairly small, and $|\lambda_2| = \mathcal{O}(1)$. In this case, we write the spectral representation (16) as

$$r(x, t) = a_1 \omega(x) \varphi_1(x) e^{\lambda_1 t} + e^{\lambda_2 t} g(x, t),$$

with

$$\|g(\cdot, t)\|_1 \leq \|g(\cdot, t)\|_2 \leq C, \quad C = \sum_{k=2}^{\infty} \alpha_k^2,$$

with α_k given by equation (20). Hence, if t is large enough, we conclude that $e^{\lambda_2 t} \|g(\cdot, t)\|_1$ is exponentially small.

Remark 5.1. *A simple estimation of how large must be this time can be obtained by choosing cut-off mass e^{-m} . In this case, if*

$$t > t_m = \frac{m + \log(C)}{|\lambda_2|},$$

then we have

$$e^{\lambda_2 t} \|g(\cdot, t)\|_1 < e^{-m}.$$

For such large times t , but that also satisfy

$$t \ll \frac{1}{|\lambda_1|},$$

we then have

$$r(x, t) \approx \alpha_1 \omega(x) \varphi_1(x)$$

which is an approximation of the quasi-stationary distribution of the SIS-DTMC. This means that, if the process has not been absorbed by this time, then it will most likely remain in the meta-stable state for a long time—as a matter of fact, for an exponentially long time. See Allen (2008); Nåsell (2011) and also the discussion in Section 6.

With this in mind, we give the following definition

Definition 5.2 (Disease outbreak probability). *Given an initial disease presence probability:*

$$p_0 = r_0 + b_0\delta_1,$$

we define the disease outbreak probability by

$$\mathcal{OP}[r_0, b_0, R_0] = a_1 \int_0^1 \varphi_1(x)\omega(x) \, dx.$$

Typically, we are interested in the case of an invasion”, i.e., the case where a fraction x_0 of infected individuals is initially present, and we want to assess if the disease might become endemic given such an initial presence. In this case, we take $b_0 = 0$ and $r_0 = \delta_{x_0}$ and write

$$\mathcal{OP} = \mathcal{OP}[x_0, R_0].$$

Notice that we also can include uncertainty in the initial presence by choosing r_0 to be a gaussian of mean x_0 and variance σ , for instance.

Remark 5.3. *The definition above is the immediate extension to the continuous case of the corresponding classical discrete counterpart—see Allen (2008) for instance. Notice also that this definition is implicitly dependent on N .*

5.2. Outbreak threshold

In a recent work, Hartfield and Alizon (2013) introduced the so-called *Outbreak Threshold*, \mathcal{T}_0 , roughly defined as the number of infected individuals that would likely lead the system to the deterministic dynamics. More precisely, their definition is equivalent to set

$$\mathcal{OP} = 1 - c,$$

and then to choose the particular level $c = e^{-1}$, or equivalently to set the outbreak level to approximately 63%. Their choice seems to be geared by the classical approximation for the outbreak in the SIS-DTMC model given (see Allen, 2008) by

$$\mathcal{OP} = 1 - \exp(-i_0 \log(R_0)). \tag{22}$$

Hence, choosing $c = e^{-1}$ yields

$$\mathcal{T}_0 = \frac{1}{\log(R_0)}. \tag{23}$$

Since (22) is an approximation, a first natural question is how good it performs. Figure 7 shows the relative error of (22) vis a vis the corresponding numerical solution.

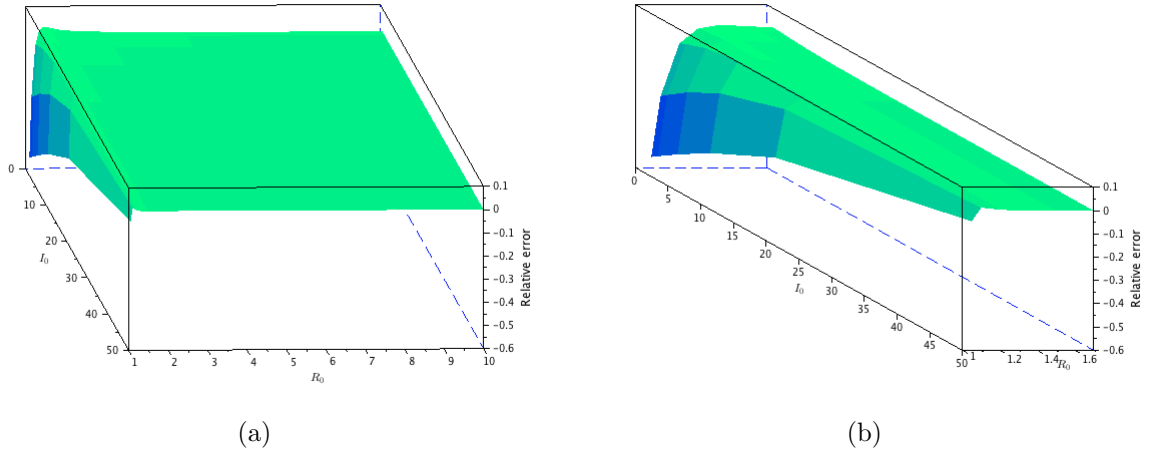


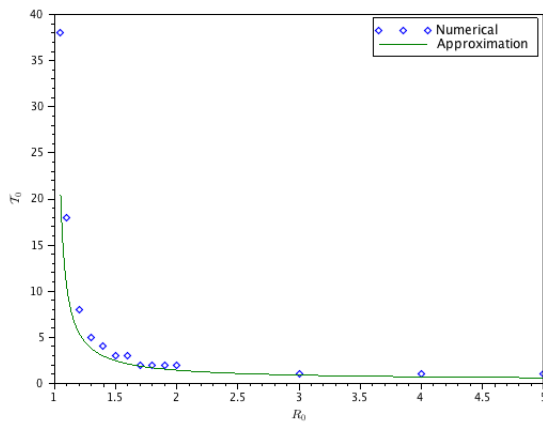
Figure 7: (a) Relative error of the outbreak probability, for an initial condition of I_0 individuals infected on a population of $N = 200$, for various R_0 computed using approximation (22) and compared with the numerical solution of (10). Negative values indicate that the approximation underestimated the corresponding probability. (b) Zoom for $R_0 \in (1, 1.6)$.

These results indicate that the approximation (22) might seriously underestimate \mathcal{OP} for slightly supercritical R_0 and moderately large populations — the error magnitude for slightly supercritical R_0 is similar for larger populations of $N = 500$ for instance.

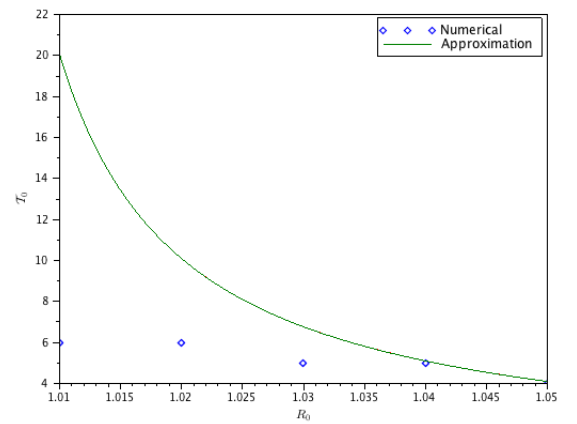
Such errors will correspondingly bias the calculations for \mathcal{T}_0 , and this can be seen in Figure 8.

6. Extinction time

In what follows, we study the mean extinction time of the disease. First we describe the governing equation, and obtain a representation for the solution in integral form. Such a representation can be used to produce numerical evaluations, and some examples are provided. These examples show



(a)



(b)

Figure 8: (a) For the level choice in Hartfield and Alizon (2013), we see (23) super estimates the threshold for R_0 that are not significantly above one. (b) The combination of R_0 even closer to critical (but still supercritical) together with a more stringent requirement that the outbreak probability is about 18% (choose $c = e^{-0.2}$) then shows that the corresponding threshold, Hartfield and Alizon (2013) given by $\mathcal{T}_0 = \frac{0.2}{\log(R_0)}$, seriously underestimates the threshold risk.

the boundary-layer nature of the solution when we have $\varepsilon \ll 1$, and thus we evaluate asymptotically this solution, for $R_0 > 1$.

6.1. Formulation, integral representation and numerical examples

Let $\tau_\varepsilon(x)$ denote the mean extinction time given that there are a fraction of x infected individuals at time zero.

Then, cf. Ewens (2004), we have that

$$\frac{\varepsilon}{2}\tau_\varepsilon'' + \Pi(x)\tau_\varepsilon' = \frac{-1}{\omega(x)}, \quad \tau_\varepsilon(0) = 0, \text{ and } \tau_\varepsilon'(1) = 0. \quad (24)$$

In (24), we have that

$$\omega(x) = x(R_0(1-x) + 1)$$

and

$$\Pi(x) = 1 - \frac{2}{R_0(1-x) + 1}.$$

If we take τ_ε' as the dependent variable, then (24) is a first order ODE for τ_ε' , satisfying $\tau_\varepsilon'(1) = 0$.

Its solution is readily seen to be

$$\tau_\varepsilon'(x) = \frac{2}{\varepsilon} \int_x^1 \frac{e^{\frac{2}{\varepsilon}(s-x)}}{\omega(s)} \left[\frac{R_0(1-s) + 1}{R_0(1-x) + 1} \right]^{\frac{4}{\varepsilon R_0}} ds.$$

Hence

$$\tau_\varepsilon(x) = \frac{2}{\varepsilon} \int_0^x \int_r^1 \frac{e^{\frac{2}{\varepsilon}(s-r)}}{\omega(s)} \left[\frac{R_0(1-s) + 1}{R_0(1-r) + 1} \right]^{\frac{4}{\varepsilon R_0}} ds dr. \quad (25)$$

Equation (25) can be rewritten as

$$\tau_\varepsilon(x) = \frac{2}{\varepsilon(R_0 + 1)} \int_0^x \int_0^{1-r} e^{\varepsilon^{-1}\phi(z,r)} \left[\frac{1}{z+r} + \frac{1}{\hat{x} - (z+r)} \right] dz dr, \quad (26)$$

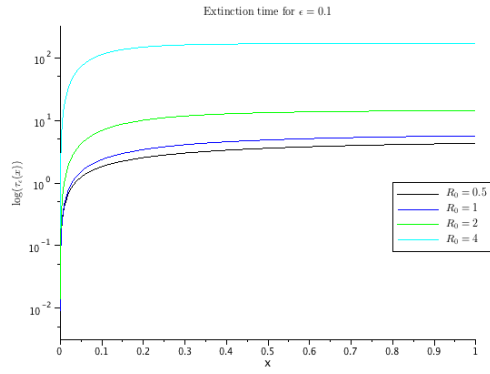
where $s = r + z$.

$$x^* = 1 - \frac{1}{R_0}, \quad \hat{x} = x^* + \frac{2}{R_0},$$

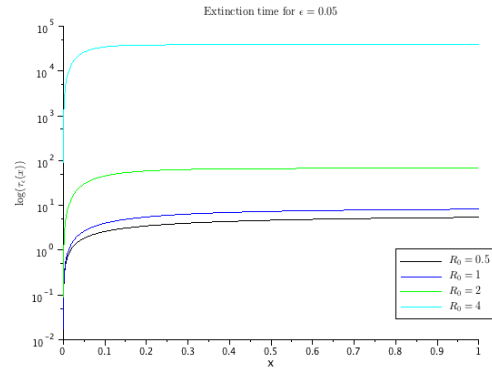
and

$$\phi(z, r) = 4 \left[\frac{z}{2} + \frac{1}{R_0} \log \left(1 - \frac{z}{\hat{x} - r} \right) \right].$$

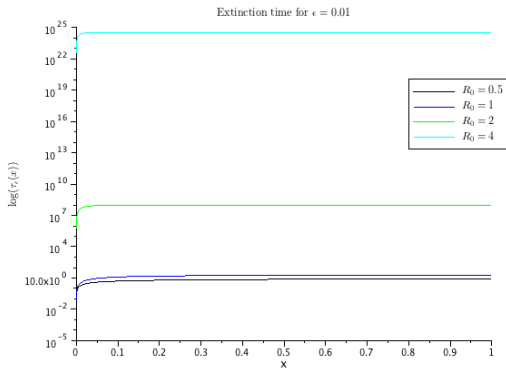
In Figure 9 we see a number of solutions of equation (25)—actually computed using the representation given by (26). See caption for further discussion.



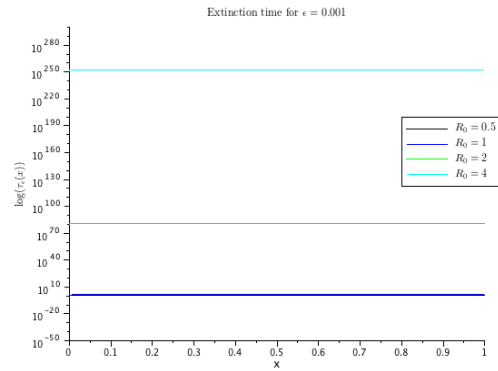
(a)



(b)



(c)



(d)

Figure 9: Log plot of the mean extinction times as a function of the initial frequency of infected. Graphs display such times for $R_0 = 1/2, 1, 2, 4$ and various values of ε . Notice that apart from a boundary layer close to $x = 0$ of order ε , these times are nearly constant.

6.2. Asymptotic evaluation

In order to evaluate (25) when $\varepsilon \ll 1$, we first need to evaluate $\tau'_\varepsilon(x)$. From (26), we have that:

$$\tau'_\varepsilon(x) = \frac{2}{\varepsilon(R_0 + 1)} \int_0^{1-x} e^{\varepsilon^{-1}\phi(z,x)} \left[\frac{1}{x+z} - \frac{1}{x+z-\hat{x}} \right] dz. \quad (27)$$

Before proceeding to evaluate (27) when $\varepsilon \ll 1$, we collect some useful facts about ϕ .

1. $\phi(0, r) = 0$ and, if $R_0 \leq 1$, we have $\phi(z, r) < 0$, for $z > 0$, and $r \geq 0$.
2. For $R_0 > 1$, we have $\partial_z \phi(0, r) > 0$, for $r < x^*$. In this case, $\phi(\cdot, r)$ has a positive maximum at

$$z^* = x^* - r.$$

This maximum will be relevant for the asymptotic evaluation provided that $r < x^*$.

We shall study the case $R_0 > 1$. The case $R_0 \leq 1$ is of less interest and will be discussed elsewhere.

Notice that $z^* > 0$, if $x < x^*$. Additionally, we compute

$$\partial_z^2 \phi(x^* - x, x) = -R_0.$$

Thus, provided that $0 \leq x \leq x^*$ —and hence that $1 - x \geq z^* \geq 0$ —and using the steepest descent method, we obtain

$$\begin{aligned} \tau'_\varepsilon(x) &= 2e^{\varepsilon^{-1}\phi(x^*-x,x)} \frac{f(x^*-x)}{x^*} \left(\frac{2\pi}{R_0} \right)^{1/2} \\ &\quad \times \left[N \left((1-x^*) \left(\frac{R_0}{\varepsilon} \right)^{1/2} \right) - N \left((x-x^*) \left(\frac{R_0}{\varepsilon} \right)^{1/2} \right) \right], \end{aligned}$$

where N is the cumulative Normal distribution.

For $x > x^*$, we have $\phi(z, x) < 0$ for $z \in [0, 1-x]$, and hence that the integrand is exponentially small, and can be neglected.

Integrating the representation for τ'_ε and evaluating using Laplace's method yields

$$\tau_\varepsilon(x) = 2e^{x^*\varepsilon^{-1}} \frac{f(x^*)}{|f'(x^*)|x^*} \left(\frac{2\pi}{R_0} \right)^{1/2} \left(1 - e^{-|f'(x^*)|x/\varepsilon} \right). \quad (28)$$

The asymptotic expression given by (28) confirms that, when $R_0 > 1$, we can have a very long persistence of the disease before it is eventually extinct. Figure 10 displays the behaviour of $\tau_\epsilon(1/N)$ for different values of R_0 , with $N = 50$, and confirms this first impression. Nevertheless, this result should be considered together with the results in Section 4; see the discussion in Section 7.

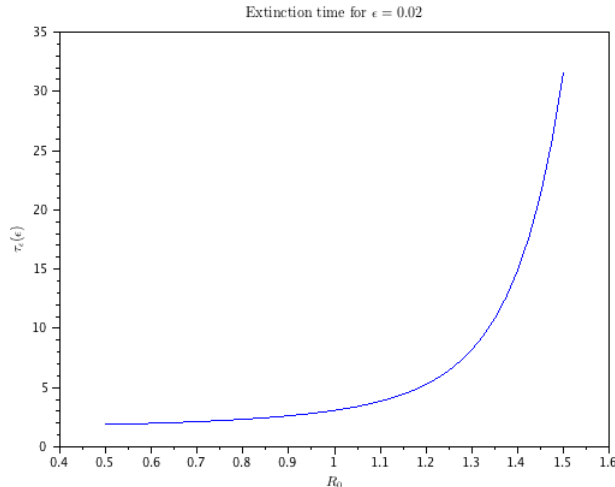


Figure 10: Mean extinction times for the disease when there is a single infected individual initially. Notice that even for such a moderate size population, the mean time of extinction increases exponentially with R_0 . Simulations were performed with $N = 50$.

7. Conclusion

We have revisited the SIS model in two different classical formulations: the mass-action ODE model (SIS-ODE) and the discrete time Markov chain model (SIS-DTMC). As already observed by many authors (cf. Allen, 2008; Nåsell, 2011) the discrete version can have quite a different behaviour from the continuous one in many situations. This naturally leads to the search of a more comprehensive and unifying model.

Our contest in this work is that the diffusion approximation derived in the spirit of the Kramer-Moyal expansion or system size expansion (cf. van

Kampen, 2001), but following a more analytical route as pursued by the authors in Chalub and Souza (2009a,b) (and also more recently in Chalub and Souza (2013)) leads to a continuous equation for the probability density, that we term SIS-PDE, together with a boundary condition at one end, and with a conservation law.

Interestingly, this equation does not have classical solutions, but it admits measure solutions—which are unique. Such measure solutions are then the correct diffusion approximation of the system. This situation provides an example where the correct biological solution is not the most regular one.

This measure solution can be represented conveniently as

$$p(x, t) = a(t)\delta_0 + r(x, t) \tag{29}$$

where r satisfies (13) and a is given by (14). The representation in (29) has a quite natural interpretation: r contains the probability of the transient states, and a the extinction probability. As is well-known, when $R_0 > 1$ the SIS-DTMC model has a metastable state. Within the diffusion framework, the probability that governs such transient states (the quasi-stationary probability) is associated to the principal eigenfunction of the classical version of the PDE.

The measure solution then contains, in a convenient representation, all the information about the model. This was also confirmed by several numerical examples, where we also can verify the good approximation properties of the diffusion model. In particular, we verify numerically that the ODE models the mode of the non-absorbed process if the initial infection is significant, or after some time is the initial infection is small.

The results thus obtained, were applied in two situations: firstly, we study the so-called outbreak probability. On using the SIS-PDE representation, we give a definition of outbreak probability for an initial probability distribution. For a certain initial presence, we have a Dirac initial condition, and this definition is then the continuous counterpart of the usual discrete definition—cf. Allen (2008). We then compared the numerical results with a standard approximation, also given in Allen (2008), and show that it the latter might deviate significantly for slightly supercritical R_0 . In addition, this formulation is then used to study the outbreak threshold (\mathcal{T}_0) recently introduced by Hartfield and Alizon (2013). Secondly, we study the mean extinction time (MET), and obtain approximations that can be seen as complementary both to the results of Näsell (2011) and Doering et al. (2005).

These results then suggest that diffusion approximation, together with their correct mathematical set up can be useful as modelling tool. In particular, they bring a powerful arsenal of analytical techniques that can contribute for the understanding of epidemiological problems, in the realm of finite population models.

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