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Research Report 2015-09

April, 2015

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STOCHASTIC SIS EPIDEMIC MODELS - THE PDE-APPROACH

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Abstract. SIS models comprise one of the simplest classes of epidemiological models. Stochastic versions, formulated by stochastic differential equations, have been recently discussed in literature, mainly with a focus on the existence and uniqueness of stationary distributions. With applicability in view, the present paper uses the Fokker-Planck equations related to SIS stochastic differential equations, not only in order to derive basic facts, but also to derive explicit expressions for stationary densities or other characteristics related to the asymptotic behavior. Two types of models are analyzed here: The first one is a stochastic version of the SIS model with saturated incidence, introduced in By Chen and Kang, 2014 and the second one is based on the Kramers-Moyal approximation of the simple SIS Markov chain model.

1. Introduction

In the following we discuss several diffusion-based versions of SIS epidemic models. Such models assume that individuals can be infected multiple times throughout their lives and that no immunity happens after infection. Typical examples for diseases that can be modeled in this way - at least in first approximation - are rota-viruses, some sexually transmitted infections and many bacterial infections, see e.g. Hethcote and Yorke [1984], Brauer et al. [2008].

We denote the number of infected individuals at time $t \in \mathbb{R}$ by $X(t)$ and the number of susceptible persons by $Y(t)$. In the simplest case, the overall population with size N is assumed to be constant. Note that because of

$$N = X(t) + Y(t) \tag{1.1}$$

it suffices to model the number of infected, replacing $Y(t)$ by $N - X(t)$ whenever necessary. Two parameters are relevant in the simplest case: the disease transmission coefficient (or strength of infection) $\beta > 0$, leading to a force of infection (or incidence rate) of $\beta X(t)$, and the recovery rate $\gamma > 0$. The force of infection models the rate at which susceptible individuals become infected, while the recovery rate is interpreted as the rate at which infected individuals become susceptible again, which means that $1/\gamma$ is the average duration of infection. As the population is constant, we will not make any difference between mass action and pseudo mass action ($\beta = \lambda/N$ for some per capita contact rate λ) in the following.

A large branch of research in mathematical epidemiology is based on deterministic differential equations, which directly interpret $X(t)$ as a smooth, deterministic function of time. In this context, the simplest SIS model can be formulated by the ordinary differential equation

$$\frac{dX(t)}{dt} = \beta X(t)\left(N - X(t)\right) - \gamma X(t), \tag{1.2}$$

$$X(0) = x_0 \in (0, N]. \tag{1.3}$$

Note that, because the size of population is constant, in view of (1.1) equation (1.2) is sufficient to describe the whole system $(X(t), Y(t))$. The properties of the simple deterministic SIS model are well known, as it is equivalent to the logistic equation used to describe population growth in ecology, see e.g. Murray [1989]. Because of its simplicity, equation (1.2) can be solved explicitly, which leads to

$$X(t) = \frac{N\beta - \gamma}{\beta - e^{-(N\beta-\gamma)\left(t - \frac{1}{x_0}\right)}} \tag{1.4}$$

if $\frac{N\beta}{\gamma} \neq 1$, and

$$X(t) = \frac{1}{\beta t + \frac{1}{x_0}} \tag{1.5}$$

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if $\frac{N\beta}{\gamma} = 1$. Clearly, we have $X(t) \in (0, N]$ if the process starts in this interval. The basic reproduction number, i.e. the expected number of secondarily infected individuals by a single initial case of infection, is given by the quotient $R_0 = \frac{N\beta}{\gamma}$. Based on the solutions (1.4) and (1.5) the long term behavior of the system is described by

$$\lim_{t\to\infty} X(t) = \begin{cases} N - \frac{\gamma}{\beta} & \text{if } R_0 > 1 \\ 0 & \text{else.} \end{cases} \tag{1.6}$$

Irrespectively of the initial value x_0 the system for $R_0 \leq 1$ exhibits a disease free stable equilibrium in the long run, while for $R_0 > 1$ the disease does not die out, which leads to a stable endemic equilibrium. Convergence here is monotonic with no oscillatory behavior.

While many different approaches for stochastic epidemiological models exist in literature, see e.g. Diekmann et al. [2013] or Chapter 6 of Keeling and Ross [2008], the present paper analyzes diffusion-type versions of the SIS model. This means that equation (1.2) is replaced by a stochastic (Ito) differential equation

$$\begin{aligned} dX(t) &= (\beta(X)X(t)\,(N - X(t)) - \gamma X(t))\,dt + \sigma(X(t))dW(t),. \\ X(0) &= x_0 \in (0, N]. \end{aligned} \tag{1.7}$$

Here, $W(t)$ represents Brownian motion and the resulting $X(t)$ is a stochastic process, which is adapted to the filtration generated by the (standard) Wiener process $W(t)$. The measurable functions $\sigma(\cdot) : \mathbb{R} \to \mathbb{R}$ (the diffusion term) and $\beta(X(t))\,(N - X(t))\,X(t) - \gamma X(t)$ have to fulfill some technical condition like a local Lipschitz condition together with a linear growth condition (see e.g. Fleming and Rishel [1975], p 118) or the Yamada-Watanabe condition (Yamada and Watanabe [1971]), in order to guarantee existence and uniqueness of a (strong) solution.

Many papers use stochastic differential equations mainly for numerical simulation. In this manner, not only SIS but also SIR and more complex models can be treated easily. Especially for highly complex models this is an important (sometimes even the only) option, see e.g. Sani and Pollett [2007] for a four dimensional epidemiological system. For the stochastic SIS model more can be achieved.

An important question is the existence of stationary solutions. Gray et al. [2011] analyze a SIS model, where β is modified by random fluctuations, resulting in a stochastic SIS model (1.7) with $\beta(X(t)) = \beta$ and $\sigma(X(t)) = \sigma \cdot (N - X(t))X(t)$. By purely probabilistic arguments they prove necessary conditions for exponential extinction and on the other hand for the existence of a stationary solution. Enhancing this model, Chen and Kang [2014] analyze a version of (1.7) with $\beta(X(t)) = \beta/(1 + hX(t)),\ h \geq 0$ and $\sigma(X(t)) = \sigma \cdot X(t)(N - X(t))/(1 + hX(t))$. The term $X(t)(N - X(t))/(1 + hX(t))$ aims at modeling reductions in incidence for larger levels of infected individuals, which can e.g. result from more cautious behavior. This effect is called "saturated incidence". Chen et al. give necessary conditions for exponential extinction and for persistence in the mean. Furthermore, they prove the existence of a unique stationary distribution under certain conditions. It also should be mentioned that Lin et al. [2014] prove existence and uniqueness of a stationary solution for a SIS model with vaccination, which is more complex than the simple SIS model because of the additional class of vaccinated individuals.

While those proofs are interesting on their own, the present paper aims at a different approach, namely using the related forward and backward Fokker-Planck (or Kolmogorov) equations and their properties for analyzing the properties of the process and its asymptotic behavior. Both of these PDEs characterize diffusions and can be related to stochastic differential equations like (1.7). In particular they describe the (transition) density function of $X(t)$ at any time t. The equations can also be used to derive and analyze stationary distributions. Moreover, it is possible to calculate further interesting characteristics like absorption times and quasi stationary distributions. Unfortunately, Gray et al. [2011] do not even mention the paper Roberts and Saha [1999], which derives the stationary distribution of a slight generalization of the Gray model directly from the Fokker-Planck equation and exhaustively analyzes the different cases of stationary density and extinction. Lin et al. [2014] mention the Fokker-Planck equation, but make no attempt to use it beyond the mere proof of existence of a stationary distribution.

In order to demonstrate the strengths of the approach we will paradigmatically analyze two cases: In section 2 we use the Fokker-Planck approach to analyze the Chen and Kang [2014] model. This model is an example of a fundamentally deterministic model, but the parameter β is assumed to be uncertain and is modeled as disturbed by random noise. The second case, analyzed in section 3, applies the PDE-approach to a genuine stochastic model: here the analyzed stochastic differential equation is understood as

a diffusion approximation of a continuous time (discrete state space) Markov chain with jump intensities β and γ. General approaches for such diffusion approximation can be found e.g. in Gardiner [2009], Chapter 11. See also applications to epidemiology in Fuchs [2013].

We will see that both models show quite different qualitative behavior, closely related to the basic models from which they are derived.

2. A stochastic SIS-model with Saturated Incidence

The stochastic model of Chen and Kang [2014] is defined by the stochastic differential equation

$$dX(t) = \left(\beta\frac{X(t)\,(N-X(t))}{1+hX(t)} - \gamma X(t)\right)dt + \sigma\frac{X(t)\,(N-X(t))}{1+hX(t)}dW(t) \tag{2.1}$$

We assume that the process starts within the interval $(0, N]$. If it has a known deterministic value x_0 at the beginning, the initial condition is given by

$$X(0) = x_0 \in (0, N]. \tag{2.2}$$

Alternatively, we may consider $X(0)$ as a random variable with density $p_0(x)$, if $X(0)$ cannot be observed. Stilln in this case (2.1) holds almost surely for any starting value and can be used to describe the process conditional on starting points x_0. However, the full distributions of $X(t)$ can only be constructed by adding the information about the starting distribution, $p_0(x)$.

Compared to the simple SIS model the denominator $1 + hX(t)$ models the reduction in infection risk caused by more cautious behavior and disease control, when the number of infected increases. The diffusion term $\sigma\frac{(N-X(t))X(t)}{1+hX(t)}dW(t)$ is added in order to model random perturbations of the parameter β. Clearly the stochastic model in Gray et al. [2011] is a special case with $h = 0$.

Denoting the density of any random variable $X(t)$ by $p(x,t)$, the related Kolmogorov forward, or Fokker-Planck equation is

$$\begin{aligned}\frac{\partial p(x,t)}{\partial t} &= -\frac{\partial}{\partial x}\left[\left(\beta\frac{x\,(N-x)}{1+hx} - \gamma x\right)p(x,t)\right] + \frac{1}{2}\sigma^2\frac{\partial^2}{\partial x^2}\left[\frac{x^2\,(N-x)^2}{(1+hx)^2}p(x,t)\right] \\ p(x,0) &= p_0(x).\end{aligned} \tag{2.3}$$

If $X(0) = 0$ is known with certainty, the initial condition reduces to $p_0(x) = \delta(x - x_0)$, where δ denotes the Dirac delta function. We use the notation

$$A(x) = \beta\frac{x\,(N-x)}{1+hx} - \gamma x$$

and

$$B(x) = \sigma^2\frac{x^2\,(N-x)^2}{(1+hx)^2}.$$

These functions are continuous (therefore also measurable) and locally bounded on the interval $[0, N]$.

The diffusion term equals zero at two points, $x = 0$ and $x = N$. Because we let the process start between these points, we have to consider the related boundary conditions. At $x = N$ the drift is negative, $A(N) = -\gamma N$, hence there is a reflecting boundary. For $x = 0$ we have $dB(x)/dx = 0$ and $A(x) = 0$, which indicates a natural boundary (see e.g. Gardiner [2009] p 119). The point $x = 0$ is absorbing, but it is never reached if the process starts in $(0, N]$. Altogether, by continuity of the sample paths, any process $X(t)$ starting in $(0, N]$ will stay in this interval forever. Consequently, as on $(0, N]$ both, drift and diffusion term, are Lipschitz continuous and bounded, there exists a unique solution on this interval (see e.g. Fleming and Rishel [1975], Theorem 4.1).

In order to solve the Fokker-Planck equation (2.3), one has to account for the nature of the boundary points. In particular, the boundary condition

$$A(N)p(N,t) - \frac{1}{2}\frac{\partial}{\partial x}\left[B(N)p(N,t)\right] = 0,$$

which accounts for the fact that there is a reflecting boundary at N, has to be added. At zero, no boundary condition is needed (see e.g. the synopsis in Appendix A of Cacio et al. [2012]).

Figure 2.1 shows two instances of the process $X(t)$ and the related density function $p(x,t)$ over time. While the case $N = 100$, $\beta = 0.013$, $\gamma = 1$, $\sigma = 0.06, h = 0.05$ shows fast extinction of the disease, reducing

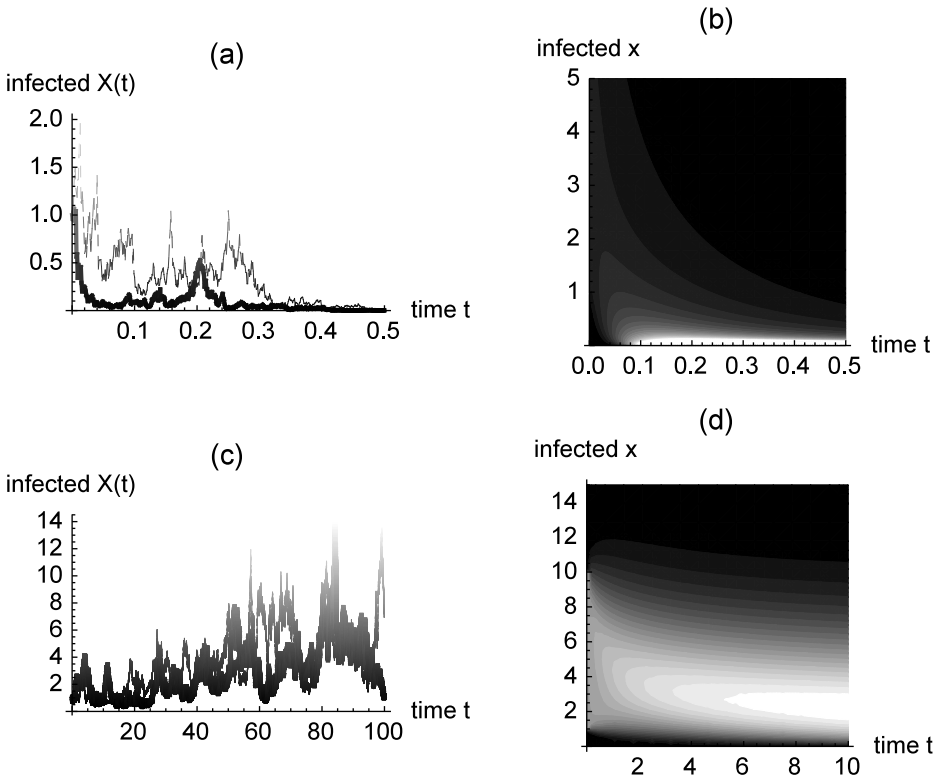

FIGURE 2.1. First line - Stochastic SIS model with saturated incidence with $N = 100$, $\beta = 0.013$, $\gamma = 1$, $\sigma = 0.06, h = 0.05$.
Second line - Stochastic SIS model with saturated incidence with $N = 100\,\beta = 0.013$, $\gamma = 1$, $\sigma = 0.005, h = 0.05$.
Parts (a), (c) respectively show two simulated trajectories of the respective processes, while (b),(d) are contour plots of the time dependent density function $p(x,t)$. The contour lines depict combinations of x and t with equal density $p(x,t)$. Regions with higher density are depicted in brighter nuances. The density at time zero is assumed as a uniform distribution on [1, 10].

the volatility to $\sigma = 0.005$ seemingly leads to endemic behavior of the system. We will see later that these conjectures are indeed true.

The asymptotic behavior of the deterministic model $\dot{X}(t) = A(X(t))dt$ depends on the basic reproduction number

$$R_0 = N\beta/\gamma \tag{2.4}$$

and can be characterized as follows: if $R \leq 1$, the disease free equilibrium $X(t) = 0$ is globally stable while if $R > 1$ the disease free equilibrium becomes unstable and the system has a unique (globally stable) endemic equilibrium

$$X(t) = \frac{N\beta - \gamma}{\beta + h\gamma}. \tag{2.5}$$

The stochastic diffusion process (2.1) with the related Fokker-Planck equation (2.3) fulfills the conditions of Theorem 2.1 in Zhang and Chen [2013] on $[0, N]$, therefore there exists a unique (limiting) stationary distribution. If it has a density $p_0(.)$, then $\lim_{t\to\infty} p(x,t) = p_s(x)$ almost everywhere. The stationary distribution can be analyzed with the help of the Fokker-Planck equation. If $p(x,t)$ is the density of the stationary distribution, we must have $\frac{dp(x,t)}{dt} = 0$, hence the stationary distribution solves the equation

$$\frac{\partial}{\partial x}\left[A(x)p_s(x)\right] = \frac{1}{2}\frac{\partial^2}{\partial x^2}\left[B(x)p_s(x)\right]. \tag{2.6}$$

This means that the probability flux

$$J(x,t) = A(x)p(x,t) - \frac{1}{2}\frac{\partial}{\partial x}\left[B(x)p(x,t)\right] \tag{2.7}$$

must be constant over x if $p(x,t) = p_s(x)$. Because the boundaries at N and zero are reflecting and natural, no probability mass is lost at any time (i.e. $J(x,t) = 0$), so the stationary density $p_s(\cdot)$ fulfills

$$A(x)p_s(x) - \frac{1}{2}\frac{\partial}{\partial x}\left[B(x)p_s(x)\right] = 0. \tag{2.8}$$

Classical solutions $p_s(x)$ of this equation have the form

$$p_s(x) = C \cdot (1+hx)^2 e^{-\frac{2\gamma(1+hN)^2}{N\sigma^2(N-x)}} (N-x)^{2\cdot\left(1-\frac{(hN+1)(N(\beta+h\gamma)-\gamma)}{N^2\sigma^2}\right)} x^{2\cdot\left(\frac{N\beta-\gamma}{N^2\sigma^2}-1\right)} \tag{2.9}$$

on the open interval $(0,N)$. In order to ensure that $p_s(\cdot)$ is a density, C has to be chosen such that

$$\int_0^N p_s(x)dx = 1. \tag{2.10}$$

If this is possible, it makes sense to extend the density to the whole set $\mathbb{R}$ by extending it as zero outside $(0,N)$.

Note that this extension is continuous at $x = N$, because

$$\lim_{x\to N} e^{-\frac{2\gamma(hN+1)^2}{N\sigma^2(N-x)}} (N-x)^{2\cdot\left(1-\frac{(hN+1)(N(\beta+h\gamma)-\gamma)}{N^2\sigma^2}\right)} = 0$$

To see this, use the notation $D = 2\cdot\left(\frac{(hN+1)(N(\beta+h\gamma)-\gamma)}{N^2\sigma^2} - 1\right)$ and $E = \frac{2\gamma(hN+1)^2}{N\sigma^2(N-x)}$ and note that E is positive. If therefore $D \le 0$, the limit equals zero. If $D > 0$, use L'Hospital's rule m times, where m is the first integer such that $D \le m$, which leads to the limit

$$\lim_{x\to N} \frac{(N-x)^{-D}}{e^{\frac{E}{(N-x)}}} = \lim_{x\to N} \frac{(N-x)^{-D+m}\prod_{k=0}^{m-1}(D-k)}{E^m e^{\frac{E}{N-x}}} = 0.$$

At $x = 0$ it depends on the model parameters, whether the density is continuous with value equal to zero, jumps to a finite value or even goes to infinity.

If $\frac{\beta N-\gamma}{N^2\sigma^2} - 1 \ge 0$, or equivalently

$$R_0 - \frac{N^2\sigma^2}{\gamma} \ge 1 \tag{2.11}$$

where R_0 is defined in (2.4), then

$$\lim_{x\to 0^+} p_s(x) = \begin{cases} 0 & \text{if } R_0 - \frac{N^2\sigma^2}{\gamma} > 1 \\ C\cdot e^{-\frac{2(1+hN)^2}{R-1}} N^{2\cdot\left(1-\frac{(hN+1)(N(\beta+h\gamma)-\gamma)}{N^2\sigma^2}\right)} & \text{if } R_0 - \frac{N^2\sigma^2}{\gamma} = 1, \end{cases} \tag{2.12}$$

In the first case the (extended) density is continuous at zero, in the second case it jumps to a finite value, but anyway it is still integrable.

On the other hand, if (2.11) is violated, the limit at $x = 0$ is $+\infty$. Still a normalizing constant exists under the weaker condition

$$R_0 - \frac{1}{2}\frac{N^2\sigma^2}{\gamma} > 1. \tag{2.13}$$

This can be seen from the fact that (2.13) is equivalent to $p = 2\left(1 - \frac{N\beta-\gamma}{N^2\sigma^2}\right) < 1$, which implies finiteness of the integral $\int_0^N \frac{1}{(N-x)^p}dx$ and - by the limit comparison test - finiteness of $\int_0^N p_s(x)dx$.

Figure 2.2 shows the three possible cases of density functions by example. The parameters N, β, γ, h are fixed at the same values as before. The first case, $\sigma = 0.005$, in fact is the second case of figure 2.1 and fulfills (2.11), so we actually have a stationary density for this example. The second case, $\sigma \approx 0.00548$, which still leads to integrability, fulfills (2.11) with equality, see also the second case in (2.12). The final case, $\sigma = 0.007$ violates (2.11) but still fulfills the critical integrability condition (2.13). Clearly the first case of figure 2.1 violates the critical condition, hence no normalizable density exists.

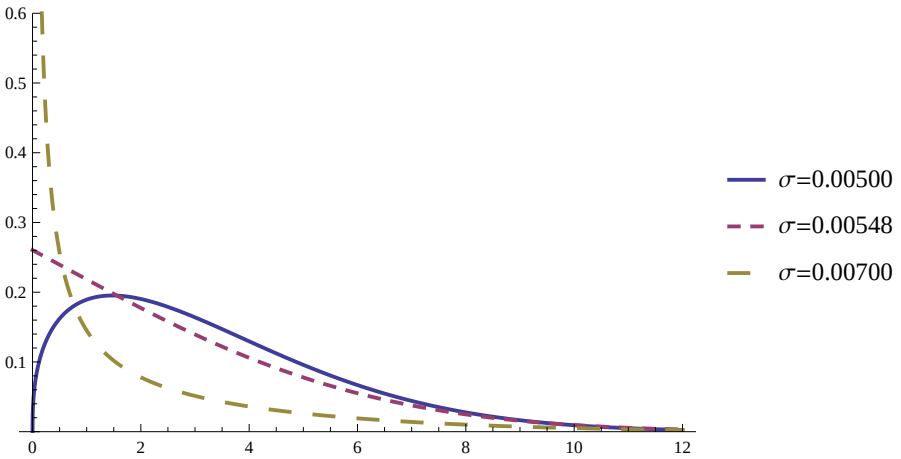

FIGURE 2.2. Three stationary densities with $N = 100\,\beta = 0.013$, $\gamma = 1$, $h = 0.05$, but different distortion parameter σ.

By analyzing the derivative of the stationary distribution it is possible to shed even more light on its behavior near zero. To this end, observe

$$\lim_{x\to 0^+} \frac{\partial p_s(x)}{\partial x} = \\ cx^{\frac{2\beta N-2\gamma}{N^2\sigma^2}-3}\left[2(hx+1)^3(N\beta-\gamma-x(\beta+\gamma h))-2\sigma^2(hx+1)(N-x)(N-x(hx+2))\right],$$

where c denotes some positive constant. By similar arguments as above one can derive that $\lim_{x\to 0^+}\frac{\partial p_s(x)}{\partial x} = -\infty$ if (2.13) holds but (2.11) is violated (the third case in figure (2.2)). Furthermore, depending on the values of the parameters h, N, β, γ and σ, we have $\lim_{x\to 0^+}\frac{\partial p_s(x)}{\partial x} \in \mathbb{R}$, if (2.11) holds with equality (the second case in figure (2.2)). Finally, there are three possible cases, if (2.11) holds without equality: If $R_0 - \frac{3}{2}\frac{N^2\sigma^2}{\gamma} > 1$, we have $\lim_{x\to 0^+}\frac{\partial p_s(x)}{\partial x} = 0$. The case $R_0 - \frac{3}{2}\frac{N^2\sigma^2}{\gamma} = 1$ leads to $\lim_{x\to 0^+}\frac{\partial p_s(x)}{\partial x} \in \mathbb{R}^+$, and finally $\lim_{x\to 0^+}\frac{\partial p_s(x)}{\partial x} = +\infty$ if $R_0 - \frac{3}{2}\frac{N^2\sigma^2}{\gamma} < 1$. Only the last case is depicted in figure (2.2) (with $\sigma = 0.005$).

If (2.13) is violated, no classical solution exists for the flux-equation (2.8), as (2.9) is not integrable between zero and N. In this case the stationary probability distribution is degenerate: the number of infected is zero with probability one, which is the disease free equilibrium. This extinction effect generalizes the exponential extinction derived in Theorem 2 of Chen and Kang [2014]. The generalized density of this probability distribution is given by the Dirac-delta function, i.e. $p_s(x) = \delta(x)$.

In fact, besides the classical solution (2.9) on $(0, N)$, equation (2.8) has $p_s(x) = \delta(x)$ as a weak (distributional) solution on $[0, N]$ because we have

$$\int_0^N A(x)\delta(x)\varphi(x)dx - \int_0^N B(x)\delta(x)\frac{\partial\varphi(x)}{\partial x}dx = A(0)\varphi(0) - B(0)\frac{\partial\varphi(0)}{\partial x} = 0 \tag{2.14}$$

for any test function φ with compact support on $(0, N)$. As long as the integrability condition (2.13) is fulfilled, the classical solution (2.9) exists and $\delta(x)$ cannot be the stationary solution: as a counterexample, it is not possible to approach $\delta(x)$, if (2.9) is used as starting distribution. Therefore (2.9) is the stationary probability distribution for $X(0) > 0$ in this case. If on the other hand (2.13) is violated, $\delta(x)$ remains as the only possible solution of (2.8).

Altogether, the criterion (2.13) makes out the difference between an endemic equilibrium, expressed by a stationary probability distribution with a density function, and extinction of the disease. Clearly there are parameter constellations that lead to an endemic equilibrium for the deterministic model, but result in extinction for the stochastic model, if σ is large enough. It should also be noted that the critical inequalities (2.13) and (2.11) do not depend on the saturation parameter h. Hence models with and without saturation show qualitatively the same asymptotic behavior.

Remark 1. It seems that in the light of (2.13) the basic reproduction number of the stochastic model should be $R = R_0 - \frac{1}{2}\frac{N^2\sigma^2}{\gamma}$. However, it can be shown that the basic reproduction number is the same for the deterministic and the stochastic model: consider an infected individual within a population of N susceptible individuals and let τ denote the random length of the infectious phase with $\mathbb{E}[\tau] = \frac{1}{\gamma}$. Then the expected number of secondary infections (with fixed population size N) is given by

$$R = \mathbb{E}\left[\int_0^\tau \beta N dt + \int_0^\tau \sigma N dW(t)\right] = N\,\mathbb{E}\left[\beta\tau + \sigma W(\tau)\right] = \frac{\beta N}{\gamma} = R_0.$$

Accounting for this fact, the term $\frac{1}{2}\frac{N^2\sigma^2}{\gamma}$ should rather be interpreted as a kind of risk adjustment for the basic reproduction number R_0.

3. A genuinely stochastic model

Consider now the stochastic differential equation

$$\begin{aligned} dZ(t) &= \left[\beta Z(t)\left(1 - Z(t)\right) - \gamma Z(t)\right] dt + \frac{\sqrt{\beta Z(t)\left(1 - Z(t)\right) + \gamma Z(t)}}{\sqrt{N}} \cdot dW(t) \\ Z(0) &= x_0 \in (0, N], \end{aligned} \tag{3.1}$$

with the related Fokker-Planck equation

$$\begin{aligned} \frac{\partial p(z,t)}{\partial t} &= -\frac{\partial}{\partial z}\left[\left(\beta z\left(1 - z\right) - \gamma z\right) p(z,t)\right] + \frac{1}{2N}\frac{\partial^2}{\partial z^2}\left[\left[\beta z\left(1 - z\right) + \gamma z\right] p(z,t)\right] \\ p(z,0) &= p_0(z). \end{aligned} \tag{3.2}$$

We write

$$A(z) = \beta z\left(1 - z\right) - \gamma z \tag{3.3}$$

and

$$B(z) = \frac{\beta z\left(1 - z\right) + \gamma z}{N} \tag{3.4}$$

in the following. Looking at the drift term of (3.1), this is definitely a SIS model. However, now the process $Z(t)$ denotes the proportion of infected individuals in a population of size N, i.e. $Z(t) = X(t)/N$. We have $B(z) = 0$ at $z = 0$ and at $z = 1 + \gamma/\beta$. If $p_0(\cdot)$ has its support in $[0, 1 + \gamma/\beta]$, then the process will stay in this interval at any time $t \geq 0$. Using the Yamada-Watanabe condition (Yamada and Watanabe [1971]) it is possible to show that because $A(\cdot)$ is Lipschitz continuous and $\sqrt{B(\cdot)}$ is Hölder continuous on the interval $[0, 1 + \gamma/\beta]$, solutions of (3.1) are pathwise unique (see e.g. Altay and Schmock [2013], Corollary 2.19). While the extension of the state space to the larger interval $[0, 1 + \gamma/\beta]$ is an unpleasant fact, it can be seen easily that for $x \geq 1$ the drift term $A(z)$ is negative and its magnitude increases with z, whereas the diffusion term $\sqrt{B(z)}$ decreases to zero, when z approaches $1 + \gamma/\beta$. Therefore, the process falls quickly back below one, if it ever rises above one.

The motivation for equations (3.1), (3.2) and (3.5) ccomes from a continuous time Markov chain model, where the number of infected individuals $X(t)$ takes values in $\{0, 1, \ldots, N\}$, the process $X(t)$ has right continuous sample paths and a probability measure P is related such that

$$\begin{aligned} P\left[X_{t+h} = x_t + 1 \,|\, X_t = x_t\right] &= N\beta x_t\left(N - x_t\right) h + o(h), \\ P\left[X_{t+h} = x_t - 1 \,|\, X_t = x_t\right] &= N\gamma x_t h + o(h) \\ P\left[X_{t+h} = x_t \,|\, X_t = x_t\right] &= 1 - N\beta x_t\left(N - x_t\right) h - N\gamma x_t h + o(h) \end{aligned} \tag{3.5}$$

and $P\left[X_{t+h} = x_t + k \,|\, X_t = x_t\right] = o(h)$ in all other cases $k \notin \{0, 1, -1\}$. Keep in mind that here parameters β and γ have been rescaled such that $N\beta$ is the transmission coefficient and $N\gamma$ is the recovery rate.

Based on (3.5) the Fokker-Planck equation (3.2) then can be derived as the Kramers-Moyal expansion of the Master equation related to the Markov chain (3.5). See e.g. section 11.2 of Gardiner [2009] for the general approach, or section 4.3 in Fuchs [2013], which also includes further examples from epidemiology. The same result can also be achieved by using the Langevin Approach (see 4.3.3 in Fuchs [2013]). It should be noted that the analyzed model is expressed using a single stochastic risk factor, namely one instance of the Brownian motion $W(t)$. For a proposal of a stochastic SIS model with two risk factors see e.g. Allen [2007], pp. 147.

Note that the Markov chain has a finite state space and all states are transient with the exception of zero, which is absorbing and reachable from the other states. As a result, the stationary distribution of the Markov chain is trivial, because irrespectively of the starting point the process finally is absorbed at zero. It is possible to be absorbed at zero in finite time, which is a main distinction from the stochastic model with saturated incidence (2.1) and also the simple deterministic model (1.2). As we will see, the approximating stochastic model (3.1), (3.2) keeps this basic property of the Markov chain (3.5), which is not true for some other approximation methods like e.g. the Van Kampen system size expansion.

There exists no stationary density on the interval $(0, 1+\gamma/\beta)$. In fact, using the new definitions (3.3), (3.4) of $A(\cdot)$ and $B(\cdot)$, the classical solution of the flux equation (2.8) is given by

$$p_s(z) = C \cdot \frac{Ne^{2Nz}(\beta+\gamma-\beta z)^{\frac{4N\gamma}{\beta}-1}}{z}, \tag{3.6}$$

with positive proportionality factor C. However, (3.6) is not integrable on $(0, 1+\gamma/\beta)$ because of the factor $1/z$. On the other hand the flux equation (2.8) still has the weak solution $p_s(z) = \delta(z)$, because (2.14) also holds for $A(\cdot)$, $B(\cdot)$ as defined in (3.3) and (3.4). Altogether, this indicates that the process converges almost surely to the disease free state in the long run.

The deeper reason can be seen, if one analyzes the critical points $z = 1+\gamma/\beta$ and $z = 0$ in more detail: The upper boundary $z = 1+\gamma/\beta$ is reflecting, because $A(1+\gamma/\beta) < 0$. On the other hand the lower boundary $x = 0$ is absorbing, because the flux (2.7) at zero is

$$J(0,t) = -\frac{(\beta+\gamma)p(0,t)}{2N},$$

which is negative for $p(0,t) > 0$ and indicates that some probability mass is leaving the region $[0, N]$ at any point in time. The intensity of the flux out of the region is proportional to the sum of the parameters β and γ and indirect proportional to the population size N.

Accounting for this facts, in order to use the Fokker-Planck equation (3.2) one has to combine it with the boundary conditions $p(0,t) = 0$ and $J(1+\gamma/\beta) = 0$. Note that $G(t) = \int_0^{1+\gamma/\beta} p(z,t)\,dz < 1$ for $t > 0$ then represents the probability of being not absorbed at the disease free state $x = 0$ up to time t. Summarizing, the probability distribution of $Z(t)$ can be described by the generalized density

$$P(z,t) = p(z,t) + (1 - G(t))\delta(z). \tag{3.7}$$

Figure 3.1 shows three examples for the development of $p(x,t)$ over time. In the first case, (a) and (b), extinction of the disease happens very slowly, whereas the cases (c) and (d) show quick extinction.

The qualitative differences in the long-term behavior can be analyzed further based on the eigenfunctions and eigenvalues related to the partial differential equation (3.2). In particular one question in this field is the existence of the so called quasi-stationary distribution, i.e. the long-term limit of the conditional distribution, given that extinction has not happened so far (see e.g. Collet et al. [2012]).

Instead of such approaches, we analyze the expected time until absorption (mean exit time, mean first passage times) when starting at x. If the random variable τ denotes the first time at which the process hits zero, i.e. $\tau = \inf\{t : Z(t) = 0\}$ then the expected absorption time is given by $T(z) = \mathbb{E}[\tau | X(0) = z]$ and the expectation $T(z)$ fulfills the differential equation (see e.g. Gardiner [2009], section 5.5.2)

$$A(z)\frac{\partial T(z)}{\partial z} + \frac{1}{2}B(z)\frac{\partial^2 T(z)}{\partial z^2} + 1 = 0$$

with boundary conditions $\frac{\partial T}{\partial z}(1+\gamma/\beta) = 0$ and $T(0) = 0$. With

$$\psi(z) = \exp\left[2\int_0^z A(y)/B(y)\,dy\right] = e^{2Nz}\left(\frac{\beta\cdot(1-z)+\gamma}{\beta+\gamma}\right)^{\frac{4\gamma N}{\beta}}$$

the solution can be written as

$$T(z) = 2\int_0^z \frac{1}{\psi(y)}\int_y^b \frac{\psi(x)}{B(x)}dx\,dy. \tag{3.8}$$

Figure 3.2 shows the expected time to absorption at zero as a function of the starting value for two of the models, also depicted in figure 3.1.

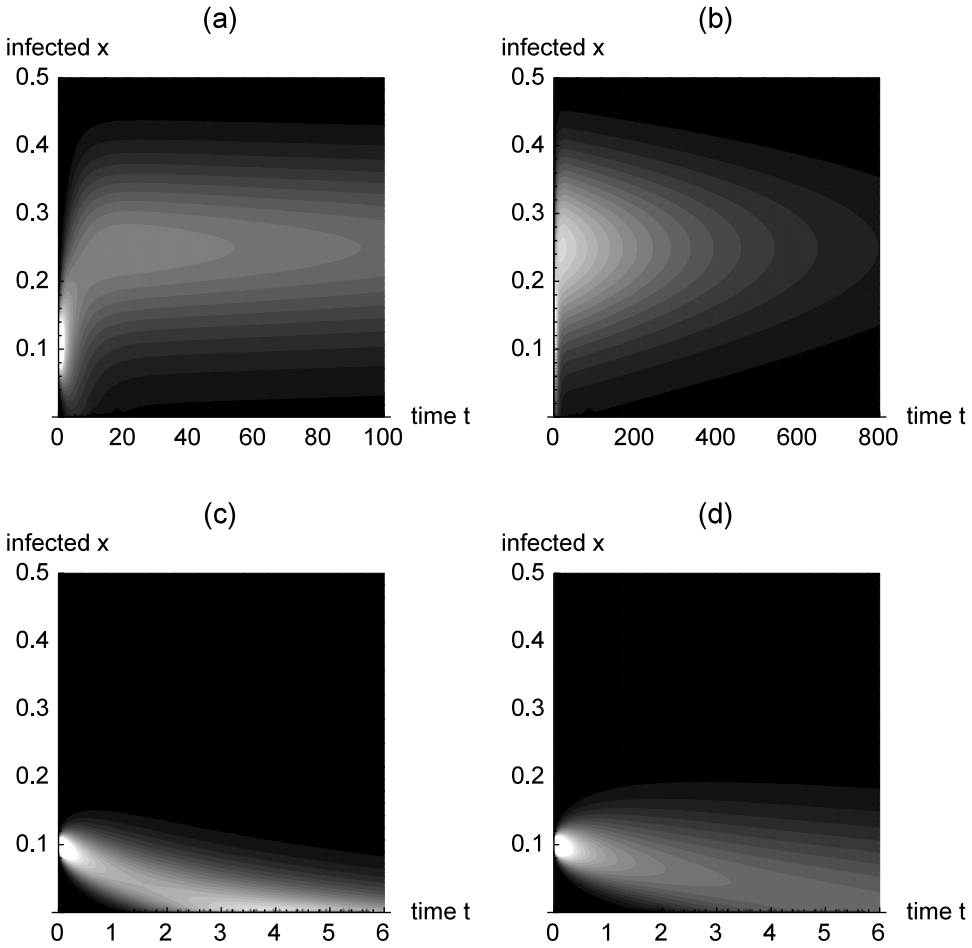

FIGURE 3.1. First line: Markov chain approximation with $\beta = 1.1$, $\gamma = 0.8$ and $N = 100$. Plot (a) seems to show the building-up of a stationary distribution. Over a longer time horizon, (b) demonstrates that probability mass is lost over time, and there is no stationary distribution.
In the second line (c) depicts the development for the model with $\beta = 0.8$, $\gamma = 1.1$ and N=100, and (d) shows the model $\beta = \gamma = 0.9$, $N = 100$. Both cases show quick extinction of the disease.

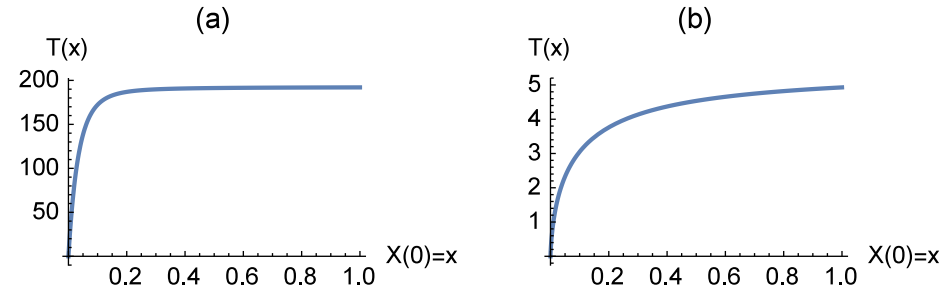

FIGURE 3.2. Expected time until absorption at zero as a function of the starting value.
(a) Slow extinction of the disease: model $\beta = 1.1$, $\gamma = 0.8$ and $N = 100$
(b) Quick extinction: The model $\beta = 0.8$, $\gamma = 1.1$ and N=100

4. Conclusions

We analyzed two kinds of stochastic SIS models in order to derive some of their basic properties from the related Fokker-Planck equations. The first type of model was recently presented in Chen and Kang [2014]: a model with saturated incidence was augmented with random disturbances of the strength of infection β. It turns out that under a suitable condition (2.13) on the risk adjusted basic reproduction number there is a well behaved stationary density related to the process of infected individuals. Moreover, the shape of the density, in particular its behavior at zero, was analyzed further. Besides the pure question of existence, it is possible to calculate the stationary density explicitly. If the basic condition (2.13) is violated, the only possible (generalized) density is given by the Dirac delta function at zero, which indicates that the process of infected converges (almost surely) to zero. Briefly worded, the stochastic model shows either a stochastic endemic equilibrium or the disease is extincted. In principle this resembles the two possible cases – endemic equilibrium and extinction – for deterministic versions of the SIS model with saturated incidence.

The second type of model treated in this paper can be derived from a continuous time Markov chain approach by applying the Kramers-Moyal approximation. No stationary density except the Dirac function exists in this case. Convergence of the process of infected to zero – extinction of the disease – happens almost surely. Moreover, absorption at zero happens almost surely in finite time and it is possible to calculate quantities like the expected time until extinction. This behavior is different from the case with extinction in the first model. In fact, the stochastic diffusion approximation inherits this property from the underlying Markov chain.

Many open question remain for further research. The overall approach can be applied to higher dimensional models with more than two relevant classes of individuals. Furthermore, several other approximation methods can be compared to the Kramers-Moyal approximation used in the present paper. Given that extinction happens for sure in the treated approximation model, it is important to observe that the time to absorption can be very large. Therefore it will be interesting to analyze the possibility of quasi-stationary distributions in such cases.

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