Probabilistic Drift Formulation of SIRS Models based on SPDEs and the Kolmogorov Equation

Günter Schneckenreither

Institute for Analysis and Scientific Computing, Vienna University of Technology, Wiedner Haupstraße 8-10, 1040 Vienna, Austria; *guenter.schneckenreither@tuwien.ac.at*

Simulation Notes Europe SNE 25(3-4), 2015, 199-206
DOI: 10.11128/sne.25.tn.10317
Received: October 5, 2015; Revised October 30, 2015;
Accepted: November 25, 2015

Abstract. The SIR (susceptible-infected-recovered) differential equations model for the spread of infectious diseases is very prominent in mathematical literature. The key interest of investigations is often to find and analyse extensions of the basic model structure in order to introduce a more detailed and supposedly realistic representation of the underlying disease and population dynamics. It is however true that problems and solutions in healthcare and health economics actually tend to require more and more sophisticated modelling approaches which are also capable of incorporating larger data sets as parametrisation in an effective way. This makes the comparison and combination of different modelling techniques and results an important research topic. This paper investigates a probabilistic drift formulation of the basic differential equations model which allows a very fine-grained parametrisation of the progression of diseases. It is shown that this formulation is capable of reproducing results from models with delay. Aggregation leads directly back to the traditional compartment approach and, in the heterogeneous case, a discrete representation can be interpreted as a system of local Markov processes. Furthermore some preliminary results on epidemiological measures like the basic reproduction number are presented.

Introduction

The SIR (susceptible-infected-recovered) epidemic model was originally derived by Kermack and McKendrick using a time-discrete scheme with multiple stages of disease progression [5]. For constant transition rates this model can be represented as a system of ordinary differential equations with three compartments, often referred to as the classical SIR(S) ODE model (1).

$$\begin{aligned} \partial_t s(t) &= -as(t)i(t) + cr(t) \\ \partial_t i(t) &= as(t)i(t) - bi(t) \\ \partial_t r(t) &= bi(t) - cr(t) \end{aligned} \tag{1}$$

Among the basic principles of the epidemic model the following are key to this paper:

(D1) The rates b and c and as well as the force of infection $f := ai(t)$ determine the (linear) flow or transition rates between the three compartments.

(D2) Since the force of infection acts on the number of susceptible individuals in a linear fashion, the incidence rate $ai(t)s(t)$ as a bilinear function implies a homogeneous mixing of the population.

(D3) For the basic SIRS model, the epidemic threshold (basic reproduction number) is given by $R_0 = \frac{a}{b}$. This threshold controls the existence of an endemic equilibrium.

It is well known that complex dynamic patterns such as bifurcations or periodic behaviour are more likely to occur when the spread of infection is constrained by heterogeneity or delay [1, 4, 10, 11].

Nevertheless, as a fist step towards a system description which is capable of simulating such behaviour, we regard the following one-dimensional SIS simplification ($S = R$).

$$d\xi(t) = -b\xi(t)dt + a\big(1-\xi(t)\big)\xi(t)dt \tag{2}$$

The state space of this dynamical system is the continuous bounded *disease space* $\Xi := [0,1]$, where a disease state $\xi \in \Xi$ indicates the strength or concentration of an infectious disease in a population, $\xi = 1$ meaning fully infected.

In order to obtain a discrete signal from this model define a susceptible domain $\Xi_s := [0, \frac{1}{2})$ and a infected domain $\Xi_i := [\frac{1}{2}, 1]$ such that $\Xi = \Xi_s \dot\cup \Xi_i$.

In this paper we extend the one-dimensional SIS model (2) with the periodic disease space $\Xi := [0,1)_{\text{per}}$ and define instead of the susceptible and infected regions, the domains *susceptible*, *infected*, *exposed*, *contagious* and *recovered/immune*, Ξ_s, Ξ_i, Ξ_e, Ξ_c, Ξ_r, such that

$$\Xi = \Xi_s \dot\cup \Xi_i \dot\cup \Xi_r, \qquad \Xi_e \dot\cup \Xi_c \subseteq \Xi_i. \tag{3}$$

It is obvious that the resulting disease space leads to a model for SEIRS-type epidemics (see Figure 1). The

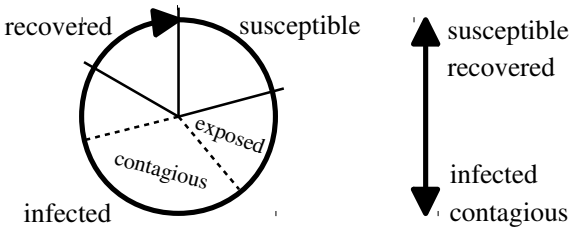

Figure 1: Periodic disease space (SIRS) compared to bounded disease space (SIS).

basic structure of the equations investigated in this paper is

$$d\xi(t) = g_1\big(\xi(t)\big)dt + g_2\big(\xi(t)\big) f\big(\xi(t)\big)dt. \tag{4}$$

Using a probabilistic approach (Kolmogorov forward equation) [1] and nonlinear incidence rates [4] it can be shown that the resulting transport (and diffusion) formulation (4) is a generalisation of the classical compartment approach and also of delay models.

For completeness this paper also introduces an arbitrary heterogeneity space X and takes into account random additive (white) noise such that theoretically we finally arrive at a stochastic partial differential equation (SPDE). For simplicity and readability most equations are written without the noise or respective diffusion term. For noise in the bounded domain $\Xi = [0,1]$ we have to ensure that the boundaries of the domain are honoured (compare [11]).

1 Heterogeneity States

The introduction of heterogeneity states in epidemic models has a long tradition and leads to abstract Cauchy problems and parabolic PDEs [8] with richer patterns of behaviour. From a modelling point of view, heterogeneity allows to simulate variable rates and parameters (i.e. heterogeneous populations) and to obtain inhomogeneous propagation and spread of a disease.

Let X be a finite dimensional topological vectorspace such that $\xi(t,x)$ indicates the abundance of a infectious disease (think of viral concentration, number of infected individuals, ...) with heterogeneity state $x \in X$. The topological vectorspace X can for example represent a spatial domain or the ages of the individuals of a population. The most natural idea for epidemic models with heterogeneous populations is that the force of infection depends on an aggregated (nonlocal) concentration of the disease $\xi_N(t,x)$ instead of the local state $\xi(t,x)$.

The aggregated state can for example be a weighted integral

$$\xi_N(t,x) := \int_X \lambda(x,y)\xi(t,y)\,dy \tag{5}$$

with kernel $\lambda(x,y)$, which allows to model different types of nonlocal interaction [9]. For example a Gaussian kernel can simulate a diffusive interaction process and the Taylor series expansion of $\xi(t,y)$ around x allows to find a differential representation $\xi_N = \lambda_1\xi + \lambda_2\Delta_x\xi$.

Technically the aggregated state $\xi_N(t,x)$ replaces the actual local state $\xi(t,x)$ in the argument of the force of infection $f(\cdot)$.

The epidemic threshold for heterogeneous SIS systems [11] corresponds under some assumptions (separable and symmetric kernel λ, constant population density) to the integral

$$R_0 = \int_X \lambda(x,x)\frac{a(x)}{b(x)}\,dx. \tag{6}$$

2 Probabilistic Formulation and Semilinear Incidence

A probabilistic formulation of homogeneous SIR models can be found in [1] for example, where the authors transform the homogeneous SIR model into a transport problem and derive the Kolmogorov forward equation from a discrete SIR approach. They also state that the probabilistic representation with no diffusion has degenerate distributions as its limit, conforming with the theory on epidemic thresholds and equilibria.

2.1 SIS Model

For the heterogeneous SIS model (nonlocal force of infection) the *stochastic formulation*

$$d\xi(t,x) = -b\xi(t,x)dt + a\big(1-\xi(t,x)\big)\xi_N(t,x)dt \tag{7}$$

is (locally) a Itô diffusion since the drift term is Lipschitz continuous and the corresponding Kolmogorov forward equation [7] (*probabilistic approach*) is

$$\frac{\partial}{\partial t}p(t,x,\xi) = (a\xi_N+b)p(t,x,\xi) + \\ +\big((a\xi_N+b)\xi - a\xi_N\big)\frac{\partial}{\partial \xi}p(t,x,\xi). \tag{8}$$

The drift term can be separated into

$$g_1(t,x,\xi) := -b\xi \tag{9}$$
$$g_2(t,x,\xi) := a\big(1-\xi\big) \tag{10}$$
$$f(t,x,\xi_N) := \xi_N \tag{11}$$

such that $g_1 \leq 0$ defines a constant drift towards the susceptible state $\xi = 0$ and, depending on the force of infection $f(\xi_N)$, $g_2 \geq 0$ generates drift towards the infected state $\xi = 1$. Note that all three functions are linear in ξ or ξ_N respectively (see Figure 2). In the

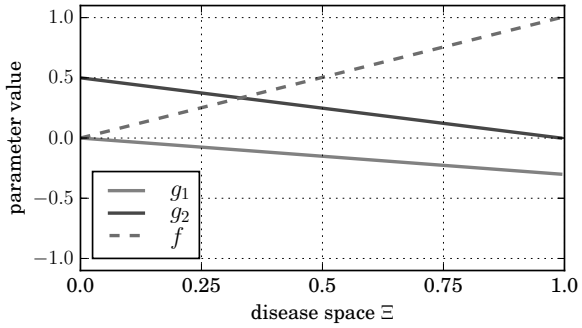

Figure 2: Parameter functions of the SIS model for arbitrary a and b.

stochastic formulation (7) $\xi(t,x)$ is actually a random field. In the probabilistic representation (8) it is necessary to replace the aggregated random variable ξ_N respectively the force of infection $f(\xi_N)$ with a statistic [8]. A plausible choice is of course the expectation value

$$\mathbb{E}\big[f(\xi_N)\big] = \int_\Xi f(\eta)p_N(t,x,\eta)\,d\eta \tag{12}$$

where $p_N(t,x,\xi) = \int_X \lambda(x,y)p(t,y,\xi)\,dy$ is the distribution of the accumulated random variable ξ_N.

2.2 SEIRS Model

For the stochastic SEIRS approach, i.e. when the disease space is periodic, and the corresponding Kolmogorov forward equation

$$\frac{\partial}{\partial t}p(t,x,\xi) = -\frac{\partial}{\partial \xi}\Big\{\Big(g_1(\xi) + \\ +g_2(\xi)\int_\Xi f(\eta)p_N(t,x,\eta)\,d\eta\Big)p(t,x,\xi)\Big\} \tag{13}$$

drift happens in positive direction only (except maybe for local Ξ areas with negative drift).

Let g_1 define a constant drift, which describes normal disease progression like entering and leaving a contagious phase Ξ_c, which is a subset of the infected phase Ξ_i, or transition from infected to recovered/immune Ξ_r and from recovered to susceptible Ξ_s. The drift from the susceptible domain to the infected domain however shall be generated by g_2 with strength controlled by the force of infection f. From a certain point of view, the function g_2 compensates for the lack of drift generated by g_1 in the susceptible domain Ξ_s. The total drift is given by

$$g(\xi) := g_1(\xi) + g_2(\xi)\int_\Xi f(\eta)p_N(t,x,\eta)\,d\eta. \tag{14}$$

Figure 3 shows a possible configuration of the parameter functions.

We make the following heuristic (and not fully necessary) but plausible assumptions on the parameter functions.

(P1) The basic drift term g_1 is mostly constant with a value $k \in [0,1]$ except for a region around the interface between the susceptible and the infected domain, where g_1 vanishes. The value k determines the speed of disease progression. Of course the exact shape of g_1 is a result of modelling decisions. Also the value k is actually only a scaling variable which relates disease progression to time (see section 3).

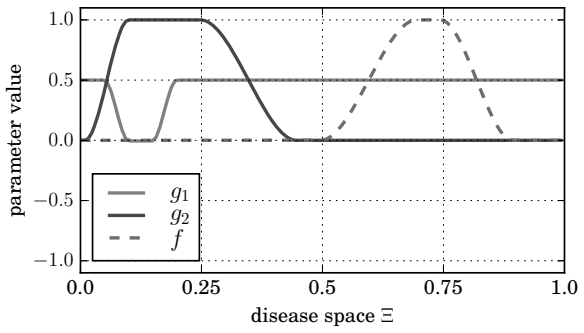

Figure 3: Parameter functions of the SIRS model with periodic disease space. Here g_1 actually takes (small) negative values in the susceptible domain and g_2 takes very large values compared to g_1.

(P2) The incidence drift term g_2 is larger than zero only in a region around the interface between the susceptible and the infected domain.

(P3) As a consequence in the infected region the total drift term is mostly constant $g|_{\Xi_i} \equiv k$ independent of the shape of p_N (i.e. the force of infection).

(P4) The function f is larger than 0 only in the contagious domain or in other words, the support of f determines the contagious domain (compare fuzzy sets). We can assume that f is normalised $\|f\|_{L^\infty(\Xi)} = \|f\|_\infty = 1$.

(P5) There exists a configuration p_i of p_N such that the force of infection takes a maximum value.

Set $F^{-1} := \|f\|_1$ then this configuration is given by $p_i(\xi) = Ff(\xi)$ and the inequality

$$1 = F\|f\|_1 \leq F\|f\|_2 \leq F\|f\|_\infty = F \tag{15}$$

shows that $F^{-1} \leq \|f\|_2 \leq 1$.

We can interpret this configuration as a situation in which locally all susceptible individuals come into contact with infected individuals and the speed of spread is at the maximum. In other words p_i is fully infectious.

Note that these assumptions restrict the drift SIRS model to a small subset of possible configurations. They however simplify some technical considerations in the following sections. But still the probabilistic formulation is capable of producing patterns that cannot be obtained with the stochastic formulation (see Figure 6).

3 Delay Models

As mentioned before delay plays an important role in the dynamics of epidemiology. In [4] three different types of delays are distinguished:

- *Temporary immunity* corresponds to the recovered/immune phase after the infection period.
- *Delay caused by the latency in a vector* happens when infection is spread by agents (e.g. mosquitoes).
- The *latent period in a host* is the time delay between infection and the contagious phase.

The following delay differential equation

$$d\xi(t) = -b\xi(t)dt + a\big(1-\xi(t)\big)\xi(t-\tau)dt \tag{16}$$

models a latent period in the host [4]. The corresponding Kolmogorov forward equation is

$$\frac{\partial}{\partial t}p(t,\xi) = -\frac{\partial}{\partial \xi}\Big\{\big(-b\xi + a(1-\xi)\xi_T\big)p(t,\xi)\Big\} \tag{17}$$

where again we replace the stochastic force of infection $f(\xi_T)$ with the expectation $\mathbb{E}\big[f\big(\xi(t-\tau)\big)\big]$. Since in (P4) it was assumed that the support of the force of infection f is Ξ_c, the expectation can be written as

$$\mathbb{E}\big[f\big(\xi(t-\tau)\big)\big] = \int_{\Xi_c} f(\eta)p(t-\tau,\eta)\,d\eta. \tag{18}$$

From (P3) it follows that drift (velocity) is constant $g(\xi) \equiv k$ in Ξ_i and especially in a region around Ξ_c such that

$$\frac{\partial}{\partial t}p(t,\xi)\Big|_{\Xi_i} = -k\frac{\partial}{\partial \xi}p(t,\xi)\Big|_{\Xi_i} \tag{19}$$

which by the formal argument with scaling $\xi = k\tau$

$$\lim_{\tau\to 0}\frac{p(t,\xi)-p(t-\tau,\xi)}{\tau} = \lim_{\tau\to 0}\frac{kp(t,\xi+k\tau)-kp(t,\xi)}{k\tau} \tag{20}$$

leads to

$$p(t-\tau,\xi) = p(t,\xi+k\tau) \tag{21}$$

in Ξ_i. Inserting into (18) results in

$$\mathbb{E}\big[f(\xi(t-\tau))\big] = \int_{\Xi_c} f(\eta)p(t,\eta+k\tau)\,d\eta = \int_{\Xi_c+k\tau} f(\eta-k\tau)p(t,\eta)\,d\eta. \quad (22)$$

Setting $\Xi_{c'} := \Xi_c + k\tau \subset \Xi_i$ finally means that the new contagious domain was shifted to the right by the scaled time delay $k\tau$ and we arrive back at the original model with different (shifted) force of infection $f(\xi)$.

This shows that the probabilistic approach is very suitable for modelling delay in epidemic spread. Or in other words, the probabilistic formulation is inherently delayed.

4 Discretisation and Numerical Scheme

For the sake of regularisation [2, 3] a relatively small noise respectively diffusion term is added to the equations. As a numerical scheme for the stochastic equations the straight-forward explicit Euler-Maruyama method [6] is used.

4.1 Discretisation of the Probabilistic Formulation

For the probabilistic formulations (Kolmogorov equations), a local Markov chain [8] representation can be constructed.

To that end let $\mathbf{p}(t,x) \in \mathbb{R}^n_+$ be a discretisation of $p(t,x,\xi)$ in the disease space Ξ with $\sum_{i=1}^n p_i(t,x) = 1$ for all t and x. The local Kolmogorov equation with discretised disease space is formally given by

$$\frac{\partial}{\partial t}\mathbf{p} = -\frac{\partial}{\partial \xi}\{\mathbf{g}\odot\mathbf{p}\} \quad (23)$$

where $\odot$ denotes the element-wise multiplication and $\mathbf{g}$ is the vectorised drift term. From (13) we conclude that

$$\mathbf{g} = \mathbf{g}_1 + \mathbf{g}_2(\mathbf{f}\cdot\mathbf{p}_N) \quad (24)$$

where $\mathbf{f}\cdot\mathbf{p}_N$ is the scalar product. The accumulated discrete distribution $\mathbf{p}_N$ is calculated from a finite number of discretised heterogeneity states from X.

Define the differentiation matrices

$$D_+ = \begin{pmatrix} -1 & & +1 \\ +1 & -1 & \\ & +1 & -1 \\ & & +1 \; -1 \end{pmatrix}, \quad D_- = \begin{pmatrix} -1 & +1 & & \\ & -1 & +1 & \\ & & -1 & +1 \\ +1 & & & -1 \end{pmatrix},$$

which calculate the derivative in negative and positive ξ direction respectively (up to discretisation length and here displayed for the periodic case only), then

$$\frac{\partial}{\partial t}\mathbf{p} = D_+(\mathbf{g}\odot\mathbf{p})_{>0} + D_-(\mathbf{g}\odot\mathbf{p})_{<0} \quad (25)$$

$$= \Big(D_+\,\mathrm{diag}(\mathbf{g}_{>0}) + D_-\,\mathrm{diag}(\mathbf{g}_{<0})\Big)\mathbf{p} \quad (26)$$

$$= M(\mathbf{g})\mathbf{p}, \quad (27)$$

where $\mathrm{diag}(\mathbf{a})$ is the diagonal matrix with the vector $\mathbf{a}$ as its diagonal.

Accordingly the SIRS model with discretised disease space, without diffusion and parameters as in Figure 3 locally corresponds to the Markov model visualised in Figure 4 with transition matrix $I + dtM(\mathbf{g})$ portrayed in Figure 5 and also to a system of ODEs with a finite number of compartments.

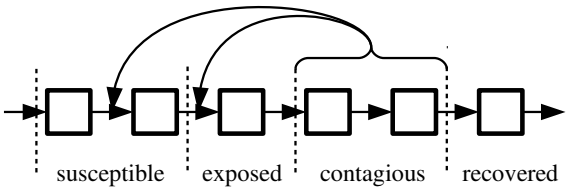

Figure 4: Discretised periodic disease space (SIRS) with feedback (concentration in Ξ_c controls force of infection which determines the flow from Ξ_s to Ξ_i).

4.2 Global Explicit Scheme

The corresponding global explicit iteration scheme to (25)-(27) can be formulated as a cellular automaton [8]. The discretisation of the heterogeneity space X can be interpreted as a lattice of cells, which leads to a formal description of a cellular automaton with

(X) a discretisation of X as cellular space,

(S) vectors $\mathbf{p} \in \mathbb{R}^n_+$ as cell states,

(N) a finite sum (discretisation of (5)) as a discrete version of the accumulated (neighbourhood-) state $\mathbf{p}_N$,

(L) and a local iteration rule given by (25)-(27).

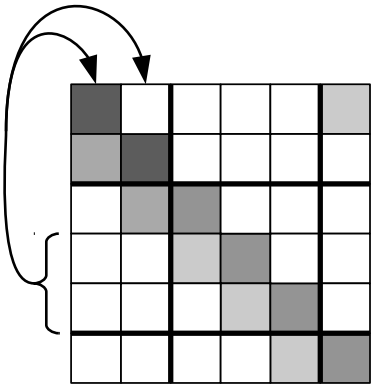

Figure 5: Basic structure of the transition matrix. Concentration in the contagious domain controls flow from the susceptible to the infected domain. For simplicity no negative flow is shown.

In a more abstract fashion also the stochastic formulation can be interpreted in the context of cellular automata.

(S) Instead of discretised distributions (vectors), the states of the cells shall be random variables in Ξ.

(N) Accordingly the accumulated random state is a multivariate random variable or defined by a measurable function $\Xi^d \to \Xi$, which maps "neighbouring" random variables onto a "accumulated" random variable.

(L) The local iteration rule is defined by a conditional probability or Markov kernel.

From a modelling point of view, the cellular automaton approach can be classified as a direct modelling approach (local Markov model) with discrete heterogeneity space, whereas the discretisation of the differential equation formulations is a numerical scheme. Visualisations of a simulation run can be seen in Figure 6.

5 The Probabilistic Formulation Extends The Compartment Approach

For the SIRS model let the dimension of the discretisation of the disease space be $n := 3$ and identify the first dimension with Ξ_s, the second dimension with $\Xi_i = \Xi_c$ and the third dimension with Ξ_r. For the discretisation

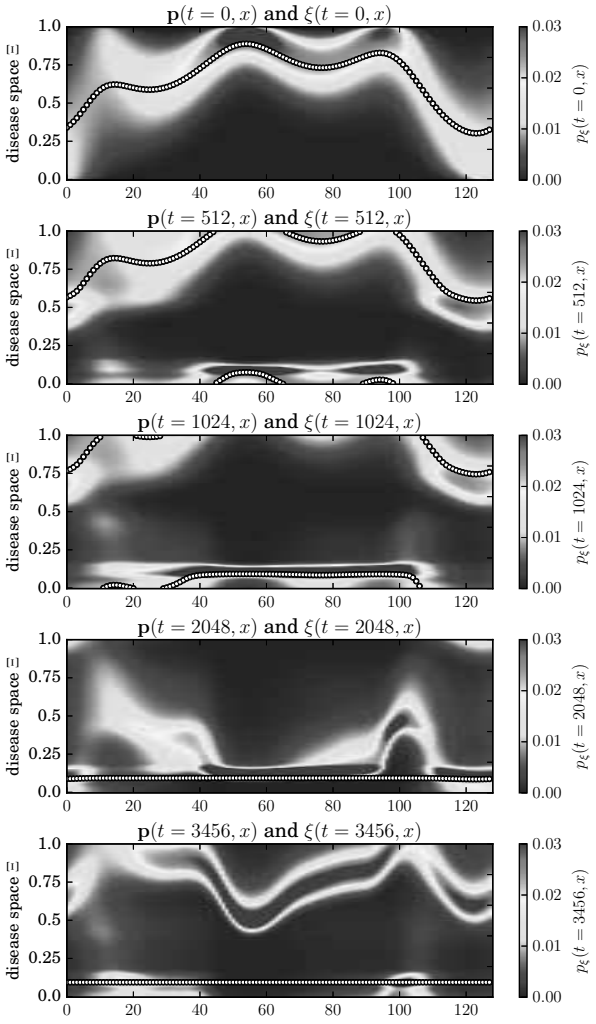

Figure 6: Visualisation of the probabilistic heterogeneous SIRS model ($\mathbf{p}(t,x,\xi)$, colorscale) and the corresponding stochastic model ($\xi(t,x)$, black/white line) without diffusion. The (periodic) heterogeneity space X is displayed in horizontal direction.

of the parameter functions g_1, g_2 and f we conclude the following from (P1)-(P4).

$$0 \approx g_{11} := \int_{\Xi_s} g_1(\xi)\, d\xi \tag{28}$$

$$b := g_{12} := \int_{\Xi_i} g_1(\xi)\, d\xi \tag{29}$$

$$c := g_{13} := \int_{\Xi_r} g_1(\xi)\, d\xi \tag{30}$$

$$a := g_{21} := \int_{\Xi_s} g_2(\xi)\, d\xi \tag{31}$$

$$0 \approx g_{22} := \int_{\Xi_i} g_2(\xi)\,d\xi \tag{32}$$

$$0 \approx g_{23} := \int_{\Xi_r} g_2(\xi)\,d\xi \tag{33}$$

$$0 \approx f_1 := \int_{\Xi_s} Ff(\xi)\,d\xi \tag{34}$$

$$1 \approx f_2 := \int_{\Xi_i} Ff(\xi)\,d\xi \tag{35}$$

$$0 \approx f_3 := \int_{\Xi_r} Ff(\xi)\,d\xi \tag{36}$$

Instead of discretisation by integration as shown above, it should also be possible to use point evaluations at the interfaces between the different subdomains. According to (P4), in (34)-(36) also the L^∞-norm could be used.

Inserting in the evolution equation (25)-(27) leads to

$$\begin{aligned}\frac{\partial}{\partial t}\begin{pmatrix}p_1\\p_2\\p_3\end{pmatrix} &= D_+\,\mathrm{diag}(ap_{N,2},b,c)\begin{pmatrix}p_1\\p_2\\p_3\end{pmatrix}\\ &= \begin{pmatrix}-ap_{N,2} & & +c\\ +ap_{N,2} & -b & \\ & +b & -c\end{pmatrix}\begin{pmatrix}p_1\\p_2\\p_3\end{pmatrix}.\end{aligned} \tag{37}$$

This corresponds to the compartment formulation of the heterogeneous SIRS model. Additionally setting $\mathbf{p}_N := \mathbf{p}$ results in the classical homogeneous SIRS ODE model (1).

6 Outlook – Basic Reproduction Number

The basic reproduction number R_0 can be interpreted as the number of secondary infections created by one infectious individual in a fully susceptible population [11, 1, 10, 4].

In order to find a similar measure for the probabilistic drift formulation we start from a fully susceptible homogeneous population which can be represented by the equilibrium distribution p_s of (13) when starting from distributions with support in Ξ_s or an arbitrary non-equilibrium distribution p_s with support in Ξ_s. There also exists a configuration p_k with *unit force of infection*, which is characterised by

$$g(\xi) = g_1(\xi) + g_2(\xi)\int_\Xi f(\eta)p_k(\eta)\,d\eta \approx k. \tag{38}$$

For example using (P5) and p_s we can set

$$p_k := \left(1 - \frac{k}{\max g_2}\right)p_s + \frac{k}{\max g_2}p_i. \tag{39}$$

6.1 Ratio of Infectiousness

If we set $p_N := p_k$ and assume that the force of infection stays constant over time, (13) can be written as

$$\frac{\partial}{\partial t}p(t,\xi) = -k\frac{\partial}{\partial \xi}p(t,\xi). \tag{40}$$

Let us calculate the infectiousness of the solution of the initial value problem (40) with initial condition $p(0,\xi) = p_k(\xi)$ at some later time t and compare it with the infectiousness of p_k. From (21) we know that $p(t,\xi) = p(0,\xi - kt) = p_k(\xi - kt)$.

Accordingly the function $Q_1 : [0,1)_{\mathrm{per}} \to \mathbb{R}$,

$$\begin{aligned}Q_1(t) &:= \frac{\exp\left(\int p_k(\xi - kt)f(\xi)\,d\xi\right)}{\exp\left(\int p_k(\xi)f(\xi)\,d\xi\right)}\\ &= \frac{\exp\left(\mathbb{E}^{p_k}\left[f(\xi + kt)\right]\right)}{\exp\left(\mathbb{E}^{p_k}\left[f(\xi)\right]\right)}\end{aligned} \tag{41}$$

can be used for measuring the delayed (t) infectiousness of an initially susceptible population with one unit force of infection and stores information about the strength and succession of epidemic waves. Due to the simplifications made, this measure may not be very accurate in practice.

6.2 Balance of Infectiousness

From (29) and (31) we may assume that

$$R_0 := \frac{\int_{\Xi_s} g_2(\xi)\,d\xi}{\int_{\Xi_c} g_1(\xi)\,d\xi} \tag{42}$$

is useful as a measure for secondary infections. This is motivated by the definition of the basic reproduction number of the compartment SIRS model, which is the ratio between the flow rate to the contagious domain a and the flow rate from the contagious domain b under the condition of a fully susceptible population with one unit force of infection.

In- and outflow of the contagious domain under the same conditions in the probabilistic model at time t is accessible through the balance equation

$$\begin{aligned}\frac{\partial}{\partial t}\int_{\Xi_c} p(t,\xi)\,d\xi &= -k\int_{\Xi_c}\frac{\partial}{\partial \xi}p(t,\xi)\,d\xi\\ &= k\big(p(t,\xi_0) - p(t,\xi_1)\big)\end{aligned} \tag{43}$$

where ξ_0 and ξ_1 are the boundaries of Ξ_c. But since the contagious domain is defined by f, with integration by

parts we arrive at

$$\begin{aligned}\frac{\partial}{\partial t}\int_{\Xi} p(t,\xi)f(\xi)\,d\xi &= -k\int_{\Xi} f(\xi)\frac{\partial}{\partial \xi}p(t,\xi)\,d\xi \\ &= k\int_{\Xi} p(0,\xi)f'(\xi+kt)\,d\xi = k\mathbb{E}^{p_k}\left[f'(\xi+kt)\right]\end{aligned} \tag{44}$$

which yields

$$Q_2(t) := \frac{\exp\left(kp(t,\xi_0)\right)}{\exp\left(kp(t,\xi_1)\right)} = \frac{\exp\left(kp_k(\xi_0-kt)\right)}{\exp\left(kp_k(\xi_1-kt)\right)} \tag{45}$$

and

$$Q_3(t) := \exp\left(k\mathbb{E}^{p_k}\left[f'(\xi+kt)\right]\right) \tag{46}$$

as possible time-dependent measures for secondary infections. As before we must note that these measures rely on a great number of simplifications.

For the extremely simplified case

$$g_1(\xi) := \mathbb{I}_{[\frac{0}{4},\frac{1}{4})}(\xi) \tag{47}$$

$$g_2(\xi) := k\mathbb{I}_{[\frac{1}{4},\frac{4}{4})}(\xi) \tag{48}$$

$$f(\xi) := \mathbb{I}_{[\frac{2}{4},\frac{3}{4})}(\xi) \tag{49}$$

$$p_k(\xi) := 4(1-k)\mathbb{I}_{[\frac{0}{4},\frac{1}{4})}(\xi) + 4k\mathbb{I}_{[\frac{2}{4},\frac{3}{4})}(\xi) \tag{50}$$

we obtain (partially in a formal way only) $R_0 = \frac{1}{k}$ and the functions Q_1, Q_2, Q_3 portrayed in Figure 7. In a next step numerical tests must be conducted in order to find information about the reliability of these measures.

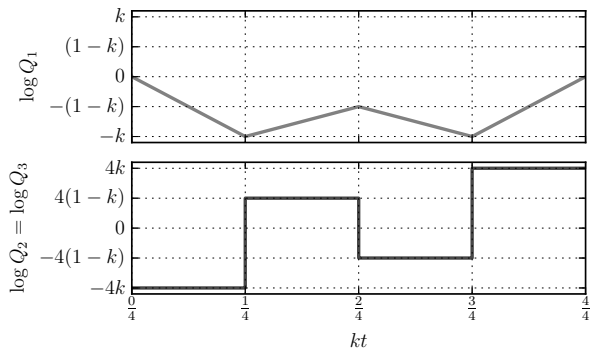

Figure 7: With the parameter functions defined in (47)-(50) the functions Q_2 and Q_3 coincide in a formal way. In logarithmic scale the flow-based measure $Q_2 = Q_3$ is the derivative of the delay-based measure Q_1. This figure assumes $k \approx \frac{2}{3}$.

Acknowledgements

This work was funded by the K-Project DEXHELPP, which is supported by Austrian BMVIT and BMWFW as well as the state of Vienna via COMET – Competence Centers for Excellent Technologies. The COMET program is processed by FFG.

References

[1] Chalub FAAC, Souza MO. The SIR epidemic model from a PDE point of view. *Mathematical and Computer Modelling*. 2011; 53(7-8): 1568–1574. doi: 10.1016/j.mcm.2010.05.036.

[2] Gyöngy I. Existence and uniqueness results for semilinear stochastic partial differential equations. *Stochastic Processes and their Applications*. 1998; 73(2): 271–299.

[3] Gyöngy I, Nualart D. Implicit scheme for quasi-linear parabolic partial differential equations perturbed by space-time white noise. *Stochastic Processes and their Applications*. 1995; 58(1): 57–72.

[4] Huang G, Takeuchi Y. Global analysis on delay epidemiological dynamic models with nonlinear incidence. *Journal of Mathematical Biology*. 2010; 63(1): 125–139. doi: 10.1007/s00285-010-0368-2.

[5] Kermack WO, McKendrick AG. A Contribution to the Mathematical Theory of Epidemics. *Proceedings of the Royal Society A: Mathematical, Physical and Engineering Sciences*. 1927; 115(772): 700–721.

[6] Lord G, Powell CE, Shardlow T. *An introduction to computational stochastic PDEs*. Cambridge University Press; 2014.

[7] Øksendal BK. *Stochastic differential equations; an introduction with applications*. Berlin: Springer; 2007.

[8] Schneckenreither G. *Developing Mathematical Formalisms for Cellular Automata in Modelling and Simulation* [master thesis]. Vienna University of Technology; 2014.

[9] Schneckenreither G. Modelling SIR-type epidemics by ODEs, PDEs, difference equations and cellular automata – A comparative study. *Simulation Modelling Practice and Theory*. 2008; 16(8): 1014–1023, doi: 10.1016/j.simpat.2008.05.015.

[10] Vynnycky E, White RG. *An introduction to infectious disease modelling*. Oxford University Press; 2010.

[11] Widder A, Kuehn C. Heterogeneous Population Dynamics and Scaling Laws near Epidemic Outbreaks. *ArXiv e-prints*. 2014; 1411.7323.