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The SIR epidemic model from a PDE point of view

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ABSTRACT

We present a derivation of the classical Susceptible-Infected-Removed (SIR) and Susceptible-Infected-Removed-Susceptible (SIRS) models through a mean-field approximation from a discrete version of SIR(S). We then obtain a hyperbolic forward Kolmogorov equation, and show that its projected characteristics recover the standard SIR(S) model. Moreover, for the SIRS model, we show that the long time limit of the SIRS model will be a Dirac measure supported on the corresponding isolated equilibria. For the SIR model, we show that the long time limit is a Radon measure supported in a segment of nonisolated equilibria.

1. Introduction

A very fruitful modeling paradigm in epidemiology is the so-called compartmental models, with dynamics governed by mass–action laws. Most classical epidemiological models are of this type, and this has led to a number of both quantitative and qualitative predictions in the disease dynamics [1]. More recently, there is a growing interest in discrete, agent-based, models [2]. See also [3] for a comparison between different models. In many cases, these models are thought to be more realistic, and able to capture important dynamical features that are not present in the continuous models.

Here, we follow the ideas in [4], to study the large-population regime of the discrete versions of the SIR(S) (Susceptible-Infected-Removed-(S)usceptible) that can be found in [5]. In this way, we obtain a Hyperbolic Forward Kolmogorov equation for the probability density evolution. It is well known, through the method of characteristics, that there is a strong linkage between solutions to first-order Partial Differential Equations (PDEs) and systems of Ordinary Differential Equations (ODEs). This link has already been used by Chalub and Souza [4], in establishing the connection between the Replicator Dynamics and some regimes of the Moran process. Thus, it is not entirely surprising that, the projected characteristics of this PDE will also be related to the classical SIR and SIRS ODE models. Despite the differences in the qualitatively behavior of the solutions of the SIR and the SIRS models, we shall abuse language and use only "SIR" for both models, unless stated otherwise.

The modeling through PDEs has some advantages. In particular, it allows the introduction of higher-order effects as, for example, stochasticity, through the addition of a second-order term to the equation. On the other hand, if we consider a Markov Chain discrete model in population dynamics, and consider its limit of large population (under suitable conditions), we naturally obtain a PDE for $p(t, x)$, the probability density to find the population at state x at time t. The ODE can then be obtained as the hyperbolic limit of the PDE. Such a limit can be achieved in two ways: either by considering the initial dynamics of the PDE; or by working in the parameter regimes that are convection dominated. In short, this means that the dynamics of the discrete population can be approximated for short times and for a large population by a certain ODE—the derivation of this ODE requires an introduction of intermediate models, a stochastic differential equation or a partial differential one.

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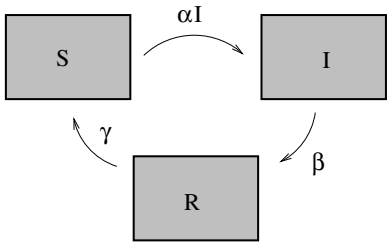

Fig. 1. The SIRS model represented as a compartmental model. Arrows denotes possible changes and transition probabilities.

The ODE approach can be seen in [6], while the PDE modeling was the subject of a previous work from the authors, where the replicator equation was obtained as the limit of the finite-population discrete Moran process [4]. The resulting equation is of singular type and required a specific analysis of its behavior [7].

In this work, we will study in a certain level of detail the SIR epidemic model from the PDE point of view.[1] This is one of the most elementary and well studied model in mathematical epidemiology. See [8,1].

In this vein, we shall look at the Markov chain model and, from a first order expansion in the inverse of the population size, we shall obtain a Hyperbolic forward Kolmogorov equation that contains the SIR–ODE system as its characteristics. Moreover, for the SIRS system, we shall prove that the solution converges, as $t \to \infty$, to a Dirac measure supported at the unique stable equilibrium of the particular regime. For the SIR system in the epidemic-regime, we obtain a somewhat weak result: the solution will converge to a measure supported at the segment of equilibria.

The outline of the work is as follows: in Section 2, we present a review of compartmental models, including discrete-deterministic, discrete-stochastic, and continuous-deterministic models. In 2.4, we derive a hyperbolic Kolmogorov partial differential equation for the discrete-stochastic model, in the large population limit. Section 3 studies the problem as a transport problem, and shows some existence results. In Section 4 we present large time asymptotics for the SIRS and SIR PDE problem. Concluding remarks are given in Section 5.

2. Discrete and continuous SIR models

2.1. Compartmental models

Compartmental models can be seen as structured population models, where the population is classified with respect to their role in the epidemiological process. See Fig. 1. While they seem to be the simplest paradigm for modeling diseases that are transmitted by contact, and are not directly lethal, compartmental models enjoy a long and successful tradition, which dates back at least to the work of [9] and the sequence of works by Kermack and McKendrick [10–12].

We now briefly review the classical compartmental models without vital dynamics, i.e., without considering the birth and death of individuals. These models seems to be appropriate for modeling on a timescale much smaller than the average population turnover. In all subsequent models, we shall denote by $S(t)$ the fraction of the population that is healthy and by $I(t)$ the fraction of the population that is unhealthy and contagious. Additionally, $R(t)$ will denote the fraction of the population that was no longer contagious, and it usually thought as cured and immune to the particular disease being studied.

SI, SIR, and SIRS models can be described by the following ODE system:

$$\begin{aligned} S' &= \gamma R - \alpha IS \\ I' &= \alpha IS - \beta I \\ R' &= \beta I - \gamma R. \end{aligned} \tag{1}$$

We always assume in this work, without loss of generality, the normalization $S + I + R = 1$. When $\beta = \gamma = 0$, we have the SI (Susceptible-Infected) model. In this model, the population fate is to become totally infected. The SIS model can be modeled by the system:

$$\begin{aligned} S' &= \gamma I - \alpha IS, \\ I' &= \alpha IS - \gamma I, \end{aligned} \tag{2}$$

with $\alpha \neq 0 \neq \gamma$. System (2) has two equilibria: the so called disease-free equilibrium, $S = 1$ and $I = 0$; and the so called endemic equilibrium: $S^* = \gamma/\alpha$, and $I^* = 1 - S^*$. The long term behavior of a population modeled by (2) depends on the

[1] The expression SIR appears in the literature in two different contexts: one as a discrete evolutionary system, used in general in computer simulations; the second as an ODE system. We hope that all these different meanings are clear from the context.

basic reproductive factor:

$$R_0^{\mathrm{SIS}} = \frac{\alpha}{\gamma}. \tag{3}$$

On noticing that (2) implies $S + I = 1$, we have that (2) is equivalent to the logistic equation

$$S' = \alpha \left(\frac{1}{R_0^{\mathrm{SIS}}} - S \right) (1 - S).$$

Then, we have that if $R_0^{\mathrm{SIS}} \leq 1$, then the dynamics converges to the disease-free equilibrium, and if $R_0^{\mathrm{SIS}} > 1$, the dynamics converges to the endemic equilibrium.

The next natural step is to consider immunity either temporary or permanent. The simplest models in this vein are the SIR and SIRS, respectively, with dynamics given by (1) (with $\gamma = 0$ for the SIR model). The SIR and SIRS models display somewhat different dynamics: the former has a non-isolated segment of disease-free equilibria, the latter having the disease free equilibrium $S = 1, I = R = 0$, and the endemic equilibrium $S^* = 1/R_0^{\mathrm{SIR}}$, $I^* = (1 - 1/R_0^{\mathrm{SIR}})(1 + \beta/\gamma)^{-1}$, and $R^* = (1 - 1/R_0^{\mathrm{SIR}})(1 + \gamma/\beta)^{-1}$, where $R_0^{\mathrm{SIR}} = \alpha/\beta$.

For more information on the dynamics of classical SI(S)/SIR(S) models, we refer to standard texts on mathematical epidemiology such as [1,13,8,14].

2.2. Probabilistic discrete-time SIR models

We now consider a stochastic version of the SIR, as in [5].

At each time step of size $\Delta t > 0$ we select one individual at random:

- If it is **S**, then it changes to **I** with probability proportional to the fraction of **I** in the remainder, $\alpha N I(t)/(N - 1)$;
- If it is **I**, then it changes to **R** with constant probability β;
- If it is **R**, then it changes to **S** with constant probability γ.

This can be summarized in the following diagram:

$$\mathbf{S} + \mathbf{I} \xrightarrow{\alpha} \mathbf{I} + \mathbf{I}, \qquad \mathbf{I} \xrightarrow{\beta} \mathbf{R}, \qquad \mathbf{R} \xrightarrow{\gamma} \mathbf{S}.$$

2.3. Deterministic discrete-time SIR models

The deterministic SIR model is given by

$$\begin{aligned} S(t + \Delta t) &= S(t)(1 - A(\Delta t) I(t)) + R(t) C(\Delta t) \\ I(t + \Delta t) &= I(t)(1 - B(\Delta t)) + A(\Delta t) I(t) S(t) \\ R(t + \Delta t) &= R(t)(1 - C(\Delta t)) + B(\Delta t) I(t). \end{aligned} \tag{4}$$

It is natural to assume that

$$A(\Delta t) \approx A_0 \Delta t, \qquad B(\Delta t) \approx B_0 \Delta t, \qquad C(\Delta t) \approx C_0 \Delta t. \tag{5}$$

System (4), with the assumptions (5), can be seen as an Euler discretization for (1). In the limit of $\Delta t \to 0$, standard numerical analysis arguments [15,16] can show that (4) is a good *discrete approximation* to the continuous model (1). Alternatively, as advocated by Chalub and Souza [4], we can see (1) and (5) as good *continuous approximations* to the discrete (and more biologically sensible) model.

We also refer to [5] for results, similar to the continuous one, to the dynamics of (4).

2.4. From discrete to continuous modeling

Constants α, β and γ depend, in principle, in N and Δt. As we are interested in the limit behavior when $N \to \infty$, $\Delta t \to 0$ we will assume the following *scaling* relations

$$\lim_{N \to \infty,\ \Delta t \to 0} \frac{\alpha}{N \Delta t} = a, \qquad \lim_{N \to \infty,\ \Delta t \to 0} \frac{\beta}{N \Delta t} = b, \qquad \lim_{N \to \infty,\ \Delta t \to 0} \frac{\gamma}{N \Delta t} = c, \tag{6}$$

with $ab \neq 0$. In the example of Section 2.2, we have that, in fact, $\alpha = \alpha(N, \Delta t)$, but $\beta = \beta(\Delta t)$ and $\gamma = \gamma(\Delta t)$. We shall consider, however, the most general case (6), for completeness. This approach will be useful in the sequel (see Eq. (7)). Moreover, we emphasize that the limits in (6) are to be understood in a pathwise sense. Thus, different paths can lead to different limits. For further information on how the different scalings can lead to quite different dynamics, see [4].

Let $P_{(N,\Delta t)}(t, n, m)$ be the probability that at time t we have n susceptible, m infected and $N - n - m$ removed, where the total population N is constant and the time step is given by $\Delta t > 0$. Therefore

$$\begin{aligned} P_{(N,\Delta t)}(t+\Delta t, n, m) &= \alpha \frac{(n+1)(m-1)}{N(N-1)} P_{(N,\Delta t)}(t, n+1, m-1) \\ &\quad + \beta \frac{m+1}{N} P_{(N,\Delta t)}(t, n, m+1) + \gamma \frac{N-n-m+1}{N} P_{(N,\Delta t)}(t, n-1, m) \\ &\quad + \left[\frac{n}{N}\left(1-\alpha\frac{m}{N-1}\right) + \frac{m}{N}(1-\beta) + \frac{N-n-m}{N}(1-\gamma)\right] P_{(N,\Delta t)}(t, n, m). \end{aligned}$$

Now, define $x = n/N$, $y = m/N$ and $p(t, x, y) = NP_{(N,\Delta t)}(t, xN, yN)$. Then, using $p(t, x, y) = p$ and keeping terms until order $1/N$:

$$\begin{aligned} p(t+\Delta t, x, y) &= \alpha \frac{\left(x+\frac{1}{N}\right)\left(y-\frac{1}{N}\right)}{\left(1-\frac{1}{N}\right)} p\left(t, x+\frac{1}{N}, y-\frac{1}{N}\right) \\ &\quad + \beta\left(y+\frac{1}{N}\right) p\left(t, x, y+\frac{1}{N}\right) + \gamma\left(1-x-y+\frac{1}{N}\right) p\left(t, x-\frac{1}{N}, y\right) \\ &\quad + \left(x\left(1-\frac{\alpha y}{1-\frac{1}{N}}\right) + y(1-\beta) + (1-x-y)(1-\gamma)\right) p(t, x, y) \\ &\approx p + \frac{1}{N}\left[(\alpha(y-x)+\beta+\gamma)\, p + (\alpha xy - \gamma(1-x-y))\, \partial_x p + (\beta y - \alpha xy)\, \partial_y p\right] \\ &= p + \frac{1}{N}\left[\partial_x\left((\alpha xy - \gamma(1-x-y))p\right) + \partial_y\left((\beta - \alpha x) y p\right)\right]. \end{aligned}$$

Finally,

$$\partial_t p = \partial_x\left((axy - c(1-x-y))p\right) + \partial_y\left((b-ax)yp\right) \tag{7}$$

subject to probability conservation, i.e,

$$\frac{\mathrm{d}}{\mathrm{d}t}\int_{\mathcal{S}} p(t, x, y)\mathrm{d}x\mathrm{d}y = 0, \tag{8}$$

where $\mathcal{S} = \{(x, y) \in \mathbb{R}^2 | x, y \geq 0, x + y \leq 1\}$ is the two-dimensional simplex.

Remark 1. Eq. (7) can be seen as a Hyperbolic Kolmogorov Equation. This needs further clarification. Considering the parameters as given, if the fraction of susceptible, infected and removed is known at a given time t, then the dynamics is given at any future time. Nevertheless, if we assume that the measurements are not accurate, and we can only obtain a probability distribution for the population, then (7) *transports* this distribution in time. Moreover, this approach allows inclusion of stochasticity as a perturbation of a higher order differential operator in a natural way. In addition, this shows that simple addition of a parabolic term to the ODE system does not seem to be the correct modeling assumption when trying to model stochastic effects in the system.

3. The SIR(S) model as a transport problem

Let $\mathbf{x} = (x, y)$ and let $\Phi_t(\mathbf{x})$ be the flow map associated to the SIR system

$$\begin{aligned} \dot{X} &= c(1-X-Y) - aXY, \\ \dot{Y} &= (aX - b)Y. \end{aligned}$$

Let $p_0(\mathbf{x}) \in C^1(R^2)$, and let Q satisfies

$$F \cdot \nabla Q = -\nabla \cdot F,$$

where F denotes the right hand side of the SIR system. Let us write

$$p(t, x, y) = \mathrm{e}^{Q(\mathbf{x}) - Q(\Phi_{-t}(\mathbf{x}))} p_0(\Phi_{-t}(\mathbf{x})),$$

with $\mathbf{x} = (x, y)$.

Finally, let $\mathcal{S}$ denote the unit simplex in $\mathbb{R}^2$ and fix $\mathbf{x}_0 \in \mathcal{S}$. Then, we have that

$$\mathrm{e}^{-Q(\phi_t(\mathbf{x}_0))} p(t, \phi_t(\mathbf{x}_0)) = \mathrm{e}^{Q(\mathbf{x}_0)} p_0(\mathbf{x}_0).$$

Hence,

$$\begin{aligned} 0 &= \mathrm{e}^{Q(\phi_t(\mathbf{x}_0))}\frac{\mathrm{d}}{\mathrm{d}t}\left[\mathrm{e}^{-Q(\phi_t(\mathbf{x}_0))}p(t,\phi_t(\mathbf{x}_0))\right] \\ &= -F(\phi_t(\mathbf{x}_0))\cdot\nabla Q(\phi_t(\mathbf{x}_0))p(t,\phi_t(\mathbf{x}_0)) + \partial_t p(t,\phi_t(\mathbf{x}_0)) + F(\phi_t(\mathbf{x}_0))\nabla p(t,\phi_t(\mathbf{x}_0)) \\ &= \nabla\cdot Fp + F\nabla p + \partial_t p \\ &= \partial_t p + \nabla\cdot(pF)\,. \end{aligned}$$

Thus, the SIR system equations are the characteristics of Eq. (7) and the probability density should be transported along them. We now make this calculation more precise.

Definition 1. Let $F : U \subset \mathbb{R}^n \to \mathbb{R}^n$, be a Lipschitz vector field, where U is an open set, and let $\Omega \subset U$ be compact. We say that Ω is regularly attracting for F, if there is an open set V with a piecewise smooth boundary, $\Omega \subset V \subset \bar{V} \subset U$, such that $\omega(V) \subset \bar{\Omega}$, where $\omega(V)$ is the omega-limit of V, obtained from the flow F restricted to V.

Theorem 1. *Let Ω be a domain with piecewise smooth boundary $\partial\Omega$. Let $F : U \subset \mathbb{R}^N \to \mathbb{R}^N$ be Lipschitz, with $\Omega \subset U$ being a regularly attracting set for F. Let $p_0 \in L^1(\Omega)$ be nonnegative. Then the equation*

$$\partial_t p + \nabla\cdot(pF) = 0, \qquad p(0,x) = p_0(x) \tag{9}$$

has a unique solution

$$p(t,\mathbf{x}) = \mathrm{e}^{Q(\mathbf{x})-Q(\Phi_{-t}(\mathbf{x}))}p_0(\Phi_{-t}(\mathbf{x})).$$

Moreover, p is nonnegative and $\mathrm{supp}(p(t,\cdot)) \subset \Omega$, where $\mathrm{supp}(\cdot)$ denotes the support of the function or of the distribution.

In addition, if $\mathrm{supp}(p_0) \subset \Omega$, then

$$\frac{\mathrm{d}}{\mathrm{d}t}\int_\Omega p(t,\mathbf{x})\mathrm{d}\mathbf{x} = 0. \tag{10}$$

Proof. Existence can be shown as follows by considering the weak formulation. Let $W = [0,\infty)\times\Omega$ and let $\psi \in C_c(W)$ be a compact supported test function.

$$\iint_W p(t,\mathbf{x})\partial_t\psi(t,\mathbf{x})\mathrm{d}\mathbf{x}\mathrm{d}t + \iint_W p(t,\mathbf{x})\nabla\psi(t,\mathbf{x})\mathrm{d}\mathbf{x}\mathrm{d}t + \int_\Omega p(0,\mathbf{x})\psi(0,\mathbf{x})\mathrm{d}\mathbf{x}\mathrm{d}t = 0. \tag{11}$$

Choose $\psi \in C_c((0,\infty)\times\Omega)$. Then (11) becomes

$$\iint_W p(t,\mathbf{x})\partial_t\psi(t,\mathbf{x})\mathrm{d}\mathbf{x}\mathrm{d}t + \iint_W p(t,\mathbf{x})\nabla\psi(t,\mathbf{x})\mathrm{d}\mathbf{x}\mathrm{d}t = 0.$$

Let $\mathbf{y} = \phi_t(x)$ and $\eta_t(x) = \det(\partial_{\mathbf{x}}\Phi_t(\mathbf{x}))$, where det means the determinant. Also let $W_t = \Phi_t(W)$. Then, the first integral becomes

$$\iint_{W_t} \mathrm{e}^{Q(\Phi_t(\mathbf{y}))-Q(\mathbf{y})}p_0(\mathbf{y})\partial_t\psi(t,\Phi_t(\mathbf{y}))\eta_t(\mathbf{y})\mathrm{d}\mathbf{y}\mathrm{d}t.$$

The second integral becomes

$$\iint_{W_t} \mathrm{e}^{Q(\Phi_t(\mathbf{y}))-Q(\mathbf{y})}p_0(\mathbf{y})\nabla\cdot\psi(t,\Phi_t(\mathbf{y}))\eta_t(\mathbf{y})\mathrm{d}\mathbf{y}\mathrm{d}t.$$

Combining both integrals, we can write

$$\iint_{W_t} \mathrm{e}^{Q(\Phi_t(\mathbf{y}))-Q(\mathbf{y})}p_0(\mathbf{y})\frac{\mathrm{d}}{\mathrm{d}t}\psi(t,\Phi_t(\mathbf{y}))\eta_t(\mathbf{y})\mathrm{d}\mathbf{y}\mathrm{d}t.$$

On integrating by parts, we have that

$$\iint_{W_t} \mathrm{e}^{-Q(\mathbf{y})}p_0(\mathbf{y})\psi(t,\Phi_t(\mathbf{y}))\mathrm{e}^{Q(\Phi_t(\mathbf{y}))}\left[F\cdot\nabla Q - \nabla\cdot F\right]\mathrm{d}\mathbf{y}\mathrm{d}t = 0.$$

We have that p is clearly nonnegative. Let $V = \mathrm{supp}(u(t,\cdot))$ and let $V_t = (\Phi_{-t}(W))$. Let ψ be a vanishing function in Ω. Then

$$0 = \int_{\Phi_t(\Omega)} u(t,\mathbf{x})\psi(\mathbf{x})\mathrm{d}\mathbf{x} = \int_\Omega \mathrm{e}^{Q(\Phi_t(\mathbf{y})-Q(\mathbf{y}))}u_0(\mathbf{y})\psi(\mathbf{y})\mathrm{div}(F)\mathrm{d}\mathbf{y}.$$

Hence $\mathrm{supp}(u(t,\cdot)) \subset \Phi_t(\Omega)$. Since Ω is regularly attracting for F, we have that $\Phi_t(\Omega) \subset \Omega$. If $\mathrm{supp}(p_0(\mathbf{y})) \subset \Omega$, then we extend p_0 by defining it to be zero in $\mathbb{R}^N - U$. Then we have that

$$\begin{aligned}\frac{\mathrm{d}}{\mathrm{d}t}\int_\Omega u(t,\cdot)\mathrm{d}\mathbf{x} &= \frac{\mathrm{d}}{\mathrm{d}t}\int_V u(t,\cdot)\mathrm{d}\mathbf{x}\\ &= -\int_V \mathrm{div}(u(t,\cdot)\hat{F})\mathrm{d}\mathbf{x} = 0. \quad \square\end{aligned}$$

Let

$$F_S(x,y) = (c(1-x-y) - \alpha xy, \alpha xy - by).$$

We define the reflected SIR field by

$$F_{RS}(x,y) = (c(1-x-y) - axy, (ax-b)|y|).$$

We immediately have

Lemma 1. *Let Ω be the simplex in the nonnegative orthant of R^2. Then F_{RS} is a C^1 vector field in $\mathbb{R}^2$ and Ω is regularly attracting for F_{RS}.*

Thus for smooth solutions, we have

Theorem 2. *Consider the Cauchy problem for (7), with a L^1 non-negative initial condition p_0. Then there exists a unique solution satisfying (8). Moreover, $p(t,x) \geq 0$.*

4. Asymptotic behavior and measure solutions

We recall that the dynamics of SIRS is controlled by the parameter $R_0 := a/b$ (see [1]), i.e,

Proposition 1. *Let $\mathbf{x}_1 = (1,0)$ and $\mathbf{x}_2 = (\frac{1}{R_0}, \frac{c}{c+b}(1-\frac{1}{R_0}))$ be the two equilibria of SIRS. $\mathbf{x}_1$ is referred to as the disease free equilibrium and $\mathbf{x}_2$ as the endemic equilibrium. If $R_0 \leq 1$, then any solution that starts in the nonnegative orthant of $\mathbb{R}^2$ approaches $\mathbf{x}_0$ for large time. If $R_0 > 1$, then $\mathbf{x}_1$ is the limiting point.*

With this point of view, if we use the Wasserstein or the Radon metric to indicate the distance between two Radon measures (cf. [7] or [17]) we have

Theorem 3. *Let p be a solution of (7), satisfying (8). Then, in the Wasserstein metric or in the Radon metric, we have that*

$$\lim_{t\to\infty} p(t,\cdot) = \begin{cases}\delta_{\mathbf{x}_1}, & R_0 \leq 1\\ \delta_{\mathbf{x}_2}, & R_0 > 1.\end{cases}$$

Proof. We deal with the case $R_0 \leq 1$; the case $R_0 > 1$ is analogous. Since $R_0 \leq 1$, we have that $\mathbf{x}_1$ is the globally asymptotic stable equilibrium. Then, given $\delta > 0$, we can find $T > 0$, such that, for $t > T$, we have that

$$\Phi_t(\mathcal{S}) \subset B_\delta(\mathbf{x}_1).$$

Let $\psi(\mathbf{x})$ be a continuous function. Then, for $t > T$, we have that

$$\int_{\mathcal{S}} p(t,\mathbf{x})\psi(\mathbf{x})\mathrm{d}\mathbf{x} = \int_{B_\delta(\mathbf{x}_1)} p(t,\mathbf{x})\psi(\mathbf{x})\mathrm{d}\mathbf{x}.$$

But, let $\epsilon > 0$ be given. Since ψ is continuous, we have $\delta > 0$, such that

$$\psi(\mathbf{x}_1) - \epsilon \leq \int_{B_\delta(\mathbf{x}_1)} p(t,\mathbf{x})\psi(\mathbf{x})\mathrm{d}\mathbf{x} \leq \psi(\mathbf{x}_0) + \epsilon,$$

and this proves the claim, since we have that

$$\int_{B_\epsilon(x_1)} p(t,\Phi_t(\mathbf{y}))\mathrm{d}\mathbf{y} = \int_{B_\epsilon(x_1)} \mathrm{e}^{Q(\Phi_t(\mathbf{y}))-Q(\mathbf{y})} p_0(\mathbf{y})\psi(\Phi_t(\mathbf{y}))\eta_t(\mathbf{y})\mathrm{d}y. \quad \square$$

When $b = 0$, a variation of the same arguments above show the following, weaker, result:

Proposition 2. *Let p be a solution of (7), with $b = 0$ and satisfying (8). Let $p_\infty = \lim_{t\to\infty} p(t,\cdot)$. Then p_∞ is a positive Radon measure supported in $[0,1] \times \{0\}$.*

5. Concluding remarks

Traditional continuous models for epidemics are based on ordinary differential equations. The use of ordinary differential equations for these models implies, implicitly, that the population under study is infinite and, explicitly, that any stochastic effect will not be considered. This has severe modeling consequences: finite population effects are totally ignored. In particular, there are no stochastic effects that might be subtle but have a large impact on the long term dynamics of the model, as shown for the Moran process by Chalub and Souza [4].

Although the method presented here seems to be awkward when compared to known results in the ODE theory, the use of PDE allows the extension of the continuous dynamics to be valid globally in time, i.e., it is possible to obtain a parabolic PDE such that its solution approximates pointwise the solution of the discrete equation for any time. In this work, we showed only the initial dynamics of the full PDE (and then, we used its hyperbolic limit), i.e, we did not consider any diffusion (stochastic) effect.

The results presented here pave the way to a more detailed study of both continuous and discrete SIR(S) models. For the SIS model, such a study is detailed in a forthcoming work. In this way, enhanced continuous models can be derived so as to obtain both quantitatively and qualitatively more accurate approximations of discrete models. This will bring together models that have been grown apart in the mathematical epidemics literature.

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