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SIR EPIDEMIC MODELS WITH SPATIAL SPREAD IN BOUNDED DOMAINS

HELMUT KNOLLE, JAIRO SANTANILLA

Abstract. The spread of an infectious disease which confers immunity after recovery from infection, can be described by a SIR model, i.e. a system of three differential equations for the dependent variables S, I, and R, which are the numbers (densities) of susceptible, infectious and recovered (immune) individuals, respectively. The equations for S and I are typically nonlinear. In this article, we consider two spatio-temporal SIR models. The first model is similar to reaction-diffusion systems in chemistry. A simple birth-and-death process is incorporated, and it is assumed, that a fraction f of the newborns is vaccinated and is then immune for life. We show how the smallest eigenvalue of the eigenvalue problem associated with the linearized equation for I is related to the basic reproduction number $\mathcal{R}_0$, a key concept in the mathematical theory of infectious diseases. Here it is defined by a variational principle. We show that the disease-free equilibrium is asymptotically stable if $\mathcal{R}_0 < 1$, or if $\mathcal{R}_0 \geq 1$ and $f > 1 - 1/\mathcal{R}_0$, and unstable if $\mathcal{R}_0 > 1$ and $f < 1 - 1/\mathcal{R}_0$. In the other model we assume that the population consists of sedentary individuals who leave their home only temporarily. Both models suggest that restriction of mobility may be counterproductive for the control of an epidemic outbreak.

1. Introduction

We consider an infectious disease, which is transmitted directly and confers life-long immunity after recovery from infection. For this type of disease, the local spread of infection in a homogeneously mixing population has been described by the equations,

$$\frac{dS}{dt} = -\beta SI, \quad \frac{dI}{dt} = \beta SI - \gamma I, \quad \frac{dR}{dt} = \gamma I,$$

where t is time, S the number of susceptibles, I the number of infectives, R the number of immunes [2], β and γ are positive constants. The trivial solution $S = N$ =constant, $I = 0$, $R = 0$ is called the *disease-free equilibrium* (DFE). It is asymptotically stable if $N\beta < \gamma$. Otherwise, if $N\beta > \gamma$, a small number of infectives can trigger a large epidemic, but in the long run I tends to zero anyway. Things are different, when fresh susceptibles are introduced by births. Therefore, when the long-term behavior of an infectious disease in a population is considered, a birth-and-death process must be incorporated into the model. If we assume constant

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population size and age-independent birth and death rate, this leads to the model

$$\frac{dS}{dt} = \mu(S+I+R) - \beta SI - \mu S$$
$$\frac{dI}{dt} = \beta SI - \gamma I - \mu I$$
$$\frac{dR}{dt} = \gamma I - \mu R$$

The *basic reproduction number* is defined as $\mathcal{R}_0 = N\beta/(\mu+\gamma)$. The DFE is stable if $\mathcal{R}_0 < 1$, and unstable if $\mathcal{R}_0 > 1$. An *endemic equilibrium* (EE), i.e., an equilibrium with $I > 0$, exists if and only if $\mathcal{R}_0 > 1$.

The question rises, how the theory must be adapted when we are dealing with a population spread over a large area. Then the variables S, I, and R are time and space-dependent, the parameters β and γ are space-dependent, and the assumption of homogeneous mixing is no longer justified, because infection will be more likely between close neighbors than between persons at greater distance. In most cases, a human population consists of sedentary individuals who move away from their place of residence only temporarily to work, study, or participate in social life. Therefore, we introduce a probability distribution h: $\mathbb{R}^2 \to \mathbb{R}_+$ for the probability of an infectious contact in unit time between a person living at place (x, y) and a person living at place $(x+u, y+v)$, and we define

$$\hat{I}(x,y,t) = \iint I(x+u, y+v, t)h(u,v)\,du\,dv\,.$$

Then the equation for I becomes

$$I_t = \beta S\hat{I} - \gamma I - \mu I\,.$$

This integro-differential equation can be approximated by a partial differential equation of parabolic type, if $I(x+u, y+v, t)$ as a function of u and v is expanded as a Taylor series up to second degree. Kendall [8] used this approach in his study of a model with only one spatial variable, and he focused on traveling waves, i.e., solutions of the form $u(x,t) = u(x-ct)$, where c is the speed of propagation of an epidemic in space. Diekmann [5] has studied traveling waves of an epidemic in a more general setting.

In another class of models, it is assumed that the individuals move at random, like the molecules of a gas. This leads to equations similar to the reaction-diffusion equations in chemistry. Allen et al. [1] consider the following spatial model of a SIS disease, which spreads over an open domain Ω in $\mathbb{R}^m$

$$S_t = d_S\Delta S - \beta\frac{SI}{S+I} + \gamma I \quad x \in \Omega,\ t > 0$$
$$I_t = d_I\Delta I + \beta\frac{SI}{S+I} - \gamma I \quad x \in \Omega,\ t > 0$$
$$\frac{\partial S}{\partial n} = \frac{\partial I}{\partial n} = 0 \quad x \in \partial\Omega,\ t > 0\,.$$

The infection mechanism described by the term $\beta SI/(S+I)$ means that the incidence of infection is independent of the density of the population. This may be the case in sexually transmitted diseases (see e.g. Bailey [3]), but not in airborne infections such as Covid 19. Since the nonlinear term is bounded, Allen et al. can

easily prove that for given non-negative initial conditions a unique classical solution exists for all time. Then they prove that a unique DFE exists, and they define

$$\mathcal{R}_0 = \sup\Big\{ \frac{\int \beta\phi^2}{\int [d_I |\operatorname{grad}\phi|^2 + \gamma\phi^2]} : \phi \in H^1(\Omega),\ \phi \neq 0 \Big\}.$$

It turns out, that the DFE is asymptotically stable if $\mathcal{R}_0 < 1$, and unstable if $\mathcal{R}_0 > 1$. Furthermore, a unique EE exists if $\mathcal{R}_0 > 1$. For the same model, Peng [9] studied the properties of equilibria, if one of the diffusion constants or both tend to zero or to infinity. The results show that reducing the mobility of the susceptible individuals can help to eliminate the infection, while reducing the mobility of the infectious individuals cannot.

Wu and Zou [10] considered a similar model, in which the infection mechanism is given by the law of mass action, i.e. the incidence of infection is proportional to SI. This model presents a new challenge due to the unboundedness of the nonlinear term. The results reveal some fundamental differences in the conclusions to be drawn from spatial models with different infection mechanisms.

2. New model

Assumptions. The model in Wu and Zou [10] is extended by the inclusion of an immune state and a simple birth-and-death process. Each person is either susceptible (healthy but susceptible to infection), infectious or immune. Every newborn is susceptible, but a fraction f of newborns become immune through vaccination. The immunity that is developed after vaccination or after surviving the infection is lifelong. Birth and death rates are the same for all three groups, regardless of age. The benefit of the greater complexity of our model, compared with that in [1, 10], outweighs the restrictions we impose on the parameters and the initial values. We assume a uniform diffusion constant for S, I and R, and a space-independent population density at $t = 0$.

Notation.

Ω	a bounded, open connected domain in $\mathbb{R}^2$
ω	area of Ω
$H^1(\Omega)$	Hilbert space of functions u defined in Ω which have square summable first derivatives and satisfy $\partial u/\partial n = 0$, $x \in \partial\Omega$
$S(x,t)$	density of susceptibles at point x at time t
$I(x,t)$	density of infectives at point x at time t
$R(x,t)$	density of immunes at point x at time t
d	constant of diffusion
f	fraction vaccinated ($f < 1$)
μ	birth and death rate
N	population size
$\beta(x)$	product of frequency of contact and infectivity at point x
γ	recovery rate.

The model consists of the following differential equations:

$$S_t = (1-f)\mu(S+I+R) + d\Delta S - \beta SI - \mu S \quad x \in \Omega,\ t > 0\,, \tag{2.1}$$

$$I_t = d\Delta I + \beta SI - \gamma I - \mu I \quad x \in \Omega,\ t > 0\,, \tag{2.2}$$

$$R_t = f\mu(S+I+R) + d\Delta R + \gamma I - \mu R \quad x \in \Omega, t > 0,\,. \tag{2.3}$$

Since S, I, and R are densities, the domain of definition of these equations is the closed non-negative orthant of $\mathbb{R}^3$. We assume throughout that $\beta(x) \geq 0$, $\gamma > 0$, $\mu > 0$, and $d > 0$.

The term βSI in (2.1) and (2.2) describes transmission of the infection according to the law of mass action. The boundary and initial conditions are

$$\frac{\partial S}{\partial n} = \frac{\partial I}{\partial n} = \frac{\partial R}{\partial n} = 0 \quad \text{on } \partial\Omega,\ t > 0, \tag{2.4}$$

$$S(x,0) = S_0(x) \geq 0, \quad I(x,0) = I_0(x) \geq 0, \tag{2.5}$$

$$R(x,0) = R_0(x) \geq 0 \quad x \in \Omega\,. \tag{2.6}$$

Concerning the initial values we assume

$$S_0(x) + I_0(x) + R_0(x) = N/\omega, \tag{2.7}$$

$$\int_\Omega I_0 > 0\,. \tag{2.8}$$

In the following, $\int$ means the integral over Ω. Since the nonlinearity in (2.1) and (2.2) is unbounded, the proof of global existence and positivity is more difficult than for the model of Allen et al. [1]. We will apply a theorem by Fellner, Morgan & Tang [6], in which the following notation is used.

For $u_i : \Omega \times (0,T) \to \mathbb{R}$, $i = 1, 2, \ldots, m$, the i-th density and $u = (u_1, u_2, \ldots, u_m)$, we consider the system

$$\partial_t u_i - d_i \Delta u_i = g_i(x,\ u), \quad (x,t) \in \Omega \times (0,T), \tag{2.9}$$

$$\nabla_x u_i \cdot \nu = 0, \quad (x,t) \in \partial\Omega \times (0,T), \tag{2.10}$$

$$u_i(x,0) = u_{i0} \quad x \in \Omega \tag{2.11}$$

where $d_i > 0$. The domain Ω, the initial data, and the nonlinearities satisfy the following assumptions.

(H1) (Smooth domain) $\Omega \subset \mathbb{R}^n$ is a bounded domain with $\partial\Omega$ of C^n class such that Ω lies locally on one side of $\partial\Omega$.

(H2) (Bounded, nonnegative initial data) For all $i = 1, 2, \ldots, m$, $u_{i0} \in L^\infty(\Omega)$ and $u_{i0}(x) \geq 0$ for a.e. $x \in \Omega$.

(H3) (Mass control) There exist $K_0 \geq 0$ and $K_1 \in \mathbb{R}$ such that

$$\sum_{i=0}^{m} g_i(x,u) \leq K_0 + K_1 \sum_{i=0}^{m} u_i$$

for all $(x,u) \in \overline{\Omega} \times \mathbb{R}^m_+$.

(H4) (Local Lipschitz and quasi-positivity) For all $i = 1, 2, \ldots, m$, $g_i(x, \cdot\ )$ is locally Lipschitz, $g_i(\cdot, u) \in L^\infty(\Omega)$ and $g_i(x,u) \geq 0$ for all $(x,u) \in \overline{\Omega} \times \mathbb{R}^m_+$ satisfying $u_i = 0$.

(H5) (Super-quadratic growth) There exist $\varepsilon > 0$ sufficiently small and $K > 0$ such that $|g_i(x,u)| \leq K(1 + |u|^{2+\varepsilon})$ for all $(x,u) \in \overline{\Omega} \times \mathbb{R}^m$, $i = 1, 2, \ldots, m$.

Proposition 2.1 ([6]). *Under assumptions* (H1)–(H5) *there exists a unique nonnegative global solution u to* (2.9)–(2.11).

Theorem 2.2. *Let $\Omega \subset \mathbb{R}^2$ be a bounded connected domain with $\partial\Omega$ of C^2 class such that Ω lies locally on one side of $\partial\Omega$. Assume that $\beta, S_0, I_0, R_0 \in L^\infty(\Omega)$, and* (2.8) *are satisfied. Then there exists a unique global nonnegative-nontrivial solution of* (2.1)–(2.5).

Proof. Corresponding to the right-hand side of (2.1)–(2.3), we let $u = (u_1, u_2, u_3)$ and

$$g_1(x, u_1, u_2, u_3) = (1-f)\mu(u_1+u_2+u_3) - \beta(x)u_1u_2 - \mu u_1\,,$$
$$g_2(x, u_1, u_2, u_3) = \beta(x)u_1u_2 - \gamma u_2 - \mu u_2\,,$$
$$g_3(x, u_1, u_2, u_3) = f\mu(u_1+u_2+u_3) + \gamma u_2 - \mu u_3\,,$$

for $(u_1, u_2, u_3) \in \mathbb{R}^3$ and $x \in \overline{\Omega}$.

Assumptions (H1) and (H2) are obviously satisfied. Assumption (H3) is satisfied with $K_0 \geq 0$ and $K_1 = 0$, since

$$\sum_{i=0}^{3} g_i(x,u) = 0 \quad \text{for all } (x,u) \in \overline{\Omega} \times \mathbb{R}^3_+\,.$$

Since $\beta \in L^\infty(\Omega)$, it follows that $g_i(\cdot\,,\ u) \in L^\infty(\Omega)$. It is also clear that each $g_i(x,\cdot)$ is locally Lipschitz. Furthermore,

$$g_i(x,u) \geq 0 \quad \text{for all } (x,u) \in \overline{\Omega} \times \mathbb{R}^3_+ \text{ satisfying } u_i = 0.$$

Hence assumption (H4) is satisfied. Finally, we show that (H5) is satisfied.

(A) We claim that there exists $c_1 > 0$ such that $|g_1(x,u)| \leq c_1(1+|u|^{2+\varepsilon})$.

$$\begin{aligned} |g_1(x,\ u_1, u_2,\ u_3)| &= |-f\mu u_1 + (1-f)\mu(u_2+u_3) - \beta(x)u_1u_2| \\ &\leq f\mu|u_1| + \beta(x)|u_1||u_2| + (1-f)\mu(|u_2|+|u_3|) \\ &\leq a_1(|u_1| + |u_1||u_2| + |u_2| + |u_3|), \end{aligned}$$

where $a_1 = \max\{f\mu, \|\beta\|_\infty, (1-f)\mu\}$.

Let $v = \max\{|u_1|,\ |u_2|,\ |u_3|\}$ and assume $v > 1$. Then

$$|g_1(x,u)| \leq 3a_1(v+v^2) \leq 6a_1v^2 \leq 6a_1(1+|u|^{2+\varepsilon})(\varepsilon > 0).$$

Suppose $|u_1| \leq 1$, $|u_2| \leq 1$, $|u_3| \leq 1$, then

$$|g_1(x,\ u)| \leq a_1(|u_1| + |u_1||u_2| + |u_2| + |u_3|) \leq 4a_1 \leq 6a_1(1+|u|^{2+\varepsilon}).$$

Then item (A) follows with $c_1 = 6a_1$.

(B) As in item (A) with $a_2 = \max\{\|\beta\|_\infty, \gamma+\mu\}$, there exits $c_2 > 0$ such that

$$|g_2(x,\ u)| \leq c_2(1+|u|^{2+\varepsilon}).$$

(C] As above with $a_3 = \max\{\mu, f\mu+\gamma\}$, there exits $c_3 > 0$ such that

$$|g_3(x,u)| \leq c_3(1+|u|^{2+\varepsilon}).$$

Finally (H5) is satisfied with $K = \max\{c_1, c_2, c_3\}$. The fact that the solution is nontrivial, follows from assumption (2.8). □

It also follows from our assumptions and the above proposition that S, I, and R are C^1 as functions of t, and C^2 as functions of x.

Theorem 2.3. *Every solution of* (2.1)–(2.7) *satisfies*

$$S + I + R = N/\omega, \quad x \in \Omega,\ t > 0 \tag{2.12}$$

Proof. We define $u := S + I + R$. Then u satisfies (2.4) and the initial condition $u(x,0) = N/\omega$, $x \in \Omega$. By adding the three differential equations we obtain

$$u_t = S_t + I_t + R_t = d\Delta(S+I+R) = d\Delta u.$$

The constant N/ω satisfies this equation, as well as the boundary and initial conditions.

Since the solution of this problem is unique, the solution (S, I, R) of (2.1)–(2.7) satisfies equation (2.12). □

Theorem 2.4. *There is a unique equilibrium without infection,* $(S_{\rm eq}, 0, R_{\rm eq})$*, with*

$$S_{\rm eq} = (1-f)N/\omega \quad \text{and} \quad R_{\rm eq} = fN/\omega.$$

In the proof we will use the following result.

Lemma 2.5. *The boundary value problem*

$$d\Delta u - cu = 0 \quad x \in \Omega, \quad (d > 0,\ c > 0)$$
$$\frac{\partial u}{\partial n} = 0 \quad x \in \partial\Omega$$

has only the solution $u \equiv 0$.

The proof follows immediately after multiplying the equation by u and integrating over Ω. By Green's formula and the boundary condition, we have

$$0 = -\int d|\operatorname{grad} u|^2\, dx - \int cu^2 dx \le -\int cu^2\, dx$$

which implies $u \equiv 0$.

Proof of Theorem 2.4. With $I \equiv 0$ and $S_t = R_t = 0$ we have

$$(1-f)\mu(S+R) + d\Delta S - \mu S = 0 \quad x \in \Omega, \tag{2.13}$$
$$f\mu(S+R) + d\Delta R - \mu R = 0 \quad x \in \Omega\,. \tag{2.14}$$

Obviously, $(S_{\rm eq}, R_{\rm eq})$ is a solution of (2.13)–(2.14) and (2.4). Equations (2.13)–(2.14) can be written as

$$d\Delta S - f\mu S = -(1-f)\mu R,$$
$$d\Delta R - (1-f)\mu R = -f\mu S\,.$$

These nonhomogeneous linear equations with the boundary conditions (2.4) have at most one solution, because the homogeneous equations $d\Delta S - f\mu S = 0$ and $d\Delta R - (1-f)\mu R = 0$ with boundary condition (2.4) have only the trivial solution. Hence $(S_{\rm eq}, 0, R_{\rm eq})$, is the only equilibrium. □

3. Stability and instability of the disease-free equilibrium

To study the stability of the equilibrium without infection, following Allen et al. [1], we define

$$\mathcal{R}_0 = \sup\Big\{ \frac{N/\omega \int \beta\phi^2}{\int [d|\operatorname{grad}\phi|^2 + (\gamma+\mu)\phi^2]} : \phi \in H^1(\Omega),\ \phi \ne 0 \Big\}. \tag{3.1}$$

It turns out that stability of the DFE depends on $\mathcal{R}_0$ and f. Furthermore, an endemic equilibrium (EE), i.e. an equilibrium, in which $\int I^2 > 0$, cannot exist, if $\mathcal{R}_0 < 1$. Indeed, when $\mathcal{R}_0 < 1$ and $\int I^2 > 0$ with $I \in H^1(\Omega)$, applying Green's formula and $(1d)$, we have

$$0 = \int Id\Delta I - \int (\gamma+\mu)I^2 + \int \beta S I^2$$

$$\leq -\int d\,\mathrm{grad}\, I|^2 - \int (\gamma+\mu)I^2 + N/\omega \int \beta I^2 < 0\,,$$

a contradiction. Hence there is no EE if $\mathcal{R}_0 < 1$.

The following classical theorem from calculus of variations [4] is the cornerstone of our stability analysis.

Theorem 3.1. *The smallest eigenvalue of the problem*

$$\begin{aligned} d\Delta u - qu + \lambda p u = 0, \quad &\text{in } \Omega,\\ \frac{\partial u}{\partial n} = 0 \quad &\text{on } \partial\Omega, \end{aligned} \tag{3.2}$$

where $d > 0$ is a constant, $q, p \in C(\Omega)$, and $q, p > 0$, is

$$\lambda_{\min} = \inf\Big\{ \frac{\int [d|\,\mathrm{grad}\, u|^2 + qu^2]}{\int pu^2} : u \in H^1(\Omega),\ u \neq 0 \Big\} \tag{3.3}$$

and the associated eigenfunction can be chosen to be positive.

Corollary 3.2. *There exists a $u \in H^1(\Omega)$, $u(x) > 0$ for $x \in \Omega$ such that*

$$\begin{aligned} d\Delta u - (\gamma+\mu)u + \frac{1}{\mathcal{R}_0}(\beta N/\omega)u = 0, \quad &x \in \Omega\\ \frac{\partial u}{\partial n} = 0 \quad &\text{on } \partial\Omega\,. \end{aligned} \tag{3.4}$$

Proof. In (3.2) we set $q = \gamma + \mu$, $p(x) = \beta(x)N/\omega$. Then the fraction in (3.3) is the reciprocal of the fraction in the definition of $\mathcal{R}_0$ (see (3.1)). Since the supremum of a fraction is equal to the infimum of its reciprocal, we have $\lambda_{\min} = 1/\mathcal{R}_0$. Since this is the smallest eigenvalue of (3.2), there is a positive function (an eigenfunction for $1/\mathcal{R}_0$), that satisfies (3.4). □

Theorem 3.3. *Let λ^* be the smallest eigenvalue of the problem*

$$\begin{aligned} d\Delta u - (\gamma+\mu)u + (1-f)(\beta N/\omega)u + \lambda u = 0, \quad &x \in \Omega\\ \frac{\partial u}{\partial n} = 0 \quad &\text{on } \partial\Omega \end{aligned} \tag{3.5}$$

Then $\lambda^* < 0$ if $(1-f)\mathcal{R}_0 > 1$, and $\lambda^* > 0$ if $(1-f)\mathcal{R}_0 < 1$.

Proof. Let w be a positive eigenfunction to the eigenvalue λ^* of (3.5). Then

$$\begin{aligned} d\Delta w - (\gamma+\mu)w + (1-f)(\beta N/\omega)w + \lambda^* w = 0, \quad &x \in \Omega\\ \frac{\partial w}{\partial n} = 0 \quad &\text{on } \partial\Omega \end{aligned} \tag{3.6}$$

Multiplying (3.4) by w and (3.6) by u, integrating over Ω, and subtracting, the boundary conditions and Green's formula lead to $\int d(w\Delta u - u\Delta w) = 0$. It follows that

$$(1 - f - 1/\mathcal{R}_0)\int (\beta N/\omega)uw + \lambda^* \int uw = 0.$$

Since both integrals are positive, $(1-f)\mathcal{R}_0 - 1$ and λ^* must have opposite signs. (The idea of this proof is due to Allen et al. [1]). □

In the following theorem we assume that at $t = 0$ the density of immunes is equal to the density in equilibrium. This is realistic, because in the disease-free equilibrium all immune individuals are immune by vaccination, and R can become greater than R_{eq} only after infection and recovery of unvaccinated individuals.

Theorem 3.4. *Assume that $R(x,0) = R_{\rm eq}$ in Ω, $f > 1 - 1/\mathcal{R}_0$ if $\mathcal{R}_0 \geq 1$, and $f = 0$ if $\mathcal{R}_0 < 1$. Then the DFE is asymptotically stable.*

Proof. With $\sigma := S - S_{\rm eq}$, $\rho := R - R_{\rm eq}$, equations (2.1)–(2.3) take the form

$$\sigma_t = d\Delta\sigma - f\mu\sigma + (1-f)\mu(I+\rho) - \beta S_{\rm eq} I - \beta\sigma I \quad x \in \Omega,\ t > 0 \tag{3.7}$$

$$I_t = d\Delta I - (\gamma+\mu)I + \beta S_{\rm eq} I + \beta\sigma I \quad x \in \Omega,\ t > 0 \tag{3.8}$$

$$\rho_t = d\Delta\rho - (1-f)\mu\rho + f\mu(\sigma + I) + \gamma I \quad x \in \Omega,\ t > 0 \tag{3.9}$$

Since $S + I + R = N/\omega$, we have $\sigma + I + \rho = 0$, and thus σ can be eliminated from (3.8) and (3.9), and ρ from (3.7). This yields

$$\sigma_t = d\Delta\sigma - \mu\sigma - \beta S_{\rm eq} I - \beta\sigma I \quad x \in \Omega,\ t > 0 \tag{3.10}$$

$$I_t = d\Delta I - (\gamma+\mu)I + \beta S_{\rm eq} I - \beta(\rho + I)I \quad x \in \Omega,\ t > 0 \tag{3.11}$$

$$\rho_t = d\Delta\rho - \mu\rho + \gamma I \quad x \in \Omega,\ t > 0 \tag{3.12}$$

Stability of the disease-free equilibrium depends essentially on equation (3.11). The linear part of (3.11) can be written as $I_t = (d\Delta - B)I$ where $B = (\gamma+\mu) - \beta S_{\rm eq}$. The nonlinear part is $-\beta(\rho + I)I$. We will show that $\beta(\rho + I)I$ is $o(I)$. Let

$$g(t) = \int_\Omega \rho(x,t)dx$$

After integrating equation (3.12) over Ω, using Green's formula, and the boundary condition, we have

$$g'(t) = -\mu g(t) + \gamma \int_\Omega I(x,t)dx\,.$$

From $\rho(x,0) = 0$ for $x \in \Omega$, it follows that $g(0) = 0$. Since I is bounded,

$$g'(t) + \mu g(t) \leq \gamma \sup_{t>0} \int_\Omega I(x,t)dx.$$

Then

$$\begin{aligned} g(t) &\leq \gamma\Big(\sup_{t>0}\int_\Omega I(x,t)\,dx\Big)e^{-\mu t}\int_0^t e^{\mu s}\,ds \\ &= \gamma\Big(\sup_{t>0}\int_\Omega I(x,t)\,dx\Big)\int_0^t e^{\mu(s-t)}\,ds \\ &\leq \frac{\gamma \sup_{t>0}\int_\Omega I(x,t)dx}{\mu}\,. \end{aligned}$$

Therefore, ρI is $o(I)$.

Now we consider the eigenvalues of the linear problem $d\Delta I - BI + \lambda I = 0$ with boundary condition (2.4). By our assumption and Theorem 3.3, the smallest eigenvalue is positive. Therefore, the equilibrium $I = 0$ of the linear approximation is uniformly asymptotically stable, and now Henry's result [7, Theorem 5.1.1] implies that the equilibrium of the nonlinear equation is uniformly asymptotically stable. Finally, we conclude that σ also approaches zero as $t \to \infty$, since $\rho + I + \sigma = 0$. So, we have $S \to S_{\rm eq}$, $I \to 0$, and $R \to R_{\rm eq}$; i.e., the DFE is asymptotically stable. □

Theorem 3.5. *Assume that $\mathcal{R}_0 > 1$ and $f < 1 - (1/\mathcal{R}_0)$. Then the equilibrium $(S_{\rm eq}, 0, R_{\rm eq})$ is unstable.*

Proof. The assumptions and Theorem 3.3 imply that the smallest eigenvalue of the problem

$$d\Delta u - (\gamma+\mu)u + (1-f)(N/\omega)\beta u + \lambda u = 0 \quad x \in \Omega$$
$$\frac{\partial u}{\partial n} = 0 \quad \text{on } \partial\Omega$$

is negative. Therefore, the equilibrium $I = 0$ of the linear approximation of equation (3.11) is unstable. Furthermore, this equation is quadratic in I. It follows from Henry's result [7, Corollary 5.1.6], that the equilibrium $I = 0$ of the nonlinear equation (3.11) is unstable. This implies that the equilibrium $(S_{\rm eq}, 0, R_{\rm eq})$ of system (2.1)–(2.4) is unstable. □

4. Kendall's model extended

Now we return to Kendall's approach as sketched in the Introduction, but we avoid the assumption that β is constant, and allow β to depend on the place where a susceptible and an infective meet. Again Ω is a bounded domain in the plain with smooth boundary, but now its points are denoted by (x, y). For the probability distribution h we assume that it is zero outside a circle with radius r and center $(0,\ 0)$. This means that movements of individuals are restricted to a circle $C(r)$ with radius r. For our analysis it is necessary to assume certain symmetries of h, namely

$$h(u, v) = h(v, u) = h(-u, v) = h(u, -v).$$

This poses no problem at points with a distance greater than r from the boundary of Ω. But near the boundary movements of persons are usually one-sided. This inconsistency can be eliminated when we assume that population density tends to zero near the boundary.

The infection parameter for a pair (S, I), where S is living at (x, y) and I is living at $(x + u, y + v)$, has a value somewhere between $\beta(x, y)$ and $\beta(x + u, y + v)$, say $(1-\lambda)\beta(x, y)+\lambda\beta(x+u, y+u)$ with $0 \le \lambda \le 1$. This includes different patterns of mobility. If $\lambda = 0$, then the infection occurs always at (x, y), i.e. the susceptibles are immobile. If $\lambda = 1$, the infectives are immobile, but a susceptible can get the infection when the susceptible meets an infective at $(x+u, y+v)$. For $\lambda = 1/2$ there is no difference in mobility. With this weighted mean of the infection parameter, the SIR model without birth-and-death process takes the form

$$\begin{aligned} S_t &= -S(x, y, t) \iint_{C(r)} I(x + u, y + v, t)\big[(1 - \lambda)\beta(x, y) \\ &\quad + \lambda\beta(x + u, y + v)\big]h(u, v)\, du\, dv \\ I_t &= -S_t - \gamma I \\ R_t &= \gamma I\,. \end{aligned} \tag{4.1}$$

Expanding the Taylor series at (x, y) up to terms of second degree and using the symmetries, we obtain the approximation

$$S_t = -\frac{1}{2}S[\sigma(\beta\Delta I + 2\lambda(\operatorname{grad} I)(\operatorname{grad}\beta)) + (\lambda\sigma\Delta\beta + 2\beta)I] \tag{4.2}$$
$$I_t = -S_t - \gamma I \tag{4.3}$$
$$R_t = \gamma I, \tag{4.4}$$

where

$$\sigma = \iint_{C(r)} u^2 h(u,v)\, du\, dv$$

which is the basic measure of mobility. The pair of equations (4.2)–(4.3) is strongly nonlinear. Therefore we cannot apply the same methods as in the preceding sections, and we assume without proof that solutions exist and are nonnegative if initial conditions are so.

Since $(S+I+R)_t = 0$, the population density at each location is constant in time but may be varying in space. Let $\tilde{A} = \max_{(x,y)\in\Omega}[S(x,y,0) + I(x,y,0) + R(x,y,0)]$. The following theorem is the analogue of Theorem 3.4.

Theorem 4.1. *If $0 < [(\lambda\sigma\Delta\beta)/2 + \beta]\tilde{A} < \gamma$ in Ω, then the disease-free equilibrium is stable.*

Proof. For any $\varepsilon > 0$, $t_1 > 0$ and all (x,y) in Ω it is impossible that $I(x,y,t) \geq \varepsilon$ for all $t > t_1$, because $R(x,y,t)$ is bounded. Therefore $\lim_{t\to\infty} I(x,y,t) = 0$ in Ω, and $I(x,y,t)$ is monotone decreasing for all (x,y) in Ω or it attains a positive maximum at some $t > 0$ and (x,y) in Ω. This would imply $I_t = 0$, $\operatorname{grad} I = 0$, $\Delta I \leq 0$, hence

$$0 \leq [(\lambda\sigma\Delta\beta)/2 + \beta]SI - \gamma I < [((\lambda\sigma\Delta\beta)/2 + \beta)\tilde{A} - \gamma]I < \ 0,$$

a contradiction. It follows that $I(x,y,t)$ is monotone decreasing in t for all $(x,\ y)$ in Ω. □

Discussion. The relevance of our first model to practice is limited because the way infectious diseases spread has been very much modified by modern life. The analogy with the diffusion of a gas is unrealistic when, as in many countries today, there are different types of mobility with very different speeds and ranges. The assumptions of the model are most likely to be fulfilled in small towns and rural areas where there is no mass transport and in holiday resorts where people only move on foot.

It seems paradoxical that, when γ is not constant, the new definition of $\mathcal{R}_0$ implies that it is a decreasing function of the diffusion constant d. But it is plausible if there is a subarea, in which $\beta N/\omega < \gamma + \mu$, but

$$(N/\omega)\int_\Omega \beta > \int_\Omega (\gamma + \mu).$$

The infectious individuals who drift into this subarea generate fewer infections there than in the rest of the area where $\beta N/\omega > \gamma + \mu$. Therefore $\mathcal{R}_0$ is reduced by high mobility of the population.

Of course, this explanation of the paradox is vague. More information is given by Theorem 4.1, which is based on Kendall's approach. It shows that stability of the disease-free equilibrium depends also on the shape of the function β, in the following sense:

- If β is constant, mobility has no influence on stability of the DFE.
- If β is concave ($\Delta\beta > 0$) and $\lambda > 0$, high mobility increases the risk of an epidemic outbreak.
- If β is convex ($\Delta\beta < 0$) and $\lambda > 0$, mobility can help to prevent an epidemic.

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Helmut Knolle

Scheyenholzstrasse 9, 3075 Rüfenacht, Switzerland.

Institute Of Social And Preventive Medicine, University Of Berne

Email address: helmut.knolle@bluewin.ch

Jairo Santanilla

Department of Mathematics, University of New Orleans, 2000 Lakeshore Dr., New Orleans, LA 70148, USA

Email address: jsantani@uno.edu