Available online at www.sciencedirect.com
SCIENCE DIRECT®
Physics Letters A 345 (2005) 129–136
PHYSICS LETTERS A
www.elsevier.com/locate/pla

On a model of spatial spread of epidemics with long-distance travel

Konstantin B. Blyuss

Department of Mathematical Sciences, University of Exeter, Exeter EX4 4QE, UK

Received 1 July 2004; received in revised form 29 June 2005; accepted 1 July 2005

Available online 11 July 2005

Communicated by A.P. Fordy

Abstract

This Letter studies the dynamics of infectious diseases which are spread geographically through long-distance travel between two regions and subsequent local redistribution through a process of diffusion. A particular case of an equiproportionate travel is considered, and the model describes migration rather than short visits. We examine uniform and nonuniform steady states together with their linear stability. Numerical simulations are performed to illustrate the evolution of initial distribution of population towards its final stage, which is represented by uniform distribution of the total population among infected individuals.

PACS: 87.19.Xx; 87.23.Ge

Keywords: Epidemic spread; Spatial diffusion

1. Introduction

In modelling a spread of infectious diseases a significant role is played by the geographic motion of individuals. Several approaches have been developed to incorporate this important feature in the analysis. One of them assumes individuals to move randomly, and therefore models their spatial distribution in terms of Fickian diffusion, i.e., when the population flux is proportional to the concentration gradient [1]. Under this assumption, diffusion is approximated by adding Laplacian diffusion terms to the ODEs that model the temporal dynamics [2–6]. In this framework each subpopulation has to be considered separately in order to be properly represented by the diffusion model [3].

Diffusion-type models are usually applied when the spread of disease is mainly characterized by local contacts between individuals. At the same time, it can be advantageous to allow for nonlocal diffusion, which can lead to contacts between susceptible individuals at one place with infected individuals at another [7–9]. This mechanism is usually represented by contact functionals, which have the form of integral operators with kernels describing the probabilities of motion between different points in the domain [6,10].

E-mail address: blyuss@maths.ex.ac.uk (K.B. Blyuss).

0375-9601/$ – see front matter
doi:10.1016/j.physleta.2005.07.004

A review of the diffusion processes as described by the formalism of contact distributions can be found in the recent work by Rass and Radcliffe [11].

Recently, additional attention has been paid to models which include populations residing in more than one region with the possibility of travel between the regions [12,13]. These models incorporate the process of mobility of individuals explicitly, thus avoiding the necessity to justify the Markovian character of mobility. It is worth noting several works devoted to stochastic modelling of the spread of epidemics in social networks as well as in worldwide transport networks (see [14–16] and references therein). These results are particularly important in view of recent outbreak of severe acute respiratory disease (SARS) and its global spread [17,18]. Still, these models do not take into account spatial structure of the populations.

In this Letter we propose a basic model which incorporates mobility of individuals between two regions and their redistribution inside those regions. Travel between the regions is governed by integral operators describing the probability of travel between two particular locations in two regions. Local distribution of individuals inside their respective regions is described by the diffusion operators. Our model considers migration rather than short-visit travel, which means that after travel to another region individuals are assumed to reside there and are considered as a part of population in that region. We will study equilibria of the model and their stability, as well as perform direct numerical simulations of the model. In particular, we shall consider a case when the model admits only *uniform* steady states of which one is the unstable uninfected state and the other is a stable infected state.

2. Model derivation

Consider the population residing in two one-dimensional regions, namely, Ω_1 and Ω_2 of sizes L_1 and L_2, respectively. We divide the populations into three classes of susceptible, infected and recovered, with spatial densities being $\mathbf{S}_i(x_i,t)$, $\mathbf{I}_i(x_i,t)$ and $\mathbf{R}_i(x_i,t)$ correspondingly, in a region i, $i=1,2$. Densities of the total population in the two regions are $N_1(x_1,t)$ and $N_2(x_2,t)$. The total population in both areas is $TP(t)=\int_{\Omega_1} N_1(x_1,t)\,dx_1+\int_{\Omega_2} N_2(x_2,t)\,dx_2$.

If one assumes that the transmission of infection occurs in a close contact (i.e., a susceptible can acquire infection only after a contact with some infected individual located at the same point) and there is no latency period, then a general model for the spatial spread of epidemics in two populations can be written in the form

$$\begin{aligned}
\frac{\partial \mathbf{S}_1}{\partial t} &= f^S(\mathbf{S}_1,\mathbf{I}_1)+\big(K_1^S*\mathbf{S}_1\big)-\mathbf{S}_1\big(K_{12}^S*1\big)\\
&\quad+\big(K_{21}^S*\mathbf{S}_2\big),\\
\frac{\partial \mathbf{I}_1}{\partial t} &= f^I(\mathbf{S}_1,\mathbf{I}_1)-r\mathbf{I}_1+\big(K_1^I*\mathbf{I}_1\big)-\mathbf{I}_1\big(K_{12}^I*1\big)\\
&\quad+\big(K_{21}^I*\mathbf{I}_2\big),\\
\frac{\partial \mathbf{R}_1}{\partial t} &= r\mathbf{I}_1+\big(K_1^R*\mathbf{R}_1\big)-\mathbf{R}_1\big(K_{12}^R*1\big)\\
&\quad+\big(K_{21}^R*\mathbf{R}_2\big),\\
\frac{\partial \mathbf{S}_2}{\partial t} &= f^S(\mathbf{S}_2,\mathbf{I}_2)+\big(K_2^S*\mathbf{S}_2\big)-\mathbf{S}_2\big(K_{21}^S*1\big)\\
&\quad+\big(K_{12}^S*\mathbf{S}_1\big),\\
\frac{\partial \mathbf{I}_2}{\partial t} &= f^I(\mathbf{S}_2,\mathbf{I}_2)-r\mathbf{I}_2+\big(K_2^I*\mathbf{I}_2\big)-\mathbf{I}_2\big(K_{21}^I*1\big)\\
&\quad+\big(K_{12}^I*\mathbf{I}_1\big),\\
\frac{\partial \mathbf{R}_2}{\partial t} &= r\mathbf{I}_2+\big(K_2^R*\mathbf{R}_2\big)-\mathbf{R}_2\big(K_{21}^R*1\big)\\
&\quad+\big(K_{12}^R*\mathbf{R}_1\big). \qquad (1)
\end{aligned}$$

Here $\mathbf{S}_i$, $\mathbf{I}_i$ and $\mathbf{R}_i$ are the vectors of susceptible, infected and recovered individuals, respectively, in both regions. In this model it is assumed that the disease confers permanent immunity, i.e., after individuals recover, they move to the class $\mathbf{R}_i$ and never become susceptible again. Recovery rate satisfies $r\geqslant 0$. The cases of cross-immunity against different strains and temporary immunity can be easily incorporated in the model. The functions f^S and f^I represent the dynamics of a disease transmission and include information about the disease transmission rate, patterns of social behaviour, etc.

Spatial dynamics of the disease spread is governed by local and nonlocal diffusion terms. Inside the regions a diffusion of individuals is described by the convolution terms $K_{1,2}^{S,I,R}$ as

$$\big(K_i^S*\mathbf{S}_i\big)(x,t)=\int\limits_{\Omega_i} K_i^S(x_i,x)\mathbf{S}_i(x_i)\,dx_i,$$

$$\begin{aligned}
\big(K_i^I * \mathbf{I}_i\big)(x,t) &= \int\limits_{\Omega_i} K_i^I(x_i,x)\mathbf{I}_i(x_i)\,dx_i,\\
\big(K_i^R * \mathbf{R}_i\big)(x,t) &= \int\limits_{\Omega_i} K_i^R(x_i,x)\mathbf{R}_i(x_i)\,dx_i, \quad i=1,2.
\end{aligned} \tag{2}$$

We assume that there is no long-distance travel inside each region, and the population is spread through the local diffusion only. This assumption is represented formally by choosing the kernels $K_i^{S,I,R}(x_i,x)$ to be of the form

$$\begin{aligned}
&K_i^{S,I,R}(x_i,x) = D_i^{S,I,R}\delta_{xx}(x-x_i),\\
&D_i^{S,I,R} \geqslant 0,
\end{aligned} \tag{3}$$

where $\delta(x)$ is the Dirac delta-function, and $D_i^{S,I,R}$ are diffusion coefficient of susceptible, infected and recovered individuals in each region. After substitution into (2) this gives

$$\begin{aligned}
&\big(K_i^S * \mathbf{S}_i\big) = D_i^S\Delta\mathbf{S}_i, \qquad \big(K_i^I * \mathbf{I}_i\big) = D_i^I\Delta\mathbf{I}_i,\\
&\big(K_i^R * \mathbf{R}_i\big) = D_i^R\Delta\mathbf{R}_i.
\end{aligned} \tag{4}$$

For the sake of generality we allow for all diffusion coefficients to be different, even though in concrete examples it possible to simplify consideration by taking, for instance, $D_1^S = D_2^S = D_1^R = D_2^R$ and $D_1^I = D_2^I$.

In a similar way, $K_{12}^{S,I,R}$ and $K_{21}^{S,I,R}$ describe the proportion of individuals travelling from region 1 to region 2 (and from 2 to 1, respectively):

$$\begin{aligned}
\big(K_{12}^{S,I,R} * g\big)(x_2,t) &= \int\limits_{\Omega_1} K_{12}^{S,I,R}(x_1,x_2)g(x_1)\,dx_1,\\
\big(K_{21}^{S,I,R} * g\big)(x_1,t) &= \int\limits_{\Omega_2} K_{21}^{S,I,R}(x_2,x_1)g(x_2)\,dx_2,
\end{aligned} \tag{5}$$

for any smooth vector-function g. Next, one can assume that the travel to another region happens in such a way that an equal proportion of the population at a particular location travels to a different region, where it is uniformly redistributed along the region. Let us denote this constant proportion of travelling individuals as μ and take μ to be the same constant for both regions. It is natural to assume that the symptoms of infectious disease such as high fever, nausea, etc., can decrease the mobility of infected individuals by a constant $0 \leqslant \sigma \leqslant 1$. At the same time, recovered individuals are able to perform travels to a different region in exactly the same way as the susceptible individuals do. With these assumptions one obtains

$$\begin{aligned}
&\big(K_{12}^S * g\big)(x_2,t) = \frac{\mu}{L_2}\int\limits_{\Omega_1} g(x_1)\,dx_1,\\
&\big(K_{21}^S * g\big)(x_1,t) = \frac{\mu}{L_1}\int\limits_{\Omega_2} g(x_2)\,dx_2,\\
&K_{12}^I = \sigma K_{12}^S, \qquad K_{21}^I = \sigma K_{21}^S,\\
&K_{12}^R = K_{12}^S, \qquad K_{21}^R = K_{21}^S.
\end{aligned} \tag{6}$$

We consider the model with no vital dynamics, i.e., without births or deaths, either natural of from a disease. For the sake of simplicity, the populations of susceptible, infected and recovered are represented by single scalar variables. We take $f^I(S,I) = -f^S(S,I) = -f(S,I)$, that is all individuals who become infected are also infectious and therefore are removed from the class of susceptible to the class of infected. Concerning functional dependence of $f(S,I)$ on its arguments we only assume

$$\begin{aligned}
&f(S,I) > 0 \quad \text{for } S>0, I>0,\\
&f(0,I) = 0 \quad \text{for } I \geqslant 0,\\
&f(S,0) = 0 \quad \text{for } S \geqslant 0,\\
&\frac{\partial f}{\partial S}(S,0) = 0 \quad \text{for } I \geqslant 0,\\
&\frac{\partial f}{\partial I}(0,I) = 0 \quad \text{for } S \geqslant 0,\\
&\frac{\partial f}{\partial S}(0,I) > 0 \quad \text{for } I > 0,\\
&\frac{\partial f}{\partial I}(S,0) > 0 \quad \text{for } S > 0.
\end{aligned} \tag{7}$$

Example of $f(S,I)$ include, among others, a standard bilinear incidence rate $f(S,I) = \beta SI, \beta > 0$ used in the classical Kermack–McKendrick model [1], its nonlinear generalizations $f(S,I) = \beta S^p I^q$, $p \geqslant 0, q \geqslant 0$, which allow one to take into account multiple exposures prior to infection [19,20], and a saturated case $f(S,I) = \alpha SI^k/(1+I^k), \alpha > 0, k > 0$, used to model cholera epidemics and avoid unbounded transmission rates [21].

Rescaling the variables as $x_1 = x$ and $x_2 = xL_1/L_2$, and introducing a domain $\Omega = [0,L]$, where

$L = L_1$, one can finally rewrite the system (1) as

$$\begin{cases} \frac{\partial S_1}{\partial t} = D_1^S \frac{\partial^2 S_1}{\partial x^2} - f(S_1, I_1) - \mu S_1 + \frac{\mu}{L}\int_\Omega S_2\,dx, \\ \frac{\partial I_1}{\partial t} = D_1^I \frac{\partial^2 I_1}{\partial x^2} + f(S_1, I_1) - rI_1 - \mu\sigma I_1 \\ \qquad + \frac{\mu\sigma}{L}\int_\Omega I_2\,dx, \\ \frac{\partial R_1}{\partial t} = D_1^R \frac{\partial^2 R_1}{\partial x^2} + rI_1 - \mu R_1 + \frac{\mu}{L}\int_\Omega R_2\,dx, \\ \frac{\partial S_2}{\partial t} = D_2^S \frac{\partial^2 S_2}{\partial x^2} - f(S_2, I_2) - \mu S_2 + \frac{\mu}{L}\int_\Omega S_1\,dx, \\ \frac{\partial I_2}{\partial t} = D_2^I \frac{\partial^2 I_2}{\partial x^2} + f(S_2, I_2) - rI_2 - \mu\sigma I_2 \\ \qquad + \frac{\mu\sigma}{L}\int_\Omega I_1\,dx, \\ \frac{\partial R_2}{\partial t} = D_2^R \frac{\partial^2 R_2}{\partial x^2} + rI_2 - \mu R_2 + \frac{\mu}{L}\int_\Omega R_1\,dx, \end{cases} \tag{8}$$

for all $t \geqslant 0$, $x \in \Omega = [0, L]$. Since this model is posed on a finite domain, one needs to specify additionally some boundary conditions. We take these to be *no-flux* or Neumann boundary conditions, i.e.,

$$\begin{aligned} &\frac{\partial S_i}{\partial x}(0) = \frac{\partial S_i}{\partial x}(L) = 0, \\ &\frac{\partial I_i}{\partial x}(0) = \frac{\partial I_i}{\partial x}(L) = 0, \\ &\frac{\partial R_i}{\partial x}(0) = \frac{\partial R_i}{\partial x}(L) = 0, \quad i = 1, 2. \end{aligned} \tag{9}$$

Physically these conditions mean that individuals cannot leave the regions they inhabit through the boundaries $x = 0$ or $x = L$. They can only move to another region by a long-distance travel. Arguments similar to those of the maximum principle [22] show that the solutions of the system (8) with nonnegative initial conditions will remain nonnegative for all times. This shows that the problem is well-posed in terms of its epidemiological interpretation. Finally, we notice that due to particular structure of the system (8), the total number of individuals in both regions

$$TP(t) = \int_\Omega (S_1 + I_1 + R_1 + S_2 + I_2 + R_2)(x, t)\,dx$$

remains constant throughout the evolution.

3. Equilibria and their stability

In order to understand possible dynamics in the system (8) we begin with the analysis of its equilibria.

3.1. Uniform steady states

Among various steady states which can be exhibited by the system an important place is occupied by the uniform (i.e., spatially homogeneous) equilibria. For the system (8) these equilibria are defined as solutions of the following system of equations

$$\begin{aligned} &-f(S_1, I_1) - \mu S_1 + \mu S_2 = 0, \\ &f(S_1, I_1) - rI_1 - \mu\sigma I_1 + \mu\sigma I_2 = 0, \\ &rI_1 - \mu R_1 + \mu R_2 = 0, \\ &-f(S_2, I_2) - \mu S_2 + \mu S_1 = 0, \\ &f(S_2, I_2) - rI_2 - \mu\sigma I_2 + \mu\sigma I_1 = 0, \\ &rI_2 - \mu R_2 + \mu R_1 = 0. \end{aligned} \tag{10}$$

From the first and the fourth equations of this system one concludes that $S_1 = S_2$. In the case of positive recovery rate $r > 0$, there is a continuum of steady states given by

$$(S_1, I_1, R_1) = (S_2, I_2, R_2) = (\hat{S}, 0, \hat{R}), \tag{11}$$

where $\hat{S} + \hat{R} = TP/2L$. These equilibria are stable provided

$$-r + \frac{\partial f}{\partial I}(\hat{S}, 0) < 0. \tag{12}$$

If, however, the recovery rate vanishes, then the steady states $(\hat{S}, 0, \hat{R})$ are linearly unstable, and there exists an additional set of linearly stable equilibria $(0, \hat{I}, \hat{R})$ with $\hat{I} + \hat{R} = TP/2L$. In view of the initial condition $R_1(0, x) = R_2(0, x) = 0$ it follows from the system (8) that the populations of recovered remain zero throughout the time evolution, and so $\hat{R} = 0$.

Inclusion of vital dynamics in our model can provide the existence of *endemic* equilibria characterized by nonzero values of all population densities.

3.2. Long-term asymptotic behaviour

As it was noted in the end of Section 2, the total population TP is constant. Using this and the fact that all S_i, I_i and $R_i, i = 1, 2$ are nonnegative for all $t \geqslant 0$, we conclude that all population densities remain bounded throughout the time evolution. This result allows one to study a long-time asymptotic behaviour of the system (8).

Assuming positive initial data, one obtains upon adding together the equations for S_1 and S_2 in the system (8) and integrating over the domain

$$\frac{\partial}{\partial t}\int_{\Omega}(S_1+S_2)\,dx = -\int_{\Omega}\big(f(S_1,I_1)+f(S_2,I_2)\big)\,dx \leqslant 0. \tag{13}$$

Since the total number $\int_{\Omega}(S_1+S_2)\,dx$ of susceptibles in both regions cannot be negative, the above inequality (13) suggests that in the limit $t\to\infty$ one must have

$$\lim_{t\to\infty}\frac{\partial}{\partial t}\int_{\Omega}(S_1+S_2)\,dx = 0.$$

Using relations (13) and the properties of the function f, one concludes that the function $f(S_i,I_i)$ approaches zero uniformly in both regions. This can happen if in each region either S or I becomes uniformly zero. Furthermore, from the first equation of the system (8) it follows that if $S_1\to 0$, then S_2 also tends to zero, and similar conclusions hold for S_2 and $I_{1,2}$. Hence, one concludes that asymptotically the system tends to a state, in which either

$$\lim_{t\to\infty} S_1(x,t) = \lim_{t\to\infty} S_2(x,t) = 0, \tag{14}$$

or

$$\lim_{t\to\infty} I_1(x,t) = \lim_{t\to\infty} I_2(x,t) = 0. \tag{15}$$

In the case (14) the long-term dynamics is determined by solutions of a reduced system

$$\begin{cases} \frac{\partial I_1}{\partial t} = D_1^I\frac{\partial^2 I_1}{\partial x^2} - (r+\mu\sigma)I_1 + \frac{\mu\sigma}{L}\int_{\Omega} I_2\,dx, \\ \frac{\partial I_2}{\partial t} = D_2^I\frac{\partial^2 I_2}{\partial x^2} - (r+\mu\sigma)I_2 + \frac{\mu\sigma}{L}\int_{\Omega} I_1\,dx, \\ \frac{\partial R_1}{\partial t} = D_1^R\frac{\partial^2 R_1}{\partial x^2} + rI_1 - \mu R_1 + \frac{\mu}{L}\int_{\Omega} R_2\,dx, \\ \frac{\partial R_2}{\partial t} = D_2^R\frac{\partial^2 R_2}{\partial x^2} + rI_2 - \mu R_2 + \frac{\mu}{L}\int_{\Omega} R_1\,dx, \end{cases} \tag{16}$$

with Neumann boundary conditions and nonnegative initial data. It is easy to see that this system of equations decouples and the first two equations form a closed system. The latter can be used using separation of variables and, moreover, the functions $\cos\frac{n\pi x}{L}$, $n\in\mathbb{Z}_+$ can be used as eigenfunctions of the corresponding Sturm–Liouville problems. Straightforward calculations yield

$$\begin{aligned} I_1(x,t) &= e^{-rt}\left\{\frac{f_0+g_0}{2} + \frac{f_0-g_0}{2}e^{-2\mu\sigma t} + 2\sum_{n=1}^{\infty} f_n e^{-(\mu\sigma+D_1^I\frac{n^2\pi^2}{L^2})t}\cos\frac{n\pi x}{L}\right\}, \\ I_2(x,t) &= e^{-rt}\left\{\frac{f_0+g_0}{2} + \frac{g_0-f_0}{2}e^{-2\mu\sigma t} + 2\sum_{n=1}^{\infty} g_n e^{-(\mu\sigma+D_2^I\frac{n^2\pi^2}{L^2})t}\cos\frac{n\pi x}{L}\right\}, \end{aligned} \tag{17}$$

where

$$f_n \equiv \frac{1}{L}\int_0^L I_1(x,0)\cos\frac{n\pi x}{L}\,dx,$$

$$g_n \equiv \frac{1}{L}\int_0^L I_2(x,0)\cos\frac{n\pi x}{L}\,dx.$$

It follows from (17) that as $t\to\infty$, the densities of infected individuals in both regions tend to equalize and simultaneously they both decrease exponentially with time, thus giving

$$\lim_{t\to\infty} I_1(x,t) = \lim_{t\to\infty} I_2(x,t) = 0.$$

Therefore, asymptotically the dynamics of the full system (8) is described by the system of equations for recovered individuals:

$$\begin{cases} \frac{\partial R_1}{\partial t} = D_1^R\frac{\partial^2 R_1}{\partial x^2} - \mu R_1 + \frac{\mu}{L}\int_{\Omega} R_2\,dx, \\ \frac{\partial R_2}{\partial t} = D_2^R\frac{\partial^2 R_2}{\partial x^2} - \mu R_2 + \frac{\mu}{L}\int_{\Omega} R_1\,dx. \end{cases} \tag{18}$$

It immediately follows that the solutions R_1 and R_2 of this system approach the uniform state

$$R_1(x) = R_2(x) = \frac{TP}{2L},$$

exponentially in time. In a particular case of vanishing recovery rate $r=0$, the solution of the system (8) approaches a linearly stable steady state of the form $(0, TP/2L, 0)$.

Similar consideration shows that in the case (15), the reduced system decouples, and the densities of susceptibles and recovered tend to equalize between the two regions thus settling on a stable steady state $(\hat{S},0,\hat{R})$ with $\hat{S}+\hat{R} = TP/2L$.

So, the general conclusion is that as the time evolves, the populations go from susceptible to recovered through being infected, and ultimately tend to equalize in both regions. If initially there are no infected individuals or their number is very small, then the time evolution will lead to equalizing susceptible populations but, obviously, there will be no transition to the infected class and further to recovered. Furthermore, if $r > 0$ and $-r + \partial f/\partial I(\hat{S}, 0) < 0$ then even if there is a small amount of infected individuals, then they will quickly move to a recovered class, and the epidemic will stop. This means that by the end of epidemic there will remain individuals in the class of susceptible who have never suffered from a disease.

On the other hand, if initially there is a nonzero number of infected individuals and sufficiently large number of susceptibles to violate $-r + \partial f/\partial I(\hat{S}, 0) < 0$, then initially there will exist an epidemic characterized by a growing number of infecteds. However, later the population densities of susceptibles will decrease thus decreasing the influx of new infecteds, and the system will eventually approach one of the stable steady states $(\hat{S}, 0, \hat{R})$. In a very special case of vanishing recovery rate, all steady states of the form $(\hat{S}, 0, \hat{R})$ are unstable, and the system settles itself on the linearly stable equilibrium of the form $(0, TP/2L, 0)$.

As we have already established, depending on initial data and parameters, the solution of system (8) tends to uniform steady states of the form $(S_1, I_1, R_1) = (S_2, I_2, R_2) = (\hat{S}, 0, \hat{R})$ with $\hat{S} + \hat{R} = TP/2L$, or $(S_1, I_1, R_1) = (S_2, I_2, R_2) = (0, TP/2L, 0)$. This implies that in the system (8) one cannot observe stable inhomogeneous steady states characterized by a non-trivial dependence of the population densities on a spatial coordinate. If the system (8) had a more involved structure possibly due to a vital dynamics or more complicated travel dynamics, then such inhomogeneous steady states could exist, and moreover, they could possess an interesting spatial dynamics similar to that of pattern forming systems [1].

4. Numerical simulations

In the previous sections we studied the steady state solutions of the model (8) and their stability. It was established that depending on initial conditions and parameters, the system has two classes of steady states which either have no infected individuals (general case of positive r) or have no susceptibles (a case of $r = 0$). At the same time, the existence of inhomogeneous steady states is prohibited within the validity of assumption on a disease transmission rate.

Now we use numerical simulations to study the temporal evolution of spatial distributions of individuals during the process of epidemic on two particular examples. At this stage one has to consider a particular form of a disease transmission function $f(S, I)$, and we take it to be of the bilinear form $f(S, I) = \beta SI$.

First, we fix $r = 0.5$ and take initial population to be equally distributed between two regions. In the first region it further equally distributed between susceptibles and infecteds as shown in the left panel of Fig. 1. Even though initially the number of infecteds grows, this trend quickly stops when the number of available susceptibles decreases. The final distribution in this case is presented in the right panel of Fig. 1 and illustrates the attractivity of the infecteds-free class of steady states. A speed of equalizing the populations in the two regions is determined by the parameters μ and σ, which characterize the rate of inter-region motion of individuals.

For the second simulation we take the same initial condition but with a vanishing recovery rate. In this case, after a short time, a redistribution of the individuals between the regions occurs, and simultaneously all of them become infected. Subsequently, infected individuals tend to equilibrium distribution in both regions shown in the right panel of Fig. 2. This confirms the conclusions from the previous section about the long-term asymptotic dynamics of the system (8).

5. Discussion

A model developed in this Letter demonstrates some important features of dynamics for the geographical spread of epidemics in the presence of a long-distance travel. When the recovery rate r is positive, for any initial condition the time evolution leads to a uniform steady state characterized by the absence of infected individuals. At the same time, in most cases, the proportion of susceptible individuals in this steady state is nonzero, and this means that after the

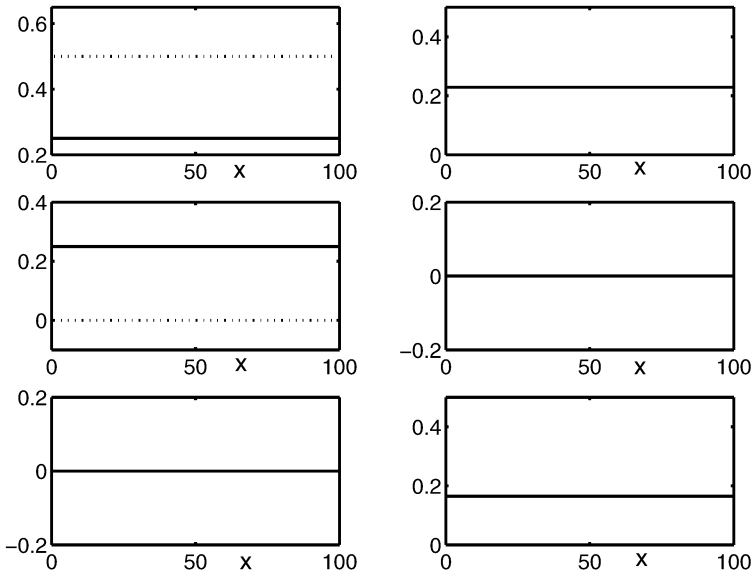

Fig. 1. Temporal dynamics with the parameter values $TP = L = 100$, $D = 1$, $\mu = 0.2$, $\beta = 0.5$, $\sigma = 1$ and $r = 0.5$. Left panel shows susceptible (top), infected (middle) and recovered (bottom) populations at time $t = 0$, while the right panel shows the eventual steady state. Densities are represented by solid lines in the first region, and by dotted lines in the second region.

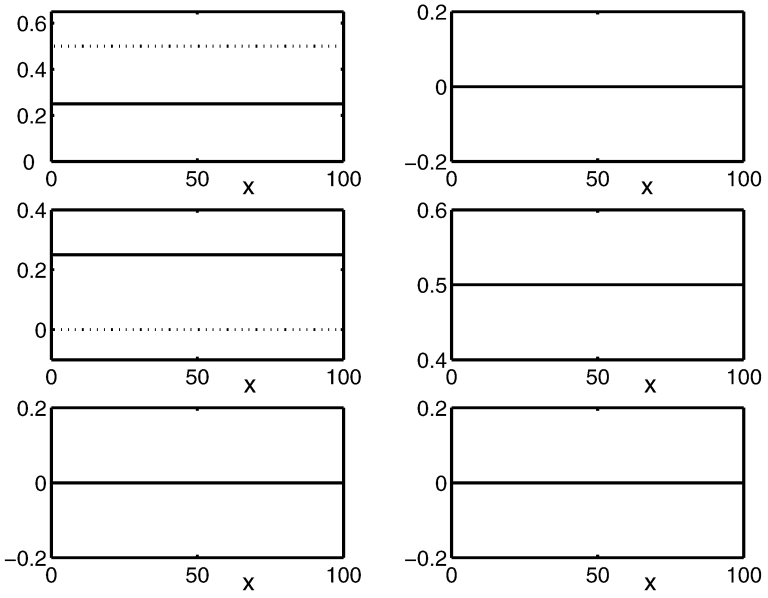

Fig. 2. The same as in Fig. 1 but with $r = 0$.

epidemic has finished there will be some individuals who have never suffered from a disease. Only when there is no recovery from a disease ($r = 0$), the final state of the system is characterized by the absence of susceptible individuals. In this case, the epidemic consists simply in transition from the class of susceptible into the class of infecteds as in the classical SI model, and the there are obviously no recovered individuals.

These conclusions are valid for any form of the disease transmission function f in (7).

There are several directions in which our model can be improved to better describe the mechanisms of spatial disease transmission. In particular, this model is a *migration* model, meaning that after an individual has travelled to another region, it has become a part of population inhabiting this region. At the same time, from the perspective of global travel, short-term visits for business or holidays seem to be more plausible source of spread of epidemics.

Another important issue to be considered is that of transmission dynamics itself, as described by the functions f^S, f^I. We took these to represent only the contacts between susceptible and infected individuals, but inclusion of vital dynamics (births and deaths, probably from disease) as well as different time delays corresponding to latency or temporary immunity periods, can substantially improve the performance of the model.

Finally, one can return to the model (1) and instead of simple diffusion mechanism for travel inside the regions, introduce the possibility of nonlocal diffusion, which is more realistic for countries with developed system of domestic air travel. Under assumption that the presence of disease reduces mobility of individuals by decreasing their diffusion coefficients, one can expect the possibility of spatial pattern formation from endemic steady states of the model. In particular, such patterning can occur in a model with vital dynamics and nonlinear incidence rate [23].

Using the kind of model considered in this Letter, together with detailed data about global air travel and understanding of local travel patterns inside different countries, can provide a possibility to determine trends in global spread of infectious diseases. This can also help develop appropriate measures for disease control and prevention.

Acknowledgements

The author would like to thank Peter Ashwin for stimulating discussions and the referees for their comments which helped improve the presentation in this Letter.

References

[1] J.D. Murray, Mathematical Biology, Springer-Verlag, New York, 1993.
[2] S.N. Busenberg, M. Ianelli, Nonlinear Anal. 7 (1983) 501.
[3] S.N. Busenberg, C.C. Travis, J. Math. Biol. 16 (1983) 181.
[4] V. Capasso, SIAM J. Appl. Math. 35 (1978) 274.
[5] W.E. Fitzgibbon, M.E. Parrott, G.F. Webb, Math. Biosci. 128 (1995) 131.
[6] G. Saccomandi, Math. Comput. Modelling 25 (1997) 83.
[7] D.G. Kendall, Mathematical model of the spread of infection, in: Mathematics and Computer Science in Biology and Medicine, H.M. Stationary Off, London, 1965, p. 213.
[8] D. Mollison, Adv. Appl. Probab. 4 (1972) 233.
[9] D. Mollison, J. R. Statist. Soc. B 39 (1977) 283.
[10] J. Medlock, M. Kot, Math. Biosci. 184 (2003) 201.
[11] L. Rass, J. Radcliffe, Spatial Deterministic Epidemics, American Mathematical Society, Providence, RI, 2003.
[12] J. Arino, P. van den Driessche, Math. Popul. Stud. 10 (2003) 175.
[13] L. Sattenspiel, K. Dietz, Math. Biosci. 128 (1995) 71.
[14] L. Hufnagel, D. Brockmann, T. Geisel, Proc. Natl. Acad. Sci. 101 (2004) 15124.
[15] M. Kuperman, G. Abramson, Phys. Rev. Lett. 86 (2001) 2909.
[16] M.E.J. Newman, Phys. Rev. E 66 (2002) 016128.
[17] M. Lipsitch, et al., Science 300 (2003) 1966.
[18] S. Riley, et al., Science 300 (2003) 1961.
[19] W. Liu, S.A. Levin, Y. Iwasa, J. Math. Biol. 23 (1986) 187.
[20] W. Liu, H.W. Hethcote, S.A. Levin, J. Math. Biol. 25 (1987) 359.
[21] V. Capasso, S.L. Paveri-Fontana, Rev. Epidém. Santé Publ. 27 (1979) 121.
[22] M.H. Protter, H.F. Weinberger, Maximum Principles in Differential Equations, Prentice–Hall, Englewood Cliffs, 1967.
[23] K.B. Blyuss, Phys. Rev. Lett. (2004), submitted for publication.