Spatial heterogeneity, social structure and disease dynamics of animal populations

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Abstract

Social groupings, population dynamics and population movements of animals all give rise to spatio-temporal variations in population levels. These variations may be of crucial importance when considering the spread of infectious diseases since infection levels do not increase unless there is a sufficient pool of susceptible individuals.

This paper explores the impact of social groupings on the potential for an endemic disease to develop in a spatially explicit model system. Analysis of the model demonstrates that the explicit inclusion of space allows asymmetry between groups to arise when this was not possible in the equivalent spatially homogeneous system. Moreover, differences in movement behaviours for susceptible and infected individuals gives rise to different spatial profiles for the populations. These profiles were not observed in previous work on an epidemic system. The results are discussed in an ecological context with reference to furious and dumb strains of infectious diseases.

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1. Introduction

Social structure has been widely observed amongst many animal species (Macdonald, 1984), and in recent years its impact on the spread of infectious diseases has been explored (see, for example, Holt and Pickering, 1985; Beardmore and White, 2001; Read and Keeling, 2003). In some cases, model systems are developed which are spatially homogeneous to consider the interaction between community and disease dynamics (Holt and Pickering, 1985; Beardmore and White, 2001). In other cases, the impact of space has been explored by its explicit inclusion in the model. Depending on both the ecological detail and on the questions which the model is used to address, different spatial approaches are employed. These include metapopulation models (Bolker and Grenfell, 1995; Lloyd and May, 1996; McCallum and Dobson, 2002; Keeling and Rohani, 2002), correlation models (Keeling, 1999; Read and Keeling, 2003), individual-based lattice models (Rand et al., 1995; Levin and Durrett, 1996) and reaction–diffusion systems (Gudelj et al., 2004).

Spatially extended models allow diseases and disease-free individuals to become spatio-temporally segregated. Any such local variations in population levels could be extremely important in an epidemiological context due to threshold effects. The threshold effect (Kermack and McKendrick, 1927, 1932, 1933) demonstrates that the disease levels will only increase if there is a sufficient supply of susceptible individuals. Consequently, if diseased individuals become isolated from susceptible individuals, infection levels for the total population may fall. This hypothesis has been successfully demonstrated with a theoretical model with a disease epidemic (Gudelj et al., 2004).

The lack of empirical studies on the behaviour of diseased individuals could be a result of the difficulties associated with observing and tracking sick animals.
Therefore many mathematical models do not distinguish between the behaviour of healthy and diseased animals. However, despite the difficulties there have been certain attempts at quantifying the behaviour of diseased animals (Cheeseman and Mallison, 1982; Artois and Aubert, 1985) and such studies have illustrated that diseased and disease–free individuals often display very different behaviours. Moreover, they have found that the behaviour of diseased animals could range from limited to highly random movements depending on the disease strain they are infected with. An example of limited movement can be found in badgers infected with tuberculosis where the abscess which is formed in the lumbar region of the animal, restricts its movement and in extreme cases causes paralysis (Cheeseman and Mallison, 1982). Another example of limited movement can be found in foxes infected with the so-called ‘dumb’ strain of rabies (Kaplan, 1977; Bacon, 1985). In this case, the virus enters the spinal cord of the animal causing photophobia and restricting the animal to its den. By contrast, it has been observed that some diseased badgers lose sense of direction as well as their fear of humans and cars resulting in the increased random movement and intrusion into neighbouring territories (Cheeseman and Mallison, 1982). Similar behaviour was observed amongst foxes infected with the so-called ‘furious’ strain of rabies (Kaplan, 1977; Bacon, 1985). We employ the terms furious and dumb throughout this paper to distinguish between the effects of different disease strains. We characterise these two strains as follows:

- a furious strain results in infected individuals increasing their random movements while paying little attention to any focal points within their home range.
- a dumb strain will give rise to infected individuals who have very restricted movement and who will often spend considerable time at a focal point within their home range.

This paper builds on previous work in which we explored the impact of social groupings on an endemic disease model for the spatially homogeneous case (Beardmore and White, 2001) and then its impact on an epidemic disease in a spatially extended system (Gudelj et al. (2004). In Beardmore and White (2001), model analysis showed that by increasing the strength of the intra-specific between group interaction for a two group system, the model could demonstrate bistability whilst, for sufficiently low levels of interaction, the endemic disease persisted with both groups at the same population levels. In Gudelj et al. (2004), disease strain was varied (furious versus dumb) and results clearly highlighted the importance of different disease strains on the infection process. Here we consider the impact of social groupings on a spatially extended system in which an endemic disease may persist, thus combining features of our previous two models. The model is deterministic, presented as a nonlinear reaction–diffusion system for which a variety of mathematical results are available to assist with analysis and to aid interpretation of model results. The system could be extended naturally to any number of interacting groups but this is likely to make the interpretation of results less clear and hence our 2 group restriction. Finally, because we assume no vertical transmission of the disease, this model is likely to be most relevant to vertebrate systems, with the examples of fox and badger as described above.

The following section describes the model structure, after which the results are presented and discussed in an ecological context. In particular, we explore how different disease strains give rise to different spatial profiles for the populations and how the potential for an endemic disease to persist affects the interaction between groups.

### 2. The model

We consider two animal groups belonging to the same species and divide each of them into the following two classes: susceptibles $S$ and infected $I$. In order to simplify the analysis and numerical simulations, we consider the problem on a one-dimensional domain $[0, l]$ where $l > 0$. However, the results described below could be extended into the two-dimensional domain. We restrict the system to that of two interacting groups, again to make the problem tractable for analysis, and simple enough to allow results to be clearly interpreted in terms of the underlying mechanisms. The reaction–diffusion system which we present is based on spatially local interactions. That is, population and disease interactions occur between individuals at a given location and time $t > 0$ and movement is governed by local gradients and distance from the group focal point. There is no explicit interaction between the dynamics (population and disease) and individual movements.

Let $S_i(x, t)$ and $I_i(x, t)$ denote the density of susceptible and infected animals respectively, in group $i$, where $i = 1, 2$. If the population density of a particular animal species is low the above state variables can be viewed as the expected density of finding an individual in the neighbourhood of $x$ at time $t$. This approach has been frequently used when modelling wolf population dynamics (see, for example, White et al., 1996; Lewis and Murray 1993).

With this format we make the following assumptions:

#### 2.1. Population dynamics

- Since we are interested in the social structure within a single animal population, our study is restricted to sympatric social groups and therefore it is reasonable to assume that they have the same demographic...
parameters (see, Lloyd and May, 1996; Beardmore and White, 2001; Keeling and Rohani, 2002). We have, however, carried out the analysis presented in the appendices and the numerical simulations for the case of asymmetric groups and have obtained qualitatively consistent results.

- All individuals give birth at a constant per capita birth rate \( a \). We assume that there is no vertical disease transmission from a mother to her unborn offspring and hence both susceptible and infected individuals give birth to susceptible offspring. This is a reasonable assumption for vertebrate populations since many studies report no evidence of individuals being born with the infection (see, Anderson and Trehella, 1985; Bacon, 1985).

- The natural death rate for susceptibles is a constant per capita rate \( b \). Infected individuals do not recover (certain death) and the per capita mortality rate for infected is \( c \). Since this includes both natural and disease-induced mortality, we have \( c > b \).

- For simplicity we assume a homogeneous disease transmission, modelled using a standard mass action law. Within group, the transmission coefficient is denoted by \( \lambda \); between groups the contact rate per capita is denoted \( q \) and the probability that this contact results in disease transmission is given as \( q \).

Note that one way of including a heterogeneous disease transmission would be through a pseudo mass action term, for examples see, Keeling and Rohani (2002) and McCallum et al. (2001). However, the simplified assumption of a homogeneous disease transmission, enables us to asses the impact of animal movement, on disease dynamics, by directly comparing our results to the work in (Beardmore and White, 2001), where animal movement was omitted. Moreover, the choice of parameter labels \( \lambda, \eta \) and \( q \) have all been made so that the population dynamic part of the system is as given in (Beardmore and White, 2001).

2.2. Movement

- Movement for both susceptible and infected individuals consists of two components: dispersal to forage and protect the territory or home range and movement towards a focal point in the domain. Note that even though the movement of both susceptible and infected animals is divided into the same components (diffusion and convection), the strength of these components is allowed to vary between the susceptible and infected animals as well as between the groups.

- Animal dispersal is modelled using a random walk approach with diffusion coefficient \( D_{ij} \).

- Animal movement towards a focal point is modelled by a convection towards that point in the domain \( \Omega \).

The analytic results (see Appendix) have been undertaken for a general class of sufficiently smooth convection functions \( C_{ij} \), with

\[
C_{ij}(0) < 0 \quad \text{and} \quad C_{ij}(l) > 0 \quad (1)
\]

for \( i = 1, 2 \) and \( j = 1, 2 \). Here \( i \) denotes susceptible (\( i = 1 \)) and infected (\( i = 2 \)) animals and \( j \) denotes group 1 (\( j = 1 \)) and group 2 (\( j = 2 \)).

In the case where it was not possible to obtain general analytic results (Section 3.2), the model outcomes are described by numerical simulations. The numerical simulations were conducted for a range of convection functions satisfying (1) all of which provided qualitatively similar results. For the purpose of illustration we present the outcome for the convection term motivated by Okubo (1980) and White et al. (1996)

\[
C_{ij}(x) = C_{ij} \tanh(x - x_d),
\]

where \( C_{ij} \) is a positive constant measuring the maximum speed of susceptible (\( i = 1 \)) and infected (\( i = 2 \)) animals in group 1 (\( j = 1 \)) and group 2 (\( j = 2 \)) when moving towards their focal points, and \( x_d \) representing the location of the focal point in group \( j \) where \( j = 1, 2 \).

- Differences in disease strains for infected individuals are explored by varying the relative magnitude of movement parameters for the infected classes. For example, the dumb form of disease within social group \( j \) can be represented by a large convection coefficient (\( C_{2j} \)) and a small diffusion coefficient (\( D_{2j} \)). By contrast, for a furious strain, we choose a small convection coefficient (\( C_{2j} \)) and a large diffusion coefficient (\( D_{2j} \)).

- The strength of the convection coefficient for healthy animals is determined by how active the focal point is. During the breeding season the focal point is highly active and this can be represented by a large convection coefficient \( C_{1j} \). Equivalently, zero or a small convection coefficient would represent a focal point with no or low activity, respectively.

Mathematically, the growth, group interaction and animal movement assumptions described above combine to give a system of coupled partial differential equations

\[
\begin{align*}
\frac{\partial S_1}{\partial t} &= D_{11} \frac{\partial^2 S_1}{\partial x^2} + \frac{\partial}{\partial x} (C_{11}(x)S_1) + f_1(S_1, I_1, S_2, I_2), \\
\frac{\partial I_1}{\partial t} &= D_{21} \frac{\partial^2 I_1}{\partial x^2} + \frac{\partial}{\partial x} (C_{21}(x)I_1) + f_2(S_1, I_1, S_2, I_2), \\
\frac{\partial S_2}{\partial t} &= D_{12} \frac{\partial^2 S_2}{\partial x^2} + \frac{\partial}{\partial x} (C_{12}(x)S_2) + f_3(S_1, I_1, S_2, I_2), \\
\frac{\partial I_2}{\partial t} &= D_{22} \frac{\partial^2 I_2}{\partial x^2} + \frac{\partial}{\partial x} (C_{22}(x)I_2) + f_4(S_1, I_1, S_2, I_2),
\end{align*}
\]

(2)
where for $i = 1, \ldots, 4$, $f_i(S_i, I_1, S_2, I_2)$ is defined as follows:

$$f_1 = (a - b)S_1 - \lambda S_1 I_1 - \eta q S_1 I_2 + a I_1,$$

$$f_2 = \lambda S_1 I_1 + \eta q S_1 I_2 - c I_1,$$

$$f_3 = (a - b)S_2 - \lambda S_2 I_2 - \eta q S_2 I_1 + a I_2,$$

$$f_4 = \lambda S_2 I_2 + \eta q S_2 I_1 - c I_2.$$

To completely specify the problem we impose no flux boundary conditions at $x = 0$ and $x = l$,

$$0 = D_{11} \frac{\partial S_i}{\partial x} + C_{11}(x) S_i,$$

for $i = 1, 2$. For the purpose of illustration homogeneous initial conditions were used in the simulations.

### 3. Model outcomes

To compare with Gudelj et al. (2004) we are interested in analysing the model to explore how an endemic (persistent) disease affects population levels and spatial distributions. Therefore, we concentrate on the long term behaviours of the model system. Mathematical details have been put in the appendices.

With $a = b = 0$, (2) reduces to a system which has infinitely many disease-free group coexistence steady states. This has been discussed elsewhere (Gudelj et al., 2004).

Depending on the value of the birth parameter $a$ the outcomes of the model can be divided into the following three cases:

#### Case I: $a < b < c$

#### Case II: $b < c < a$

#### Case III: $b < a < c$.

**Case I:** If the population birth rate $a$, is smaller than the natural death rate $b$, (which in turn is smaller than the disease-induced death rate $c$), we can show that both social groups become extinct (see Appendix A for mathematical details).

This finding corresponds to the spatially homogeneous case (no animal movement) in Beardmore and White (2001), where for $a < b < c$ the only biologically relevant stable steady state is the zero steady state.

Due to the small birth rate, in the absence of a disease the population is not able to regulate itself and always dies out. Therefore if a density of susceptible population cannot be maintained above some critical threshold value, an infection which is introduced into the population, will not be able to persist and hence the populations die out.

**Case II:** If the population birth rate is greater than the disease-induced death rate, we can show that the population levels in each social group become arbitrarily large. While in the absence of a disease, the levels of susceptible populations become arbitrarily large, the introduction of disease into both social groups restricts susceptible levels while the infected population becomes arbitrarily large (see Appendix B for details).

In this case, the sufficiently large susceptible birth rate will maintain population levels above a certain threshold, causing infection levels to become arbitrarily large. Since the disease-induced death rate is smaller than the rate at which susceptible individuals are born, infection levels will not decrease. Since we assume no vertical transmission high numbers of infected individuals will ensure a high influx of newborns into the susceptible population, keeping it above a critical threshold value. Hence, the arbitrarily large infection levels.

Note that in both Cases I and II, the outcome is independent of the model parameters, and therefore independent of the between group contact rate (see Appendices A and B for mathematical calculations).

**Case III:** In this case, the population birth rate is greater than the natural death rate, but smaller than the disease-induced death rate and both spatial structure and social contact have important impacts on the disease dynamics.

#### 3.1. The spatially homogeneous case

The spatial homogeneous model in Beardmore and White (2001) can be obtained by omitting the spatial
components from (2), to get
\[
\begin{align*}
\frac{dS_1}{dt} &= (a-b)S_1 - \lambda S_1 I_1 - \eta q S_1 I_2 + a I_1, \\
\frac{dI_1}{dt} &= \lambda S_1 I_1 + \eta q S_1 I_2 - c I_1, \\
\frac{dS_2}{dt} &= (a-b)S_2 - \lambda S_2 I_2 - \eta q S_2 I_1 + a I_2, \\
\frac{dI_2}{dt} &= \lambda S_2 I_2 + \eta q S_2 I_1 - c I_2.
\end{align*}
\] (6)

For \( b < a < c \) the model outcomes depend on the value of \( \eta \), as shown in Fig. 1. If \( \eta < \frac{c}{b} \) (6) has a stable steady state where both populations coexist with the disease. The population levels in the two groups are symmetric.

At \( \eta = \frac{c}{b} \) (6) has a continuum of steady states forming a vertical bifurcation from coexistence steady state.

For sufficiently large \( \eta \), \( \eta > \frac{c}{b} \) (6) exhibits bistability and depending on the initial population and the number of infected individuals only one group is able to coexist with the disease while the other one becomes extinct.

3.2. The spatially heterogeneous case

Finally, we consider the complete system (2) and (4) with \( b < a < c \). If there is no contact between the groups (\( \eta = 0 \)) it can be shown that there is a unique globally attractive coexistence steady state of susceptible and infected individuals (see Beardmore and Beardmore, 2003 for details). The population levels at this steady state depend on the values of \( a \). Namely, if \( a \) is close to \( b \) the total number of infected individuals is smaller than the total number of susceptibles, the opposite holds if \( a \) is close to \( c \).

However, if the contact rate between the groups \( \eta \neq 0 \) the mathematical analysis of (2) becomes much more complex. Given the results of Beardmore and Beardmore (2003), it could be expected that the disease-mediated group coexistence is present at least for small values of \( \eta \).

In this section, we present the results of numerical simulations guided by the previous studies of single group dynamics (Beardmore and Beardmore, 2003) and spatially homogeneous two group interaction (Beardmore and White, 2001). Parameter values used in the simulations shown are arbitrary and were chosen to demonstrate model outcomes.

Extensive numerical simulations indicate that disease-mediated group coexistence is possible. As in the spatially homogeneous case (6), the between group contact rate \( \eta \) influences the form of steady states of (2). If \( \eta \) is small both groups coexist in the disease-mediated coexistence with an equal number of individuals in both groups (see Fig. 2 for an illustration of the bifurcation diagram). This follows the behaviour of the spatially homogeneous case (Fig. 1).

![Fig. 1. Steady states of (6) shown for susceptible population in group 1 and continued in the between-group contact parameter \( \eta \). The solid line denotes stability while the dashed line denotes the instability of a steady state. The model parameters are: \( a = 0.6 \), \( b = 0.1 \), \( c = 0.65 \), \( q = 0.4 \) and \( \lambda = 0.5 \).](image-url)
For intermediate values of \( \eta \), this symmetry is lost and one group is always dominant with initial total population levels determining the dominance. This differs from the spatially homogeneous case where symmetrical coexistence is the only possible disease-mediated state.

If \( \eta \) is large (2) exhibits bistability with one group always out-competing the other, initial total population levels determining the winner. This result also corresponds to the spatially homogeneous case, Fig. 1.

To summarise, including spatial aspects into the model allows asymmetries to arise between groups.

### 3.2.1. Varying demographics via the birth rate \( a \)

Now we explore how long-term population levels and distributions vary with the disease strain present. As described above, we focus on two strains labelled furious and dumb both of which are characterised in terms of movement parameters. Comparisons are made as the birth rate \( a \) is varied. This choice was made as a measure of input into the system but other parameter choices would be equally as appropriate.

#### 3.2.1.1. Population levels

If \( a \) is close to \( b \), susceptible population levels at the pathogen-mediated coexistence steady state are higher than those of the infected population (Fig. 3). However, as \( a \) increases the infected population levels increase quicker than the levels of susceptible individuals and eventually the levels of infected individuals at the coexistence steady state become higher than the susceptible levels.

This somewhat counter-intuitive result can be explained as a consequence of the threshold phenomenon for microparasitic diseases—namely that infection levels will only increase in a population if the level of susceptibles exceeds some threshold level. Increasing \( a \) results in more susceptible individuals and hence more disease.
In Fig. 3, we compare the dumb and furious disease cases and see that if the disease strain is dumb then total population levels for the susceptible and infected groups are lower than those when the strain is furious. In the dumb case, infection is spatially localised and this results in a significant decline locally in susceptible levels and a corresponding decline in infection levels. By contrast, when disease is furious, the population levels are higher because this strain of the disease allows the infected population to attack more of the susceptible population but locally there is less infection which allows larger growth rates for the susceptible population.

3.2.1.2. Population distribution. From Fig. 3, we see that when \( a \) is small, the total infected population is less than the total susceptible population but that this relation is reversed as \( a \) increases.

If infected individuals in each group suffer from the dumb form of the disease they are situated in their highest numbers around their respective foci. For small \( a \) (Fig. 4(a)), since infection levels are relatively low, the distribution of susceptibles is not affected by the infected population congregating around the focal point and the population distribution remains unimodal. This changes for large \( a \) (Fig. 4(b)) where high infection levels, focussed about the focal point produce a bimodal susceptible population distribution.

If infected individuals in each group suffer from the furious form of the disease, the infected population at the coexistence steady state is almost uniformly distributed across the domain. Again with small \( a \) (Fig. 5(a)), the infection levels are low and \( S \) remains unimodal. However, as \( a \) increases (Fig. 5(b)), infected levels increase across the whole of the domain and the distribution of the susceptible individuals at the coexistence steady state is almost uniformly distributed at the threshold level.

4. Discussion

In this paper we have extended previous studies (Beardmore and White, 2001; Gudelj et al., 2004) to consider the impact of social groupings on an endemic disease model in a spatially extended system.

The results show that introduction of spatially explicit components generates new solution dynamics, not previously observed in the non-spatial case. In the non-spatial model both groups are able to coexist with each other and the disease providing that the between-group contact rate \( \eta \) is small. The population levels at this disease-mediated steady state are equal for both groups. This symmetry is expected as it is assumed that the groups belong to the same species and their demographic parameters are identical. As \( \eta \) increases, the competitive exclusion where one group out-competes the other is the only stable outcome. Initial conditions determine the winner and the surviving group coexists with the disease. These results correspond to the spatial case with one important difference. In the spatial case there also exists a range of intermediate values of \( \eta \) for which a new steady-state structure is generated. In this case, the social groups coexist with each other and the disease but the symmetry in population levels between the groups is not present. One group is dominant and initial total population levels determine the dominance. This symmetry breaking is
somewhat counterintuitive as both social groups have the same population dynamics and movement behaviour.

The impact of between-group competition on disease dynamics was considered in the spatially homogeneous case by Beardmore and White (2001). Due to the complex structure of the spatially heterogeneous model a similar detailed investigation was beyond the scope of this paper. However, in some cases numerical simulations of the spatial model agree with the observations made in Beardmore and White (2001) that between group competition could be a possible mechanism for a species to escape parasitism. For example if the interactions between the groups occur through disease transmission only, the between group contact rate is always detrimental to the susceptible and infected population levels at the coexistence steady state. This holds for both spatial (6) and non-spatial (2) models. However, if the intra-specific between group competition is present in the spatially homogeneous case, the steady-state population levels for the susceptible population at the group coexistence steady state is higher than it would be if the groups existed on their own. On the other hand corresponding levels for the infected population are lower, see Beardmore and White (2001). Interestingly, when spatial structure is introduced, the above observations arise only for certain values of the movement parameters, namely small convection and large diffusion values. This corresponds to the so-called furious disease strain and in this case the distribution of susceptible and infected individuals at the steady state is almost uniform, see Fig. 5(b). Therefore, the lack of spatial heterogeneity could explain the similarities between the non-spatial and the spatial (furious disease) cases.

Focusing on the population demographics and in particular the birth rate for the population, we have explored how this parameter affects both disease levels and population distributions. Increasing \(a\) can lead to larger increases in infected levels compared with susceptible levels which we explain in terms of the threshold phenomenon for infectious diseases. This phenomenon also explains differences in model outcomes for the dumb and furious strains of a disease. In the dumb case, localised infection reduces the pool of susceptibles locally and hence infection levels. In contrast, furious disease spreads rapidly throughout the region and results in uniform reduction in susceptible levels. This causes a uniform reduction in infection and hence the possibility for susceptibles to grow once again.

When considering spatial population distributions, differences also arise between the dumb and furious strain of a disease. The most interesting result occurs for the dumb disease where increases in \(a\) result in unimodal distributions breaking down to give a bimodal distribution about the focal point. In the furious case, this does not occur—rather, an increase in \(a\) results in spatially homogeneous distributions because disease is more spread out and susceptible individuals can avoid it more easily.

Due to the lack of experimental data on the behaviour of diseased animals the work undertaken in this study is of a theoretical nature. We have developed a general spatially explicit epidemic model that is directly comparable to the non-spatial model already studied in Beardmore and White (2001). The results demonstrate the importance of spatial heterogeneity when modelling disease dynamics of social animals.

Although this work focuses primarily on social groups of feral animals belonging to the same species, it can
easily be adapted to various host–pathogen systems. For example, the analysis conducted in the appendix carries through for the case when social groups do not belong to the same species and hence have different demographic parameters. It can also easily be shown that in that case disease-mediated group coexistence is possible only if $b < a < c$ in both groups.

Appendix A. $a < b < c$

Assume that (2)–(4) admits solutions with $(S_{1,0}, I_{1,0}, S_{2,0}, I_{2,0}) \in C(\Omega) \times C(\Omega) \times C(\Omega) \times C(\Omega)$, see Henry (1981) for details. Let us define the functional $V : X \times X \times X \to \mathbb{R}$ to be the total biomass

$$V(S_1, I_1, S_2, I_2) = \int_{\Omega} \left( S_1 + I_1 + S_2 + I_2 \right) dx = ||S_1||_{L^1} + ||I_1||_{L^1} + ||S_2||_{L^1} + ||I_2||_{L^1}.$$ 

Using (2) we find that

$$\frac{d}{dt} V(S_1, I_1, S_2, I_2) = \int_{\Omega} (S_{1t} + I_{1t} + S_{2t} + I_{2t}) dx$$

and hence

$$V(t) \leq e^{\delta t} V(0),$$

so that

$$||S_1||_{L^1} + ||I_1||_{L^1} + ||S_2||_{L^1} + ||I_2||_{L^1} \to 0.$$ 

Appendix B. $b < c < a$

For $x \in \tilde{\Omega}$ and $i, j = 1, 2$ we define

$$\phi_i(y) : = \exp \left( - \frac{1}{D_y} \int_0^y C_\gamma(\zeta) d \zeta \right).$$

Since $\phi_i \in C^1$ on a bounded domain $\tilde{\Omega}$, there exist $\phi_{i,\max}, \phi_{i,\min} \in \mathbb{R}$ such that

$$\phi_{i,\min} \leq \phi_i(x) \leq \phi_{i,\max}, \quad x \in \tilde{\Omega}.$$ 

**Proposition 1.** Let $0 < b < c < a$ and for $i = 1, 2$ let $g_i(x)$ denote a bounded $C^\infty(0, l)$ function such that

$$\frac{dg_i}{dx}(0) \geq 0, \quad \frac{dg_i}{dx}(l) \leq 0$$

and

$$g_i(x) \geq g_i(0) + \int_0^x \frac{d^2g_i}{dx^2}(y) dy + c + \beta \int_0^x \phi_i(y) \left( \int_y^l \phi_i^{-1}(\xi) d\xi \right) dy$$

for all $x \in [0, l]$. Given a continuous initial data $(S_{1,0}, I_{1,0}, S_{2,0}, I_{2,0})$ such that $S_{1,0}, I_{1,0} \geq 0$ and

$$I_{1,0} \geq \frac{(a - b)}{\eta \phi_{21,\min}} e^{\delta t} \phi_{21},$$

$$I_{2,0} \geq \frac{(a - b)}{\eta \phi_{22,\min}} e^{\delta t} \phi_{21},$$

the solution $(S_1, I_1, S_2, I_2)$ of (2)–(4) satisfies

$$|| (S_1(t), I_1(t), S_2(t), I_2(t)) ||_{C^0} \to \infty,$$

as $t \to \infty$. Moreover $S_i$ stays bounded while $I_i \to \infty$ as $t \to \infty$, for $i = 1, 2$.

**Proof.** For $f = (f_1, f_2, f_3, f_4)$ defined in (3) since $\text{sign}(\partial f_i / \partial I_i) = \text{sign}(-\lambda_S + a)$, is not constant for all $S_i \geq 0$, it follows that $f$ is not quasi-monotone and therefore, in order to prove the proposition, we are required to construct a pair of generalised upper and lower solutions of (2)–(4), (see Pao, 1992).

The following inequalities need to be satisfied for $(S_1, I_1, S_2, I_2)$ and $(S_1, I_1, S_2, I_2)$ to be a pair of generalised upper and lower solutions of (2)–(4).

First, the boundary inequalities

$$- D_{1t} \frac{\partial S_1}{\partial x} - C_{1i}(x) S_i \frac{\partial S_1}{\partial x} \geq - D_{1t} \frac{\partial S_1}{\partial x} - C_{1i}(x) S_i \text{ at } x = 0,$$

$$D_{1t} \frac{\partial S_1}{\partial x} + C_{1i}(x) S_i \frac{\partial S_1}{\partial x} \geq D_{1t} \frac{\partial S_1}{\partial x} + C_{1i}(x) S_i \text{ at } x = l,$$

$$- D_{2t} \frac{\partial I_1}{\partial x} - C_{2i}(x) I_i \frac{\partial I_1}{\partial x} \geq - D_{2t} \frac{\partial I_1}{\partial x} - C_{2i}(x) I_i \text{ at } x = 0,$$

$$D_{2t} \frac{\partial I_1}{\partial x} + C_{2i}(x) I_i \frac{\partial I_1}{\partial x} \geq D_{2t} \frac{\partial I_1}{\partial x} + C_{2i}(x) I_i \text{ at } x = l,$$
Next, the inequalities
\[
\begin{align*}
\frac{\partial S_i}{\partial t} - D_i \frac{\partial^2 S_i}{\partial x^2} - C_i \frac{\partial S_i}{\partial x} & \\
& \geq \frac{\partial C_i}{\partial x} \frac{\partial S_i}{\partial x} + (a - b) S_i - \lambda S_i I_i - \eta q S_i I_j + a I_i,
\end{align*}
\]
need to hold, for \((i, j) \in \{(1, 2), (2, 1)\}\) and for all \(I_i\) such that \(I_i \leq I_i \leq \bar{I}_i\), with \(i = 1, 2\).

And finally,
\[
\begin{align*}
\frac{\partial S_i}{\partial t} - D_i \frac{\partial^2 S_i}{\partial x^2} - C_i \frac{\partial S_i}{\partial x} & \\
& \leq \frac{\partial C_i}{\partial x} \frac{\partial S_i}{\partial x} + (a - b) S_i - \lambda S_i I_i - \eta q S_i I_j + a I_i,
\end{align*}
\]
need also to hold, for \((i, j) \in \{(1, 2), (2, 1)\}\) and for all \(S_i\) such that \(S_i \leq S_i \leq \bar{S}_i\), with \(i = 1, 2\).

Let \(\beta, K_i, K_2\) be arbitrary positive constants and let \(\gamma, M_1, M_2, E_1, E_2\) be positive constants satisfying
\[
\begin{align*}
M_1 & \geq \frac{a}{\lambda \phi_{1_{\text{min}}}}, \quad M_2 \geq \frac{a}{\lambda \phi_{1_{\text{min}}}} \quad E_1 \geq \frac{(a - b)}{\eta q \phi_{2_{\text{min}}}},
\end{align*}
\]
\[
\begin{align*}
E_2 & \geq \frac{(a - b)}{\eta q \phi_{2_{\text{min}}}},
\end{align*}
\]
\[
\gamma \geq \max \{M_1 (\phi_{1_{\text{max}}}), (\lambda + \eta q K) - c, M_2 \phi_{1_{\text{max}}} \}
\times \left( \frac{\lambda + \eta q K}{K} - c \right),
\]
where
\[
K = \frac{K_2 \phi_{2_{\text{max}}}}{K_1 \phi_{2_{\text{min}}}}.
\]

Then the pair
\[
(S_1, I_1, S_2, I_2) = (M_1 \phi_{1_{\text{max}}} (\lambda + \eta q K) - c, M_2 \phi_{1_{\text{max}}}, E_1 \phi_{2_{\text{min}}} (\lambda + \eta q K) - c, E_2 \phi_{2_{\text{min}}} (\lambda + \eta q K) - c)
\]
(B.10)
\[
(S_1, I_1, S_2, I_2) = (0, E_1 e^{\beta t} e^{g_1(x)} \phi_{2_{\text{max}}}, 0, E_2 e^{\beta t} e^{g_2(x)} \phi_{2_{\text{max}}})
\]
(B.11)
is a pair of generalised upper and lower solutions of (2)–(5). Since,
\[
D_i \phi_{1_{\text{x}}} + C_i (x) \phi_{1_{\text{x}}} = 0,
\]
for \(i, j = 1, 2\), the boundary inequalities (B.5a) and (B.5b) are clearly satisfied, while the boundary inequal-
\[
\begin{align*}
-D_2 I_{1_{\text{x}}} - C_2 (x) I_{1_{\text{x}}} & = E_1 e^{\beta t} \frac{d}{dx} \left( g_1(x) \right) (0) \phi_{2_{\text{x}}}(0) \leq 0, \quad i = 1, 2,
\end{align*}
\]
\[
-D_2 I_{1_{\text{x}}} + C_2 (x) I_{1_{\text{x}}} = E_1 e^{\beta t} \frac{d}{dx} \left( g_1(x) \right) (l) \phi_{2_{\text{x}}}(l) \leq 0, \quad i = 1, 2,
\]
which is true since (B.2) holds.

Condition (B.6a) at \((i, j) = (2, 1)\) holds provided
\[
0 \geq (a - b) M_1 \phi_{1_{\text{x}}} + a I_{1_{\text{x}}} - \lambda M_1 \phi_{1_{\text{x}}} I_{1_{\text{x}}} - \eta q M_1 \phi_{1_{\text{x}}} I_2,
\]
\[
\forall I_i \leq I_i \leq \bar{I}_i, \quad i = 1, 2.
\]
This is true if
\[
M_1 \phi_{1_{\text{x}}} ((a - b) - \eta q L_2) \leq 0 \quad \text{and} \quad I_1 (a - \lambda M_1 \phi_{1_{\text{x}}}) \leq 0,
\]
which in turn hold provided
\[
\frac{(a - b)}{\eta q} \leq E_2 e^{\beta t} e^{g_1(x)} \phi_{2_{\text{x}}} \quad \text{and} \quad \frac{a}{\lambda \phi_{1_{\text{x}}}} \leq M_1,
\]
which is true for all \(x \in \Omega\) and \(t \in (0, \infty)\), provided (B.8) hold.

Equivalently, condition (B.6a) at \((i, j) = (2, 1)\) is satisfied provided
\[
0 \geq (a - b) M_2 \phi_{1_{\text{x}}} + a I_{1_{\text{x}}} - \lambda M_2 \phi_{1_{\text{x}}} I_{1_{\text{x}}} - \eta q M_2 \phi_{1_{\text{x}}} I_2,
\]
\[
\forall I_i \leq I_i \leq \bar{I}_i, \quad i = 1, 2,
\]
which is true for all \(x \in \Omega\) and \(t \in (0, \infty)\), if (B.8) hold.

Condition (B.7a) is satisfied provided that for all \(S_i\), \(i = 1, 2\), such that \(S_i \leq S_i \leq \bar{S}_i\),
\[
\gamma M_1 e^{\beta t} \phi_{2_{\text{x}}} \geq (\lambda S_1 K_1 e^{\beta t} \phi_{2_{\text{x}}} + \eta q S_1 I_2 - c K_1 e^{\beta t} \phi_{2_{\text{x}}}) \quad \forall I_2 \leq I_2 \leq \bar{I}_2,
\]
\[
\gamma M_2 e^{\beta t} \phi_{2_{\text{x}}} \geq (\lambda S_2 K_2 e^{\beta t} \phi_{2_{\text{x}}} + \eta q S_2 I_2 - c K_2 e^{\beta t} \phi_{2_{\text{x}}}) \quad \forall I_2 \leq I_2 \leq \bar{I}_2.
\]
The above system of inequalities hold if
\[
\begin{align*}
\gamma & \geq \bar{S}_1 \left( \lambda + \eta q K_2 \phi_{2_{\text{max}}}K_1 \phi_{2_{\text{min}}} - c \right) \\
\gamma & \geq \bar{S}_2 \left( \lambda + \eta q K_2 \phi_{2_{\text{max}}}K_1 \phi_{2_{\text{min}}} - c \right),
\end{align*}
\]
which is true for all \(x \in \Omega\) and \(t \in (0, \infty)\), provided (B.9) is satisfied.

Finally, condition (B.7) is satisfied if for all \(S_i\), \(i = 1, 2\) such that \(S_i \leq S_i \leq \bar{S}_i\),
\[
\begin{align*}
\beta & \leq D_2 g_{1_{\text{x}}} (x) + D_2 (g_{1_{\text{x}}}) (x)^2 \quad + C_2 (x) g_{1_{\text{x}}} (x) + \lambda S_1 \\
& \quad + \eta q \frac{1}{E_1 \phi_{2_{\text{x}}}} e^{-\beta t} e^{-g_{1_{\text{x}}} (x)} S_1 I_2 - c, \quad \forall I_2 \leq I_2 \leq \bar{I}_2.
\end{align*}
\]
\( \beta \leq D_{22}g_{2x}(x) + D_{22}(g_{2x})^2(x) + C_{22}(g_{2x}(x) + \Delta S_2) + \eta g \frac{1}{E_2\phi_{22}} e^{-\beta t} e^{-g_2(x)} S_2 I_1 - c, \quad \forall t \leq t_1 \leq t \).

The above inequalities hold provided
\( D_{23}g_{3x}(x) + C_{23}(g_{3x}(x) - (c + \beta) \geq 0, \)
for \( i = 1, 2, \) which, in turn, is satisfied if
\( (\phi_{2i}^{-1} g_{2x}(x))_x - \frac{c + \beta}{D_{2i}^2} \phi_{2i}^{-1} \geq 0, \)
holds for \( i = 1, 2. \) This is true for all \( x \in \Omega \) and \( t \in (0, \infty), \) provided (B.3) is satisfied.

Condition (B.6b) clearly holds and therefore we can apply the Existence--comparison theorem, see Pao (1992). Suppose that \( (S_{1i}, I_{1i}, S_{2i}, I_{2i}) \) is a continuous initial data such that \( S_{10}, S_{20} \geq 0 \) and (B.4) hold. Given suitable positive constants \( M_i, E_i, K_i \) for \( i = 1, 2 \) so that
\( (0, E_i e^{\beta t} \phi_{21}, 0, E_2 e^{\beta t} \phi_{22}) \)
\( \leq (S_{10}, I_{10}, S_{20}, I_{20}) \)
\( \leq (M_1 \phi_{11}, K_1 \phi_{21}, M_2 \phi_{12}, K_2 \phi_{22}), \)
system (2)–(4) has a unique solution
\( (S_1, I_1, S_2, I_2) \in C(\bar{T}) \times C(\bar{T}) \times C(\bar{T}) \) with
\( (0, E_i e^{\beta t} \phi_{21}, 0, E_2 e^{\beta t} \phi_{22}) \)
\( \leq (S_1, I_1, S_2, I_2) \)
\( \leq (M_1 \phi_{11}, K_1 e^{\beta t} \phi_{21}, M_2 e^{\beta t} \phi_{12}, K_2 e^{\beta t} \phi_{22}), \)
provided (B.8) and (B.9) hold.  

Note that as \( t \to \infty \) the \( S \) components of the solution stay in the bounded region
\( 0 \leq S_i \leq M_i \phi_{1i}, \)
but \( ||I_i||_0 \to \infty, \) as \( t \to \infty, \) for \( i = 1, 2. \)

References
