

# QUALITATIVE ANALYSIS OF AN EPIDEMIC MODEL WITH DIRECTED DISPERSION<sup>†</sup>

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**Abstract.** Qualitative properties and the large-time existence of solutions to the Gurtin-MacCamy model are studied. The Gurtin-MacCamy model is a system of nonlinear and degenerate PDEs in which the classical Kermack- McKendrick equations are augmented with spatial dispersion. Numerical evidence supports the conjectures that i) steady-state solutions can be spatially nonconstant, including the possibility of local extinction, and ii) smooth solutions may not exist for all time is provided. A discussion of some of the limitations of the Gurtin-MacCamy model motivates the consideration of a more general class of problems.

**Key words.** epidemics, Kermack-McKendrick model, nonlinear dispersion, degenerate diffusion, method of characteristics

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**1. Introduction.** Numerous mathematical challenges arise during the analysis of systems of reaction-diffusion equations with nonlinear diffusion. One example is the Gurtin–MacCamy model for the spread of an infectious disease. Some of the open questions are: Under what conditions do solutions exist for all time? What is the large-time qualitative behavior of solutions? These are the questions addressed in this paper. Unfortunately, no answers have been found. Instead, the analysis identifies some shortcomings of this particular model. These observations lead to the consideration of a new, and more general, class of problems.

The Kermack-McKendrick model is the classical model for the kinetics of an S–I–R epidemic. For this model the population is assumed to be subdivided into three classes: the susceptible ( $S$ ), infective ( $I$ ), and recovered ( $R$ ) populations. The assumptions are that susceptibles become infected by contact with infectives, infectives either die or recover at a constant rate, and the total population is constant. The sizes of the susceptible and infective populations therefore evolve according to the equations

$$(1.1) \quad \begin{aligned} \dot{S} &= -\alpha SI, \\ \dot{I} &= \alpha SI - \gamma I, \end{aligned}$$

where  $\alpha$  and  $\gamma$  are positive constants.

The qualitative properties of solutions to (1.1) are simple to describe. Given any positive initial data, the infectives always die out,  $I(t) \rightarrow 0$ , and the susceptibles tend to a positive steady-state,  $S(t) \rightarrow S_\infty$ , as  $t \rightarrow \infty$ . Moreover,  $S_\infty < \frac{\gamma}{\alpha}$ .

Extension of the Kermack-McKendrick model to populations in which the individuals have mobility within an environment have also been studied. Assuming the motion is governed by “random diffusion” in a one-dimensional habitat leads to the system of parabolic partial differential equations

$$(1.2) \quad \begin{aligned} S_t &= k_1 S_{xx} - \alpha SI, \\ I_t &= k_2 I_{xx} + \alpha SI - \gamma I, \end{aligned}$$

with the diffusion coefficients  $k_1$  and  $k_2$  positive and constant. (Subscripts of  $x$  and  $t$  are used to denote partial derivatives with respect to space and time, respectively.)

Webb [7] considered this problem when the environment is bounded,  $0 \leq x \leq L$ , and the diffusion coefficients are equal,  $k_1 = k_2 > 0$ . The assumption that individuals cannot cross the boundary leads to the use of homogeneous Neumann boundary conditions. The long-time behavior of solutions to (1.2) are the same as for the diffusion-free model: the infection disappears and the remaining susceptible population is homogeneously distributed throughout the environment. The behavior of solutions in the case with unequal diffusion coefficients is unknown; it is conjectured to be the same as for the equal coefficient case.

There are many other migration processes which can be considered. In Section 2 the Gurtin-MacCamy model is described and some properties of its solution are reviewed. Of particular interest is the possibility of spatially non-constant steady state susceptible populations. Existence of such solutions is demonstrated numerically in Section 3. One disadvantage of the Gurtin-MacCamy model is that an infection cannot spread beyond

its initial support. The overcrowding that occurs in this situation is one example of the manner in which smooth solutions may not exist for all time. Also included in Section 4 is a modification of the Gurtin-MacCamy model which overcomes this defect.

**2. The Gurtin-MacCamy Model.** The dispersion process incorporated into the Gurtin-MacCamy model is commonly referred to as *directed dispersion*. The basis for these models is adapted from fluid flow: each subpopulation is assumed to disperse with a well-determined velocity. A discussion of the applicability of these ideas to problems in population dynamics can be found in [2] and the references therein.

The Gurtin-MacCamy model is based upon the assumption that the susceptible population moves to avoid concentrations of infectives:  $v_S = kI_x$ , with  $k$  a positive constant, while the infectives have zero velocity:  $v_I = 0$ . The resulting system of PDEs are nonlinear and have “degenerate diffusion”:

$$(2.1) \quad \begin{aligned} S_t &= k(SI_x)_x - \alpha SI, \\ I_t &= \alpha SI - \gamma I. \end{aligned}$$

The initial data,

$$(2.2) \quad \begin{aligned} S(x, 0) &= S_0(x), \\ I(x, 0) &= I_0(x), \end{aligned}$$

and no-flux (i.e. homogeneous Neumann) boundary conditions,

$$(2.3) \quad \begin{aligned} S_x(0, t) &= S_x(L, t) = 0, \\ I_x(0, t) &= I_x(L, t) = 0, \end{aligned}$$

are assumed to be compatible.

*Remark.* Since the infectives do not move of their own volition,  $(2.1)_2$  is formally the same as  $(1.1)_2$ . However the interaction with the susceptibles does effect changes in the spatial distribution of the infectives, i.e.,  $I$  depends on both  $x$  and  $t$ .

*Remark.* The boundary conditions (2.3) are obtained from the no-flux conditions,

$$(kI_x S)|_{x=0} = (kI_x S)|_{x=L} = 0,$$

the equation  $(2.1)_2$ , and the positivity of  $S$  on the boundary (see Theorem 2.1).

The questions of positivity and local (in time) existence of solutions have been addressed in [4]. The proofs of these results are simple applications of general results in PDE theory. In each case the idea is to reformulate the original problem in a suitable form. These reformulations are the basis for the numerical methods used in subsequent sections to illustrate both the interesting steady-state solutions which can occur in this model and the overcrowding which can prevent the existence of smooth solutions for all time.

Positivity of the infective population is obvious from  $(2.1)_2$ . Since the maximum principle is no longer applicable there is some difficulty in showing that  $S > 0$ . The proof is based upon a change of variables introduced for the porous media equation

in [3]. Let  $S$  and  $I$  be a solution to (2.1), (2.2), and (2.3). Introduce the family,  $\{X(\cdot; p) : p \in [0, L]\}$ , of *characteristic curves* which satisfy the following initial value problem

$$(2.4) \quad X_t(t; p) = -kI_x(X(t; p), t), \quad X(0; p) = p.$$

Let  $\mathcal{S}(p, t)$  denote the susceptible population at time  $t$  along the characteristic curve originating from location  $p$ , i.e.  $\mathcal{S}(t, p) := S(X(t; p), t)$ . It can then be shown that  $\mathcal{S}$  satisfies a homogeneous first-order linear ODE with positive initial data, and hence must itself be positive. This is the basic idea in the proof of

**THEOREM 2.1.** *Let  $S$  and  $I$  be a classical solution of (2.1) with (2.2) and (2.3) on  $[0, L] \times [0, T)$ . If  $S_0 > 0$  and  $I_0 > 0$  on  $[0, L]$ , then  $S > 0$  and  $I > 0$  on  $[0, L] \times [0, T)$ .*

One consequence of this reformulation is the finite speed of propagation of the infection. This is in direct contrast with the infinite propagation speed for the random diffusion model. For many diseases, the controlled spread of infection is more reasonable. This topic will be discussed further in Section 4.

The positivity of solutions depends upon the existence of a solution. The existence of a smooth solution for all time remains unanswered. The following local (in time) result is the best that is presently available.

**THEOREM 2.2.** *Suppose  $S_0 > 0$  and  $I_0 > 0$  on  $[0, L]$ . Then there is a  $T > 0$  such that the problem (2.1), (2.2), (2.3) has a unique solution on  $[0, L] \times [0, T)$ .*

*Proof.* The proof is based upon the observation that the problem can be equivalently reformulated as a first-order hyperbolic system. Obtain a third equation for the system by introducing the “velocity”  $Q := I_x$  as an auxiliary variable; the corresponding equation for the evolution of  $Q$  is obtained by differentiating (2.1)<sub>2</sub> with respect to  $x$ . Then (2.1) can be rewritten as

$$(2.5) \quad U_t = A(U)U_x + B(U)U,$$

where

$$U = \begin{pmatrix} S \\ I \\ I_x \end{pmatrix}, \quad A(U) = \begin{pmatrix} kI_x & 0 & kS \\ 0 & 0 & 0 \\ \alpha I & 0 & 0 \end{pmatrix}, \quad B(U) = \begin{pmatrix} -\alpha I & 0 & 0 \\ 0 & \alpha S - \gamma & 0 \\ 0 & 0 & \alpha S - \gamma \end{pmatrix}.$$

The existence result follows from the local existence theory for hyperbolic systems and the fact that  $A(U)$  has distinct eigenvalues when the  $S$  and  $I$  are positive.  $\square$

The question of large-time existence is very intriguing. On the one hand it is known that solutions of nonlinear hyperbolic systems typically break down with the formation of shocks. On the other hand, as long as  $S < \frac{\gamma}{\alpha}$ , the term  $B(U)U$  provides dissipation. The conjecture is that a global solution will exist for sufficiently small initial data, but that shocks may form if the data are too large. The remainder of the discussion in this section assumes that solutions do exist for all time.

Solutions to the Gurtin-MacCamy model are seen to possess many of the same qualitative features as the original Kermack-McKendrick equations. One example is

the following local threshold phenomenon: if  $S(x_0, t_0) > \frac{\gamma}{\alpha}$  for some  $x_0 \in [0, L]$  and  $t_0 > 0$ , then the density of infectives at  $x_0$ ,  $I(x_0, \cdot)$ , increases momentarily from time  $t_0$ .

The large-time behavior of solutions to the Gurtin-MacCamy model is much richer than for the Kermack-McKendrick or random diffusion cases. The first observation is that, just like (1.1) and (1.2), the infection cannot persist for all time.

**THEOREM 2.3.** *If  $S = S(x)$  and  $I = I(x)$  form a time-independent steady-state solution to (2.1) and (2.3), then  $I \equiv 0$ .*

*Proof.* From (2.1)<sub>2</sub> it follows that either  $I = 0$  or  $S = \frac{\gamma}{\alpha}$ . But, if  $S = \frac{\gamma}{\alpha}$  then the partial differential equation (2.1)<sub>1</sub> with boundary conditions (2.3) imply that  $I = 0$ .  $\square$

Note that the degeneracy that arises when  $I \equiv 0$  removes all constraints on the steady-state distribution of the susceptible population. There is therefore no reason to expect a spatially homogeneous steady-state. In fact, the numerical evidence provided in Section 3 indicates that the susceptible population does not need to remain positive on the entire interval. That is, the population may exhibit local extinction (see Figure 1a).

**3. Numerical Examples.** Each of the two reformulations introduced in the previous section can be implemented numerically. After a brief description of each method, two examples are presented.

To implement the reformulation along characteristic curves, define  $\mathcal{I}(t, p) := I(X(t; p), t)$  and  $\mathcal{Z}(t, p) := S(X(t; p), t)X_p(t; p)$ . Replacing the spatial variable  $x$  with the parameter  $p$  leads to the first-order nonlinear system for the functions  $X$ ,  $\mathcal{Z}$ , and  $\mathcal{I}$  in  $(t, p)$ -space:

$$(3.1) \quad \left. \begin{aligned} X_t X_p + k \mathcal{I}_p &= 0, \\ \mathcal{Z}_t + \alpha \mathcal{I} \mathcal{Z} &= 0, \\ (\mathcal{I}_t + \gamma \mathcal{I})(X_p)^2 - \alpha \mathcal{I} \mathcal{Z} X_p + k (\mathcal{I}_p)^2 &= 0, \end{aligned} \right\} \quad 0 < p < L, t > 0.$$

The use of centered differences for the  $p$ -derivatives and Euler's method for the time-stepping leads to a simple second-order finite difference discretization of this system (and the corresponding initial and boundary conditions).

The time and space grids must be selected to ensure that the characteristics do not cross. This is difficult, if not impossible, to accomplish *a priori*. Oscillations in the solution tend to be greatest at the outset, when the spatial variation in the infection is greatest. As the infectives disappear, the dispersion becomes secondary to the reaction kinetics. Thus, assuming the initial migration is not too severe, the solution can be followed for all time.

Upwind differences can be used to discretize the hyperbolic system (2.5). The success of this method depends upon the existence of a basis of left eigenvectors for  $A(U)$ . The only problem is when  $I = 0$ . Thus, this approach cannot be used in situations in which there are regions with no infection (see the example in Section 4).

Stability of this algorithm is guaranteed when the grid sizes satisfy the C-F-L condition:

$$\left( \sup_{0 \leq x \leq L} \max_{i=1,2,3} |\lambda_i(U)| \right) \frac{\Delta t}{\Delta x} < 1,$$

where the eigenvalues of  $A$  are denoted by  $\lambda_i$  and  $\Delta t$  and  $\Delta x$  denote the sizes of the time- and space-grids.

The results from two examples are presented. The only difference between the two examples is the size of the dispersion coefficient. The case with  $k = 1$  is referred to as “rapid” dispersion while  $k = 0.1$  will be called “slow” dispersion. Snapshots of the distribution of susceptible and infective populations at different times are shown in Figures 1 and 3; the steady-state distributions are indicated with  $\times$ 's. Figures 2 and 4 show the characteristic curves for the two different values of the dispersion coefficient.

The numerical findings are consistent with the aforementioned restrictions. The two numerical schemes produce comparable results for small time; however, the oscillations in the characteristics are extremely difficult to track using the characteristic method. The output from the C–I–R method (with  $\Delta x = 0.05$  and  $\Delta t = 0.004$ ) is used to produce Figures 1–4. The characteristic curves are computed a posteriori by numerical differentiation of the infective distribution.

From initial distributions of  $S_0(x) := 6$  and  $I_0(x) := 3 - \cos^5(\pi x)$  the susceptibles are seen to be moving to avoid concentrations of the infectives. Moreover, with  $\alpha = 1$  and  $\gamma = 5$ , the expected threshold effect is observed in the infective population. As the infection decays to zero the oscillations in the susceptibles subside.

The steady-state susceptible distributions are obviously non-constant. In fact, in the high dispersion case the initial avoidance of the infection was so strong as to produce a region of extinction (see Figure 1a). The diversity of the steady-state solutions is strikingly different from previous models. This richness is very intriguing, but not yet completely understood.

**4. Overcrowding.** It is also interesting to consider situations in which the initial distribution of the infection is compactly supported. Recall that the infection disperses with finite speed. However, further examination of the defining equation for the characteristics (2.4) shows that if  $I(x_0, t_0) = 0$  then  $I(x_0, t) = 0$  for all  $t > t_0$ . In particular, the infection cannot spread beyond its initial support.

One consequence of this is that each susceptible individual ceases to move as soon as an infection-free location is reached. In this manner it is possible to escape the disease. Since there are no other forces pushing the susceptibles away from the boundary of the support of the infection, overcrowding can occur. The segregation of the habitat that results from this overcrowding is one manner in which the discontinuities in the solution can arise in finite time.

To illustrate this phenomenon consider the low-dispersion model ( $k = 0.1$ ) in which the initial distribution of infectives is a smooth function with support on  $[0, 0.8]$ . Because of the compact support of  $I$ , the C–I–R method cannot be applied; Figures 5 and 6 are produced with the results of the characteristic method. In Figure 6 note that the characteristics accumulate at  $x = 0.8$ , the boundary of the support of the infection. In fact, all evidence suggests that characteristics actually coalesce in finite time.

There is biological evidence which indicates that some species migrate to sparsely populated regions even at the expense of necessary resources [1], [2]. Migration to avoid overcrowding can be modelled as directed dispersion. To modify the Gurtin-

MacCamy model to include this second source of dispersion let  $N := S + I + R$  denote the distribution of the total population. An equation for  $N$  is obtained from the sum of the modified equations for  $S$ ,  $I$ , and  $R$ . The resulting set of equations is

$$(4.1) \quad \begin{aligned} S_t &= k_1(SI_x)_x + k_2(SN_x)_x - \alpha SI, \\ I_t &= k_2(IN_x)_x + \alpha SI - \gamma I, \\ R_t &= k_2(RN_x)_x - \gamma I, \\ N_t &= k_1(SI_x)_x + k_2(NN_x)_x. \end{aligned}$$

Note that the equation for  $N$  is simply the sum of the equations for  $S$ ,  $I$ , and  $R$ . Thus, only three of the above equations is needed to form a well-defined problem. Just as in the earlier models, it seems that the equation for  $R$  is of least use to the analysis, and can be dropped.

Initial results for special cases of (4.1) have been obtained by Meade and Milner [5], [6]. The qualitative behavior of solutions to (4.1) have many of the same features as the Gurtin-MacCamy model. The infection can and does spread beyond its initial support. Moreover, spatially non-constant steady-state solutions are still possible. The analysis of this new model is assisted by its connection with the porous media equation. This observation will be exploited in the ongoing analysis of the problem.

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#### References.

- [1] E. A. CARL, *Population control in arctic ground squirrels*, Ecology, 52, pp. 395–413.
- [2] W. GURNEY AND R. NISBET, *The regulation of inhomogeneous populations*, J. Theoret. Biol., 52 (1975), pp. 441–457.
- [3] M. E. GURTIN, R. C. MACCAMY, AND E. SOCOLOVSKY, *A coordinate transformation for the porous media equation that renders the free-boundary stationary*, Quart. Appl. Math., 42 (1984), pp. 345–357.
- [4] R. C. MACCAMY AND D. B. MEADE, *An epidemic model with directed diffusion*, in Biomedical Modelling and Simulation, J. Eisenfeld and D. S. Levine, eds., IMACS, Paris, 1989, pp. 197–199.
- [5] D. B. MEADE AND F. A. MILNER, *S–I–R epidemic models with directed diffusion*, 1991, Technical Report #158 (Center for Applied Mathematics, Purdue University).
- [6] ———, *An S–I–R model for epidemics with diffusion to avoid infection and overcrowding*, in Proceedings of the 13th IMACS World Congress on Computation and Applied Mathematics (v. 3), R. Vichnevetsky and J. J. H. Miller, eds., IMACS, Dublin, Ireland, 1991, pp. 1444–1445.
- [7] G. WEBB, *An age-dependent epidemic model with spatial diffusion*, Arch. Rational Mech. Anal., 75 (1980), pp. 91–102.

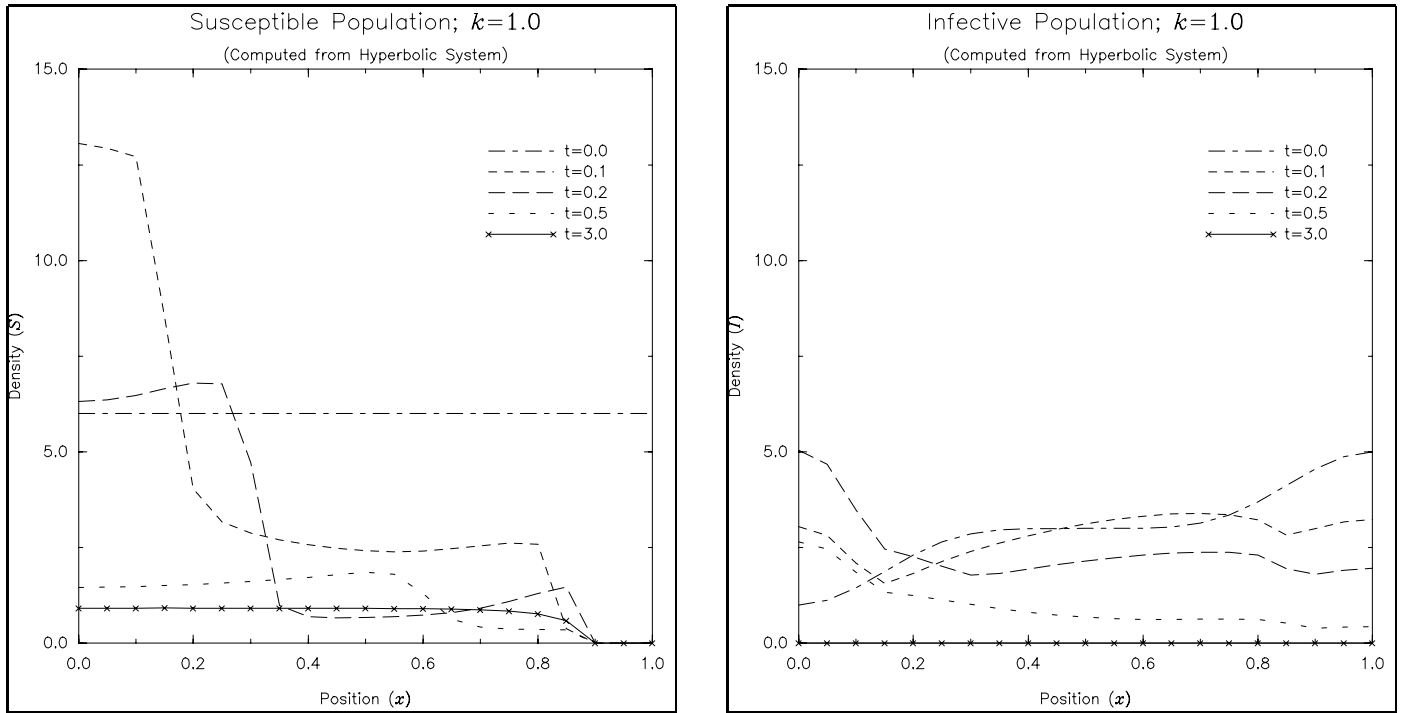


FIG. 1. Susceptible (a) and infective (b) population densities for rapid dispersion,  $k = 1.0$ .

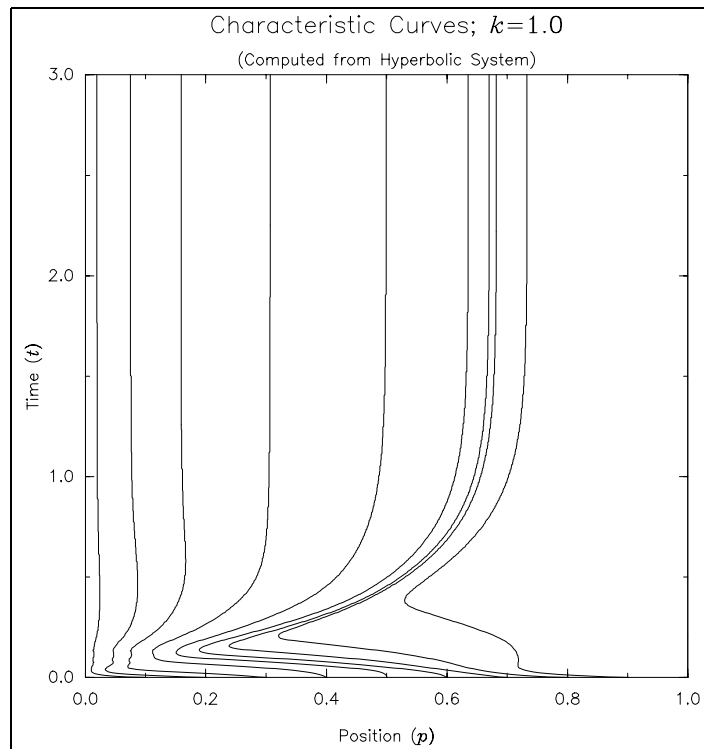


FIG. 2. Characteristic curves for rapid dispersion,  $k = 1.0$ .

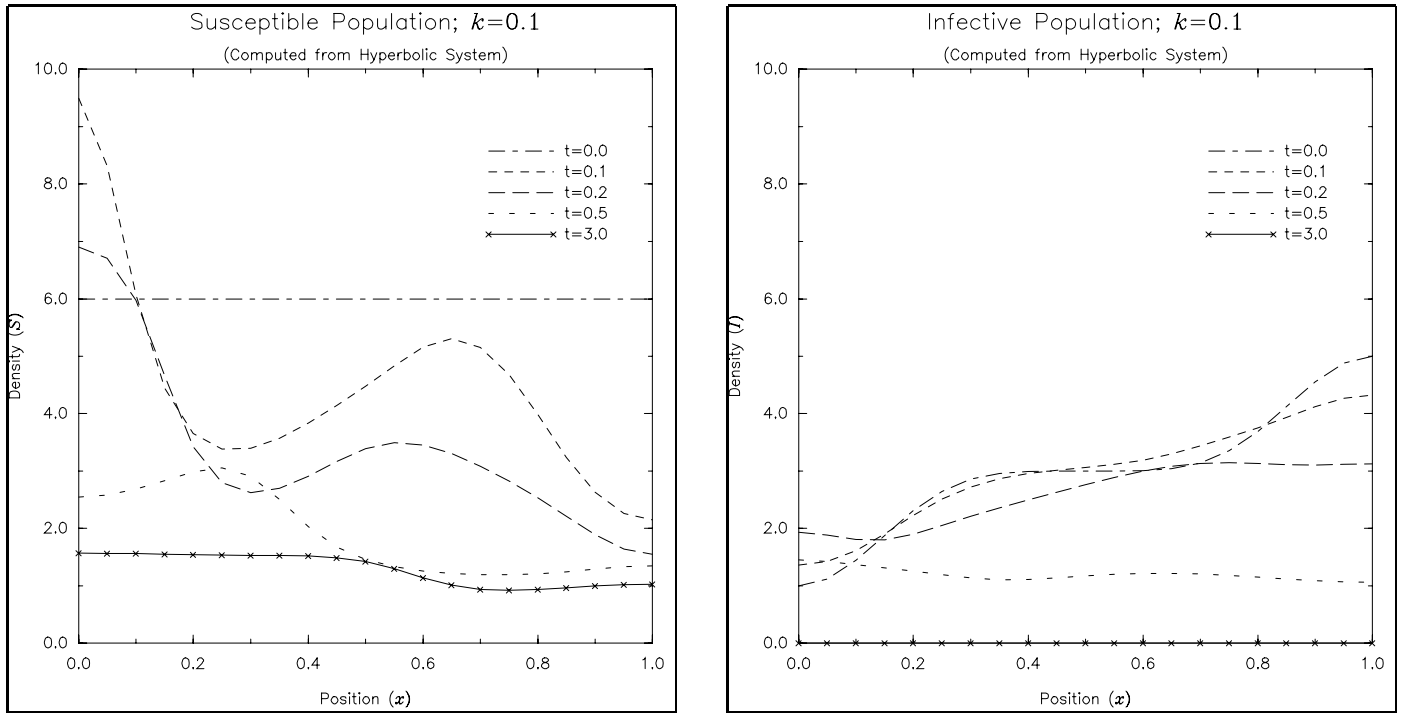


FIG. 3. Susceptible (a) and infective (b) population densities for slow dispersion,  $k = 0.1$ .

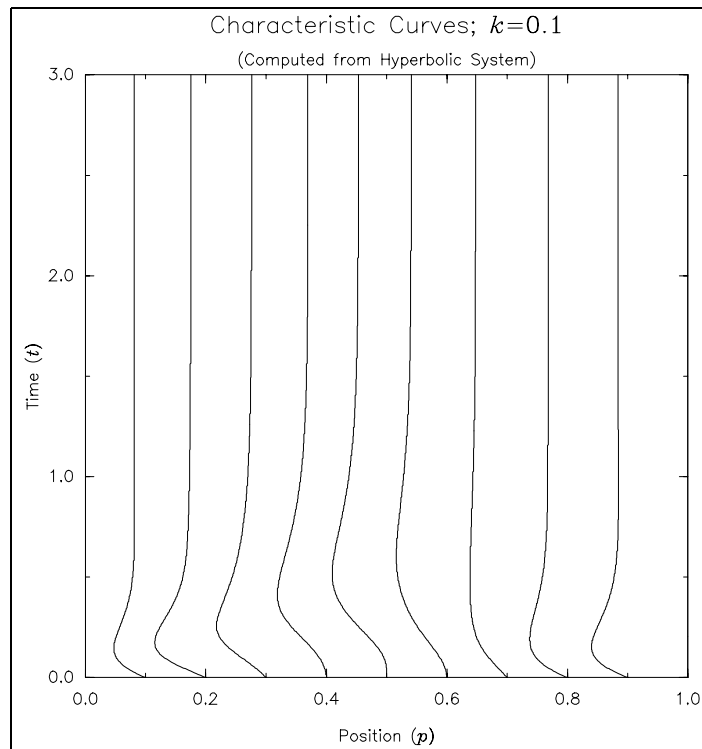


FIG. 4. Characteristic curves for slow dispersion,  $k = 0.1$ .

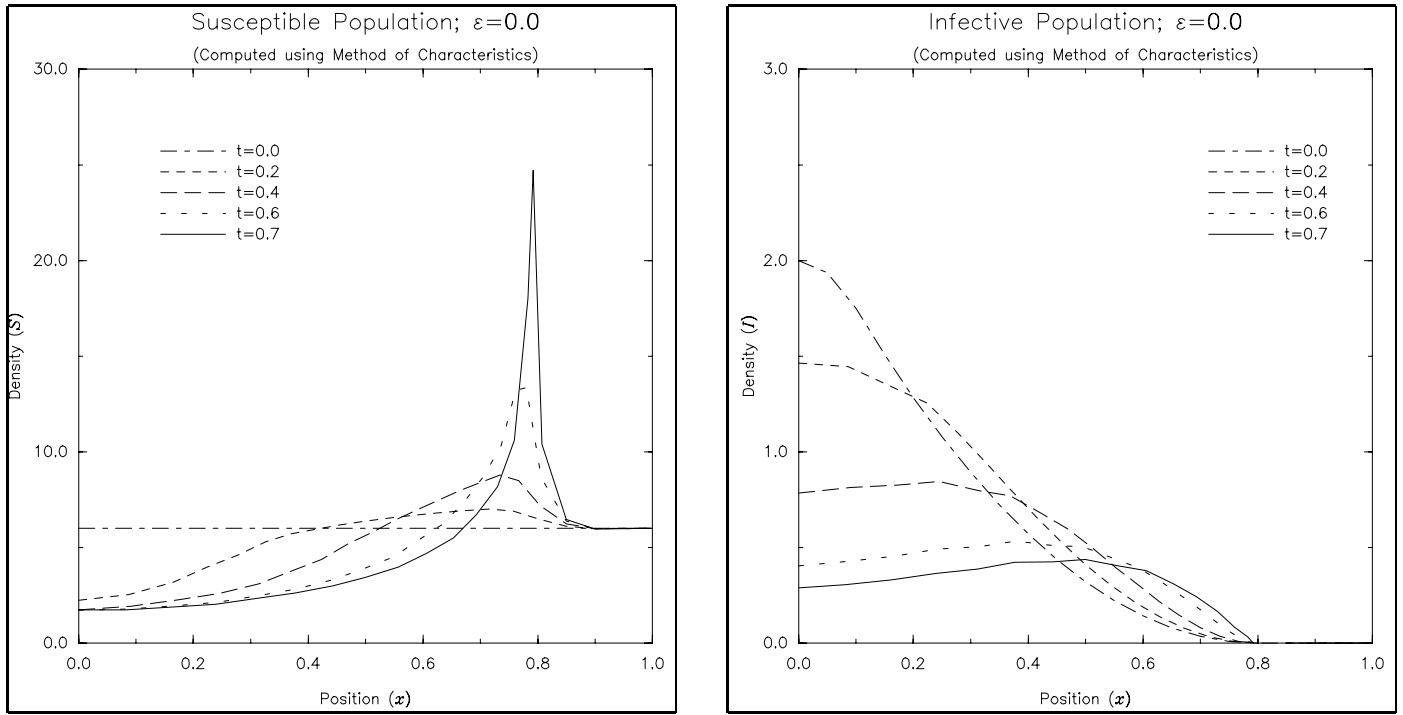


FIG. 5. Susceptible (a) and infective (b) population densities with compact support.

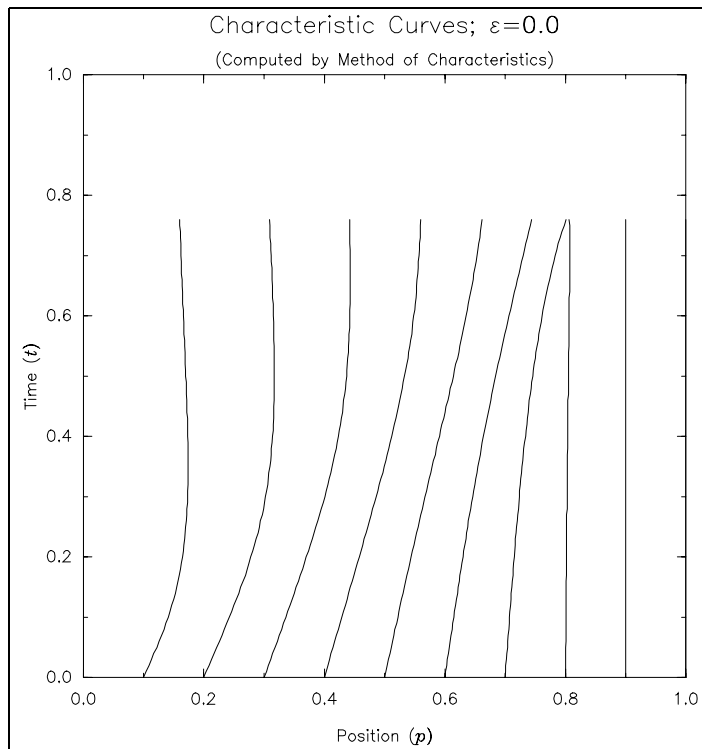


FIG. 6. Characteristic curves for an infection with compact support.