

**THE ASYMPTOTIC BEHAVIOR OF  
A REDUCIBLE SYSTEM  
OF NONLINEAR INTEGRAL EQUATIONS**

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**ABSTRACT.** The methods developed in this paper are motivated mainly by the study of models for rabies. Rabies is a multispecies disease in which the virulence of the virus, and its affect on different species, leads to models where the infection matrix is reducible.

The asymptotic behavior of a reducible system of nonlinear integral equations describing the spatio-temporal development of such an epidemic is studied. When the system is nonreducible, an approximate saddle point method can be used for a restricted model with constant infection and removal rates. This approximate method [17] indicated that the asymptotic speed of propagation is  $c_0$ , the minimum wave speed. A rigorous analytic proof of this result was given subsequently in Radcliffe and Rass [18].

A reducible set of types may be considered as split into nonreducible subsets of types, so that within each subset all types may infect every other type, possibly through a series of infections. For any two subsets, infection in at least one subset cannot cause infection to occur in the other subset. Consider an infection in the  $i$ th subset only, the density of types in the other subsets being taken to be zero. Let  $c_i$  be the corresponding asymptotic speed of propagation. Then, for the full system, the asymptotic speed of propagation differs for the different subsets. Each subset infected will force the epidemic in any subset it infects to propagate with at least its speed of propagation. The approximate saddle point method was again used for the restricted reducible model [20]. It indicated that, for a particular subset, the asymptotic speed of propagation is the maximum of the  $c_i$  over all subsets  $i$  which can cause an infection in the particular subset, and which can themselves be infected by the initial infection in the system.

In this paper, with certain conditions imposed, a rigorous proof of these results is obtained for the general reducible model. It is remarkable that these conditions cover not only all cases in which the saddle point method can be applied but

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also additional cases. A lower bound is also established for the final size of the epidemic for each type, the lower bound holding for all values of the spatial variable.

**1. Introduction.** Spatial models have been used to study the spread of rabies [12, 5]. One method of attempting to control the spread of the disease is to reduce the density of animals in a protective belt ahead of the wave front. Spatial models have been used to estimate the width of the belt and reduction in density necessary to stop the epidemic [24, 13].

Rabies is a multispecies disease which affects many animals, e.g., foxes, badgers, raccoons, opossums, mongooses, bats, skunks, and in an epidemic up to 20 species may be involved [10, 6]. Certain species act as reservoirs of infection with the disease spilling over into other species and also domestic animals [6, 9, 14, 4].

Two species in which rabies is prevalent are foxes and striped skunks. However, two-way transmission of the disease between species does not usually occur. The reasons suggested, [22], are differences in amount of the virus excreted in the saliva, and differences in the susceptibility of the species. The amount of virus in the saliva of infected skunks is much greater than that in infected foxes. Foxes are more susceptible to the virus, especially in small quantities, than striped skunks, [21]. Thus, when the virulence of the virus is low, foxes excrete too little virus to infect striped skunks, but can infect other foxes. However, striped skunks can infect both skunks and foxes. With high virulence striped skunks excrete a quantity of virus that kills foxes before they become infectious, i.e., before they excrete virus in their saliva. The transference of the virus from one species to the other will depend therefore on the virulence of the virus [6]. Lower virulence leads to a reducible model with transmission from skunks to foxes. Higher virulence leads to a reducible model with transmission from foxes to skunks. Differences between strains of rabies virus in the hosts are discussed in Wiktor et al. [26]. The argument extends to include other species.

The study of Friend [10] of the New York State rabies epizootic involved at least 20 species. It was found that, when comparing the distribution of cases in two species, those for some pairs of species were correlated but those for other pairs were not. This is consistent with

the spread of infection when the model is reducible, but not when it is nonreducible.

In order to model the spatial spread of rabies and analyze methods of attempting to control its spread, it is appropriate to develop a theory of the spatial spread of  $n$ -type epidemics in which the infection matrix is reducible. A first step is to consider the reducible model discussed in the present paper.

Rabies is the disease which motivated the study of the effect of reducibility. However, it is likely to be relevant to models for other diseases. A rather specialized case, in which the spread in one population is completely forced by the other population, occurs when infection is spread by carriers. The case of 'Typhoid Mary' is discussed in Turyn [25]. A carrier epidemic, in which the individuals for whom the disease is apparent, are removed immediately and are therefore unable to contribute to the spread of infection, is considered in Clancy [7].

This paper addresses the problem of the effect of reducibility on the spread of infection in an  $n$  type epidemic in a closed homogeneous population. The velocity of propagation has been studied for a one-type epidemic with radially symmetric contact distributions by Aronson [1] and Diekmann [8]. Thieme [23] also looks at the speed of spread of populations. An approximate saddle point method was used by Radcliffe and Rass [17] to treat the  $n$ -type nonreducible model. This was for a restricted model having constant infectivity and removal rates for each population. This method suggested that the asymptotic speed of propagation is  $c_0$ , the minimum wave speed. A rigorous proof of these results for the general  $n$ -type nonreducible model was given in Radcliffe and Rass [18].

Wave solutions for a reducible epidemic have been considered by Radcliffe and Rass [19]. Some interesting results were obtained regarding the existence of waves at different speeds. In particular cases, a multiplicity of waves is possible at certain speeds, in contrast to the nonreducible case [15] where waves (if they exist at a specific speed) are unique modulo translation. Results on wave solutions have been established in all but an exceptional case.

In this paper, attention is focused on the speed of propagation and the pandemic theorem. In order to avoid unnecessary complexity, we prove the results for a simple form of reducible epidemic. In such an

epidemic, the types can be split into two groups. No cross infection can occur from infectious individuals in the second group of types to any susceptible individuals amongst the first group of types. However, cross infection occurs in the reverse direction, namely, there exists at least one type in group 1 which can cause an infection of at least one type of susceptible in group 2. Within groups the epidemic is nonreducible, so that any type can infect any other type, possibly through a sequence of infections. Again, we only consider contact distributions which are radially symmetric.

We regard an infection for a certain type as having asymptotic speed of propagation  $c$ , when radiating out from an initial focus of infection, if both the following results hold. Firstly, if an individual sets out at a speed greater than  $c$  from the focus of infection, he will eventually leave the epidemic behind. Secondly, if he travels out from the focus of infection at a speed less than  $c$ , he will be eventually surrounded by the epidemic.

Consider an infection that occurs amongst types in group 2 only. The epidemic will not affect group 1 types. The speed of propagation of the epidemic amongst group 2 types is then the minimal wave speed for waves amongst group 2 types only.

If the initial infection involves at least one type in group 1, then infection will occur amongst all types. The asymptotic speed of propagation of infection in a type will depend on the group that it is in. For a type in group 1, this speed will be  $c_1$ , and for a type in group 2, the speed will be  $\max(c_1, c_2)$ . Here  $c_i$  denotes the speed of propagation that would occur for types in group  $i$  if the density of types in the other group were taken to be zero. There are restrictions under which these results are obtained. These conditions are in fact more general than those for which the saddle point method can be applied.

Results are also obtained for the final size of an epidemic initiated amongst types, at least one of which is in group 1. A lower bound is obtained for the eventual proportion of the population of each type in both groups eventually suffering the infection. This is the pandemic theorem.

Once results are proved for this simple form of reducible epidemic, with careful thought the extension to a general reducible form can be seen. Formal proofs for the general case do not present any extra

difficulty.

It is interesting to note that the use of saddle point methods suggests that the assumption of radial symmetry of the contact distributions is not necessary. For precise details of the saddle point results, we refer the reader to Radcliffe and Rass [17, 20].

**2. The model and specification of the problem.** Consider  $n$  populations of types each of uniform density in  $\mathbf{R}^N$ . In a similar manner to our paper [18] we are led to consider the equations

$$(2.1) \quad w_i(\mathbf{s}, t) = \sum_{j=1}^n \int_{\mathbf{R}^N} \int_0^t (1 - \exp\{-w_j(\mathbf{s} - \mathbf{r}, t - \tau)\}) p_{ij}(\mathbf{r}) \gamma_{ij}(\tau) d\tau d\mathbf{r} \\ + H_i(\mathbf{s}, t), \quad i = 1, \dots, n.$$

Here  $w_i(\mathbf{s}, t) = -\log x_i(\mathbf{s}, t)$ , where  $x_i(\mathbf{s}, t)$  is the proportion of individuals of type  $i$  at position  $\mathbf{s}$  who are susceptible at time  $t$ . Also  $\gamma_{ij}(\tau) = \sigma_j \lambda_{ij}(\tau)$  where  $\sigma_j$  is the density of type  $j$  individuals and  $\lambda_{ij}(\tau)$  is the rate of infection of susceptible individuals of type  $i$  from infected individuals of type  $j$  who were infected time  $\tau$  ago. The  $p_{ij}(\mathbf{r})$  denote the contact distributions representing the distance  $\mathbf{r}$  over which infection occurs.

$H_i(\mathbf{s}, t)$  is a term representing the effect of the infection from outside which initiates the epidemic. If  $H_i(\mathbf{s}, t) \equiv 0$  for all  $i$  in group 1, then  $w_i(\mathbf{s}, t) \equiv 0$  for all  $i$  in group 1, i.e., the epidemic occurs in group 2 only. In this case the results for the asymptotic speed of propagation and the pandemic theorem follow immediately from our paper [18]. If  $H_i(\mathbf{s}, t) \not\equiv 0$  for some  $i$  in group 1, some  $\mathbf{s}$ , and some  $t > 0$ , the epidemic occurs in both groups. We only therefore consider this case. We refer the reader to our paper [18] for a complete specification of the model. Note that, as the infection is initiated from outside the  $n$  populations of types  $x_i(\mathbf{s}, 0) \equiv 1$ , so that  $w_i(\mathbf{s}, 0) \equiv 0$ . Certain conditions are imposed. Each  $p_{ij}(\mathbf{r})$  is restricted to be a bounded continuous radial function in  $\mathbf{R}^N$  such that  $P_{ij}(\lambda) = \int_{\mathbf{R}^N} \exp(\lambda\{\mathbf{r}\}_1) p_{ij}(\mathbf{r}) d\mathbf{r}$  exists for some positive real  $\lambda$ . Here  $\{\mathbf{r}\}_1$  denotes the first entry of the vector  $\mathbf{r}$ . Also  $\gamma_{ij}(\tau)$  is taken to be bounded with continuous bounded derivative. Conditions are imposed on the infection from outside which are essentially the same as in our paper [18]. The infection from outside is in a bounded region. The infectious influence decreases sufficiently quickly so that

the integrals of the infection rates are finite. The Laplace transform of the contact distribution for infection by an infectious individual from outside of a susceptible individual of type  $i$  exists for all real positive  $\lambda$  for which the  $P_{ij}(\lambda)$  exist for  $j = 1, \dots, n$ . Thus, the infection from outside does not spread the infection faster than it is spread by infection within the  $n$  populations of types. We refer the reader to our paper [18] for precise details. In this paper, we specify the conditions in terms of  $H_i(\mathbf{s}, t)$ , this specification being given in Section 3.

Let  $\gamma_{ij} = \int_0^\infty \gamma_{ij}(\tau) d\tau$ . This paper differs from [18] in that  $\Gamma = (\gamma_{ij})$  is no longer nonreducible. For simplicity we only consider the case where  $\gamma_{ij}$  is finite for all  $i$  and  $j$ . The population of  $n$  types is taken to consist of two groups of  $n_1$  and  $n_2$  types, respectively. Within a group, infection can occur between any types, possibly through a sequence of infections. No type in group 2 can infect a type in group 1; but at least one type in group 1 can infect some type in group 2. Hence, by re-ordering the types so that  $\Gamma$  is in normal form, (see [11]),  $\Gamma$  may be partitioned so that

$$\Gamma = \begin{pmatrix} \mathbf{\Gamma}_{11} & 0 \\ \mathbf{\Gamma}_{21} & \mathbf{\Gamma}_{22} \end{pmatrix},$$

where  $\mathbf{\Gamma}_{11}$  and  $\mathbf{\Gamma}_{22}$  are nonnegative, nonreducible square matrices of sizes  $n_1$  and  $n_2$ , respectively, where  $n_1 + n_2 = n$ : and  $\mathbf{\Gamma}_{21}$  is nonnegative and is not identically zero.

We are concerned with nonnegative solutions  $w_i(\mathbf{s}, t)$  of equations (2.1), which are monotone increasing in  $t$  with  $w_i(\mathbf{s}, 0) \equiv 0$ ; and it is easily shown that such solutions have the property that  $w_i(\mathbf{s}, t)$  is continuous in  $t$  uniformly with respect to  $\mathbf{s}$  for  $t \in [0, \infty)$ .

The asymptotic speed of propagation, as defined by Aronson and Weinberger [2, 3] is  $c$  if for any  $c_1$  and  $c_2$  with  $0 < c_1 < c < c_2$ ,

(i) the solution  $w_i(\mathbf{s}, t)$  tends uniformly to zero in the region  $|\mathbf{s}| \geq c_2 t$ ,

(ii) the solution  $w_i(\mathbf{s}, t)$  is bounded away from zero uniformly in the region  $|\mathbf{s}| \leq c_1 t$  for  $t$  sufficiently large.

Section 3 establishes results concerning part (i) of the definition for the different groups. Section 4 obtains results relating to part (ii). These results are then amalgamated and summarized in Section 5, and the results are linked to those of the saddle point method and the wave solutions. In Section 6 we consider what happens if a certain

condition does not hold. The results are then extended to the general reducible case. Section 7 establishes the pandemic theorem for the simple reducible epidemic with two groups and gives the extension of the results to the general reducible case.

**3. Obtaining an upper bound for the asymptotic speed of propagation.** Let  $\Lambda_{ij}(\lambda) = \int_0^\infty e^{-\lambda\tau} \gamma_{ij}(\tau) d\tau$ . Define  $V_{ij}(c, \lambda) = P_{ij}(\lambda) \Lambda_{ij}(c\lambda)$  and  $\{\mathbf{V}(c, \lambda)\}_{ij} = V_{ij}(c, \lambda)$ .

For a matrix  $\mathbf{A}$  of finite elements,  $\rho(\mathbf{A})$  is the maximum of the moduli of the eigenvalues of  $\mathbf{A}$ . Let  $\mathbf{V}(c, \lambda)$  be partitioned by groups so that

$$\mathbf{V}(c, \lambda) = \begin{pmatrix} \mathbf{V}_{11}(c, \lambda) & 0 \\ \mathbf{V}_{21}(c, \lambda) & \mathbf{V}_{22}(c, \lambda) \end{pmatrix},$$

and  $K_1(c, \lambda) = \rho(\mathbf{V}_{11}(c, \lambda))$  and  $K_2(c, \lambda) = \rho(\mathbf{V}_{22}(c, \lambda))$ . Let  $\Delta_{V_{ij}}$  be the abscissa of convergence of  $P_{ij}(\lambda)$  in the positive half of the complex plane. For simplicity, we restrict each  $P_{ij}(\lambda)$  to be infinite at its abscissa of convergence. Let  $\Delta_{11} = \min\{\Delta_{V_{ij}} \text{ for } i, j = 1, \dots, n_1\}$ ,  $\Delta_{22} = \min\{\Delta_{V_{ij}} \text{ for } i, j = n_1 + 1, \dots, n\}$ , and  $\Delta_{21} = \min\{\Delta_{V_{ij}} \text{ for } i = 1, \dots, n_1, j = n_1 + 1, \dots, n\}$ . We restrict the  $p_{ij}(\mathbf{r})$  so that  $\Delta_{21} \geq \Delta_V$  where  $\Delta_V = \min\{\Delta_{11}, \Delta_{22}\}$ .

If  $\rho(\mathbf{\Gamma}_{ii}) > 1$ ,  $c_i = \inf\{c > 0 : K_i(c, \lambda) = 1 \text{ for some } \lambda \in (0, \Delta_{ii})\}$ . Then for  $c \geq c_i$  we define  $\lambda = \alpha_i(c)$  to be the smallest positive root of  $K_i(c, \lambda) = 1$ . This is the only root if  $c = c_i > 0$ . Define  $\alpha_i^*(c_i) = \alpha_i(c_i)$ . If  $c > c_i$  there is a second positive root which we define to be  $\alpha_i^*(c)$ . There is no positive root of  $K_i(c, \lambda) = 1$  if  $0 < c < c_i$ .

If  $\rho(\mathbf{\Gamma}_{ii}) \leq 1$ ,  $c_i = 0$ , and for each  $c > 0$  there is a single positive root of  $K_i(c, \lambda) = 1$ . We define this root as  $\alpha_i^*(c)$ .

If  $\rho(\mathbf{\Gamma}_{22}) \geq 1$ , then we impose the condition that  $\alpha_1^*(\max(c_1, c_2)) \geq \alpha_2(\max(c_1, c_2))$ .

$H_i(\mathbf{s}, t)$  is assumed to be monotone increasing in  $t$ , continuous in  $\mathbf{s}$  and  $t$ , and uniformly bounded. Also  $H_i(\mathbf{s}, t) > 0$  for some  $\mathbf{s} \in \mathbf{R}^N$ ,  $t > 0$  and some  $i = 1, \dots, n_1$ . In order for the infection from outside to trigger, but not dominate the infection in the populations under consideration, we impose the condition that  $H_i(\mathbf{s}, t) \exp(\lambda(\{\mathbf{s}\}_1 - ct)) \leq D_i(\alpha)$ , for  $0 \leq \lambda \leq \alpha$  for any  $\alpha$  such that  $\alpha < \Delta_{11}$  if  $i = 1, \dots, n_1$ , and for any  $\alpha$  such that  $\alpha < \Delta_{22}$  if  $i = n_1 + 1, \dots, n$ .

**Theorem 1.** (i) *There exists a nonnegative, monotone increasing (in  $t$ ) solution  $w_i(\mathbf{s}, t)$  to equation (2.1) with  $w_i(\mathbf{s}, 0) \equiv 0$ ,  $i = 1, \dots, n$ , which is unique.*

(ii) *For any  $c^* > 0$  such that  $K_1(c^*, \lambda^*) < 1$  for some  $\lambda^* \in (0, \Delta_{11})$ ,*

$$\limsup_{t \rightarrow \infty} \{w_i(\mathbf{s}, t) : |\mathbf{s}| \geq c^*t\} = 0 \quad \text{for } i = 1, \dots, n_1.$$

(iii) *For any  $c^* > 0$  such that  $K_1(c^*, \lambda^*) < 1$  and  $K_2(c^*, \lambda) < 1$  for some  $\lambda$  and  $\lambda^*$  where  $0 < \lambda < \lambda^* < \Delta_V$ ,*

$$\limsup_{t \rightarrow \infty} \{w_i(\mathbf{s}, t) : |\mathbf{s}| \geq c^*t\} = 0 \quad \text{for } i = n_1 + 1, \dots, n.$$

*Proof.* (i) For any  $\rho(\mathbf{\Gamma})$ , there exists a  $c > 0$  and a  $\lambda$  and  $\lambda^*$  where  $0 < \lambda < \lambda^* < \Delta_V$  such that  $K_1(c, \lambda^*) < 1$  and  $K_2(c, \lambda) < 1$  and  $\mathbf{V}_{21}(\lambda^*)$  and  $H_i(\mathbf{s}, t) \exp(\lambda(\{\mathbf{s}\}_1 - ct))$  are finite for all  $i, j$ . Take such a  $c, \lambda$  and  $\lambda^*$  and define  $y_i(\mathbf{s}, t) = w_i(\mathbf{s}, t) \exp(\lambda^*(\{\mathbf{s}\}_1 - ct))$  for  $i = 1, \dots, n_1$  and  $y_i(\mathbf{s}, t) = w_i(\mathbf{s}, t) \exp(\lambda(\{\mathbf{s}\}_1 - ct))$  for  $i = n_1 + 1, \dots, n$ .

To construct and prove the uniqueness of a solution to equations (2.1) for  $i = 1, \dots, n_1$ , i.e., for group 1 types, we proceed exactly as in Theorem 1 part (i) of our paper [18]. Note that in the proof of part (ii) of Theorem 1 of that paper, it was shown that  $y_i(\mathbf{s}, t) \leq D_i^*$  and hence  $w_i(\mathbf{s}, t) \leq D_i^* \exp(-\lambda^*(\{\mathbf{s}\}_1 - ct))$  for  $\mathbf{s} \in \mathbf{R}^N$ ,  $t \geq 0$  and  $i = 1, \dots, n_1$ .

Now consider the construction of a solution to equations (2.1) for  $i = n_1 + 1, \dots, n$ . Define

$$\begin{aligned} y_i^{(0)}(\mathbf{s}, t) &= \sum_{j=1}^{n_1} \int_{\mathbf{R}^N} \int_0^t [1 - \exp\{-w_j(\mathbf{s} - \mathbf{r}, t - \tau)\}] \\ &\quad \times \exp(\lambda(\{\mathbf{s} - \mathbf{r}\}_1 - c(t - \tau)) \\ &\quad \times [\gamma_{ij}(\tau) e^{-\lambda c \tau}] [p_{ij}(\mathbf{r}) \exp(\lambda\{\mathbf{r}\}_1)] d\tau d\mathbf{r} \\ &+ H_i(\mathbf{s}, t) \exp(\lambda(\{\mathbf{s}\}_1 - ct)), \end{aligned}$$



and recursively for  $m = 0, 1, \dots$

$$y_i^{(m+1)}(\mathbf{s}, t) = \sum_{j=n_1+1}^n \int_{\mathbf{R}^N} \int_0^t [1 - \exp\{-y_j^{(m)}(\mathbf{s} - \mathbf{r}, t - \tau) \\ \times \exp(\lambda(\{\mathbf{s} - \mathbf{r}\}_1 - c(t - \tau)))\}] \\ \times [\exp(\lambda(\{\mathbf{s} - \mathbf{r}\}_1 - c(t - \tau))] \\ \times [\gamma_{ij}(\tau)e^{-\lambda c\tau}] [p_{ij}(\mathbf{r}) \exp(\lambda\{\mathbf{r}\}_1)] d\tau d\mathbf{r} \\ + y_i^{(0)}(\mathbf{s}, t)$$

for  $\mathbf{s} \in \mathbf{R}^N$ ,  $t \geq 0$  and  $i = n_1 + 1, \dots, n$ .

Now  $H_i(\mathbf{s}, t) \exp(\lambda(\{\mathbf{s}\}_1 - ct)) \leq D_i(\lambda)$  for  $i = n_1 + 1, \dots, n$ .

Also for  $\{\mathbf{u}\}_1 \leq c\theta$ ,  $[1 - \exp\{-w_j(\mathbf{u}, \theta)\}] \exp(\lambda(\{\mathbf{u}\}_1 - c\theta)) \leq 1$  for  $j = 1, \dots, n_1$ .

When  $\{\mathbf{u}\}_1 > c\theta$ , then for  $j = 1, \dots, n_1$ ,  $[1 - \exp\{-w_j(\mathbf{u}, \theta)\}] \exp(\lambda(\{\mathbf{u}\}_1 - c\theta)) \leq w_j(\mathbf{u}, \theta) \exp(\lambda(\{\mathbf{u}\}_1 - c\theta)) \leq D_j^* \exp(-(\lambda^* - \lambda)(\{\mathbf{u}\}_1 - c\theta)) \leq D_j^*$ .

Thus  $y_i^{(0)}(\mathbf{s}, t) \leq D_i(\lambda) + \sum_{j=1}^{n_1} \max(1, D_j^*) P_{ij}(\lambda) \Gamma_{ij}(c\lambda) = B_i$  say, for  $\mathbf{s} \in \mathbf{R}^N$ ,  $t \geq 0$  and  $i = n_1 + 1, \dots, n$ .

If we let  $u_i^{(m)} = \sup |y_i^{(m+1)}(\mathbf{s}, t) - y_i^{(m)}(\mathbf{s}, t)|$ , where the sup is taken over  $\mathbf{s} \in \mathbf{R}^N$  and  $t \geq 0$ , for  $m \geq 0$  and  $i = n_1 + 1, \dots, n$ , we can proceed exactly as in the proof of Theorem 1 part (i) of our paper [18] to show that  $y_i(\mathbf{s}, t) = \lim_{m \rightarrow \infty} y_i^{(m)}(\mathbf{s}, t)$  exists for  $i = n_1 + 1, \dots, n$ , and satisfies the equations

$$y_i(\mathbf{s}, t) = \sum_{j=1}^n \int_{\mathbf{R}^N} \int_0^t [1 - \exp\{-y_j(\mathbf{s} - \mathbf{r}, t - \tau) \\ \times \exp(\lambda(\{\mathbf{s} - \mathbf{r}\}_1 - c(t - \tau)))\}] \\ \times [\exp(\lambda(\{\mathbf{s} - \mathbf{r}\}_1 - c(t - \tau))] \\ \times [\gamma_{ij}(\tau)e^{-\lambda c\tau}] [p_{ij}(\mathbf{r}) \exp(\lambda\{\mathbf{r}\}_1)] d\tau d\mathbf{r} \\ + H_i(\mathbf{s}, t) \exp(\lambda(\{\mathbf{s}\}_1 - ct)).$$

Also,  $y_i(\mathbf{s}, t)$  is uniformly bounded, i.e., for some  $D_i^*$ ,  $y_i(\mathbf{s}, t) \leq D_i^*$  for  $i = n_1 + 1, \dots, n$ . It follows that  $w_i(\mathbf{s}, t)$  exists and satisfies equations (2.1). The uniqueness and monotonicity follow in the same way as in

Theorem 1 part (i) of our paper [18], by first proving uniqueness and monotonicity for  $i = 1, \dots, n_1$ , then for  $i = n_1 + 1, \dots, n$ .

(ii) The proof is identical to the proof of part (ii) of our paper [18]. Note that we only need to consider equations (2.1) for  $i = 1, \dots, n_1$ .

(iii) Take  $c^* > 0$  and  $\lambda$  and  $\lambda^*$  such that  $0 < \lambda \leq \lambda^* < \Delta_V$ ,  $K_1(c^*, \lambda^*) < 1$  and  $K_2(c^*, \lambda) < 1$ . Since  $K_1(c, \lambda^*)$  and  $K_2(c, \lambda)$  are continuous functions of  $c$ , there exists a  $c$ , with  $0 < c < c^*$  such that  $K_1(c, \lambda^*) < 1$  and  $K_2(c, \lambda) < 1$ . For this  $c$  there exist  $D_i^*$  for  $i = n_1 + 1, \dots, n$ , such that  $w_i(\mathbf{s}, t) \leq D_i^* \exp(-\lambda(\{\mathbf{s}\}_1 - ct))$  for  $\mathbf{s} \in \mathbf{R}^N$ ,  $t \geq 0$  and  $i = n_1 + 1, \dots, n$ . The constants are not affected by rotating the axes, so we obtain  $w_i(\mathbf{s}, t) \leq D_i^* \exp(-\lambda(|\mathbf{s}| - ct))$ . If  $|\mathbf{s}| \geq c^*t$ , then  $w_i(\mathbf{s}, t) \leq D_i^* \exp(-\lambda(c^* - c)t)$ . Hence  $\sup\{w_i(\mathbf{s}, t) : |\mathbf{s}| \geq c^*t\} \leq D_i^* \exp(-\lambda(c^* - c)t)$ , for  $i = n_1 + 1, \dots, n$ . But  $c^* > c$ , hence  $\lim_{t \rightarrow \infty} \sup\{w_i(\mathbf{s}, t) : |\mathbf{s}| \geq c^*t\} = 0$  for  $i = n_1 + 1, \dots, n$ .  $\square$

**Corollary 1.** *If  $\rho(\Gamma) \leq 1$ , then the asymptotic speed of propagation for each type is zero.*

*Proof.* Observe that for each  $c > 0$ , there exist positive reals  $\lambda_1$  and  $\lambda_2$  such that  $K_i(c, \lambda) < 1$  for  $0 \leq \lambda < \lambda_i$  for  $i = 1, 2$ . We need only take  $0 < \lambda = \lambda^* < \min(\lambda_1, \lambda_2)$ , and the result follows from parts (ii) and (iii) of Theorem 1.  $\square$

**Corollary 2.** *If  $\rho(\Gamma_{11}) > 1$  and  $\rho(\Gamma_{22}) \leq 1$ , then the asymptotic speed of propagation for each type is at most  $c_1$ .*

*Proof.* For  $c > c_1$ , there exists  $\lambda^* > 0$  such that  $K_1(c, \lambda^*) < 1$ . Also  $K_2(c, \lambda) < 1$  for  $0 \leq \lambda < \lambda_2$  for some  $\lambda_2$ . Choose  $\lambda$  so that  $0 < \lambda < \min(\lambda^*, \lambda_2)$ . The result follows from parts (ii) and (iii) of Theorem 1.  $\square$

**Corollary 3.** *If  $\rho(\Gamma_{11}) \leq 1$  and  $\rho(\Gamma_{22}) > 1$ , then*

- (i) *the asymptotic speed of propagation for types in group 1 is zero.*
- (ii) *The asymptotic speed of propagation of group 2 types is at most  $c_2$ .*

*Proof.* (i) If  $\rho(\Gamma_{11}) \leq 1$ , for each  $c > 0$  there exists a  $\lambda_1$  such that  $K_1(c, \lambda) < 1$  for  $0 < \lambda < \lambda_1$ . The result follows from part (ii) of Theorem 1.

(ii) Observe that  $\alpha_1^*(c)$  is an increasing function of  $c$ , and  $\alpha_2(c)$  is a decreasing function of  $c$ . Also  $\alpha_1^*(c_2) \geq \alpha_2(c_2)$  since  $c_2 = \max(c_1, c_2)$ . Hence for  $c > c_2$ ,  $K_1(c, \lambda) < 1$  for  $\lambda$  such that  $0 < \lambda < \alpha_1^*(c_2)$ . Take  $\lambda^* \in (\alpha_2(c_2), \alpha_1^*(c_2))$ . Also, there exists a positive  $\lambda$  such that  $\alpha_2(c) < \lambda < \alpha_2(c_2)$  with  $K_2(c, \lambda) < 1$ . Choose such a  $\lambda$ . The result then follows by part (iii) of Theorem 1.  $\square$

**Corollary 4.** *If  $\rho(\Gamma_{11}) > 1$  and  $\rho(\Gamma_{22}) > 1$ , then*

(i) *the asymptotic speed of propagation for types in group 1 is at most  $c_1$ .*

(ii) *If  $c_0 = \max(c_1, c_2)$ , then the asymptotic speed of propagation of group 2 types is at most  $c_0$ .*

*Proof.* (i) If  $\rho(\Gamma_{11}) > 1$  for each  $c > c_0$  there exists a  $\lambda$  such that  $K_1(c, \lambda) < 1$ . The result follows by part (ii) of Theorem 1.

(ii) Again we observe that  $\alpha_1^*(c)$  is an increasing function of  $c$ , and  $\alpha_2(c)$  is a decreasing function of  $c$ . Also,  $\alpha_1^*(c_0) \geq \alpha_2(c_0)$ . Hence, for each  $c > c_0$  there exist  $\lambda$  and  $\lambda^*$  where  $\alpha_1^*(c) > \lambda^* > \alpha_1^*(c_0) \geq \alpha_2(c_0) > \lambda > \alpha_2(c)$  and  $K_1(c, \lambda^*) < 1$  and  $K_2(c, \lambda) < 1$ . The result follows by part (iii) of Theorem 1.  $\square$

#### 4. A lower bound for the asymptotic speed of propagation.

**Theorem 2.** *If  $\rho(\Gamma_{11}) > 1$ , then the asymptotic speed of propagation of every type is at least  $c_1$ , i.e., for  $i = 1, \dots, n$  and  $c < c_1$ , there exist  $b_i$  and  $T_i$  such that  $\min\{w_i(\mathbf{s}, t) : |\mathbf{s}| \leq ct\} \geq b_i$  for  $t > T_i$ .*

*Proof.* For types  $i = 1, \dots, n_1$  the proof follows exactly as in Theorem 2 of our paper [18].

Since an infection in group 1 can cause an infection in group 2 types, there exists an  $i > n_1$  and  $j \leq n_1$  with  $\gamma_{ij} \neq 0$ . Take such an  $i$  and  $j$ . There exist  $R > 0$  and  $T^* > 0$  such that  $\int_0^{T^*} \gamma_{ij}(\theta) d\theta = a > 0$  and

$$\int_{|\mathbf{u}| \leq R} p_{ij}(\mathbf{u}) \, d\mathbf{u} = b > 0.$$

Consider any  $c < c_1$  and take  $c^*$  such that  $c < c^* < c_1$ . From Theorem 2 of our paper [18], we can then find the corresponding  $b_j$  and  $T_j$  so that  $w_j(\mathbf{r}, \tau) \geq b_j$  for all  $|\mathbf{r}| \leq c^*\tau$  and  $\tau \geq T_j$ .

Now for  $t > T_j$ ,

$$\begin{aligned} w_i(\mathbf{s}, t) &\geq \int_0^t \int_{\mathbf{R}^N} w_j(\mathbf{r}, \tau) \gamma_{ij}(t - \tau) p_{ij}(\mathbf{s} - \mathbf{r}) \, d\mathbf{r} \, d\tau \\ &\geq b_j \int_{T_j}^t \int_{|\mathbf{r}| \leq c^*\tau} \gamma_{ij}(t - \tau) p_{ij}(\mathbf{s} - \mathbf{r}) \, d\mathbf{r} \, d\tau \\ &= b_j \int_0^{(t-T_j)} \int_{|\mathbf{u}-\mathbf{s}| \leq c^*(t-\theta)} \gamma_{ij}(\theta) p_{ij}(\mathbf{u}) \, d\mathbf{u} \, d\theta \\ &\geq b_j \int_0^{(t-T_j)} \int_{|\mathbf{u}| \leq c^*(t-\theta) - |\mathbf{s}|} \gamma_{ij}(\theta) p_{ij}(\mathbf{u}) \, d\mathbf{u} \, d\theta \\ &= b_j \int_0^{(t-T_j)} \int_{|\mathbf{u}| \leq (c^*-c)(t-\theta) + (ct-|\mathbf{s}|-c\theta)} \gamma_{ij}(\theta) p_{ij}(\mathbf{u}) \, d\mathbf{u} \, d\theta. \end{aligned}$$

Now for all  $\mathbf{s}$  such that  $|\mathbf{s}| \leq ct$  and  $t > T_j + T^*$

$$w_i(\mathbf{s}, t) \geq b_j \int_0^{T^*} \int_{|\mathbf{u}| \leq (c^*-c)t - c^*\theta} \gamma_{ij}(\theta) p_{ij}(\mathbf{u}) \, d\mathbf{u} \, d\theta.$$

Thus,  $w_i(\mathbf{s}, t) \geq b_j ab = b_i$  say, for  $|\mathbf{s}| \leq ct$  and  $t > T_j + T^*$ , provided  $((c^* - c)t - c^*T^*) \geq R$ , i.e.,

$$t \geq \max \left( \frac{R + c^*T^*}{(c^* - c)}, T_j + T^* \right).$$

This proves the result for a single  $i > n_1$ . The result can now be extended to all  $i > n_1$  by using the nonreducibility within the group 2 types and using a sequence argument with the specific  $i$  above playing the role of  $j$  to start the sequential argument.  $\square$

**Theorem 3.** *If  $\rho(\Gamma_{22}) > 1$ , then the asymptotic speed of propagation of group 2 types is at least  $c_2$ .*

*Proof.* The proof is essentially the same as Theorem 2 of our paper [18].  $\square$

**5. The asymptotic speed of propagation.** The results of Sections 3 and 4 may be put together and summarized as follows:

- (1) If  $\rho(\Gamma) \leq 1$ , then the asymptotic speed of propagation for each type is zero.
- (2) If  $\rho(\Gamma_{11}) > 1$  and  $\rho(\Gamma_{22}) \leq 1$ , then the asymptotic speed of propagation for each type is  $c_1$ .
- (3) If  $\rho(\Gamma_{11}) \leq 1$  and  $\rho(\Gamma_{22}) > 1$ , then
  - (i) the asymptotic speed of propagation for types in group 1 is zero;
  - (ii) the asymptotic speed of propagation of group 2 types is  $c_2$ .
- (4) If  $\rho(\Gamma_{11}) > 1$  and  $\rho(\Gamma_{22}) > 1$ , then
  - (i) the asymptotic speed of propagation for types in group 1 is  $c_1$ ;
  - (ii) the asymptotic speed of propagation of group 2 types is  $c_0 = \max(c_1, c_2)$ .

It is of interest to see how these results tie in with the wave solutions [19] and the approximate results using the saddle point methods obtained in Radcliffe and Rass [20].

The results for the wave solutions, under the conditions imposed in this paper (see [19, Theorems 8–11]) are as follows:

- (1) If  $\rho(\Gamma) \leq 1$ , then there are no wave solutions at any speed.
- (2) If  $\rho(\Gamma_{11}) > 1$  and  $\rho(\Gamma_{22}) \leq 1$ , then there is a unique wave solution modulo translation for each speed  $c \geq c_1$  for all types in both groups 1 and 2. No wave solution is possible for any speed  $c < c_1$ .
- (3) If  $\rho(\Gamma_{11}) \leq 1$  and  $\rho(\Gamma_{22}) > 1$ , then wave solutions are only possible in group 2 alone. There is a unique wave solution for all types in group 2 at each speed  $c \geq c_2$ . No wave solutions are possible at any speed  $c < c_2$ .
- (4) If  $\rho(\Gamma_{11}) > 1$  and  $\rho(\Gamma_{22}) > 1$ , then it is possible to have a wave solution amongst group 2 types only. Such solutions can only occur at speeds  $c \geq c_2$ , the solution at each such speed being unique modulo translation. When  $c_2 > c_1$ , so that  $c_0 = c_2$ , the wave solution at speed

$c_2$  amongst group 2 types only is the one of major interest. If  $c_1 > c_2$ , so that  $c_0 = c_1$ , then our main concern lies with the existence of a wave solution amongst both groups of type at speed  $c_1$ . Wave solutions do exist at speed  $c_1$ , but there is a multiplicity of such solutions. We refer the reader to Sections 7 and 8 of Radcliffe and Rass [19] for a statement of the results and an interpretation of this multiplicity. In the unusual case when  $c_1 = c_2 = c_0$ , although wave solutions exist in both groups at speeds  $c$ , with  $c_0 < c < A$  for some  $A$ , it was not established whether a wave solution exists at speed  $c = c_0$ .

Hence, for group 1 types, if  $\rho(\Gamma_{11}) \leq 1$  the speed of propagation is zero, and if  $\rho(\Gamma_{11}) > 1$  it is the minimum speed for which wave solutions exist in group 1 alone.

For group 2 types, if  $\rho(\Gamma) \leq 1$ , the speed of propagation is zero, and if  $\rho(\Gamma_{11}) \leq 1$  and  $\rho(\Gamma_{22}) > 1$  it is  $c_2$ , the minimum speed for which wave solutions exist in group 2 alone. Finally, if  $\rho(\Gamma_{11}) > 1$ , then the speed of propagation is  $c_0 = \max(c_1, c_2)$ . This is the infimum of the speeds at which wave solutions exist in both groups when  $c_1 \geq c_2$ . When  $c_2 > c_1$  it is the minimum speed at which waves exist in group 2 only.

We now consider the link with the saddle point results. These were obtained for a model which is a special case of the model considered in this paper. Note, however, that the contact distributions were not restricted to be radially symmetric.

For the radially symmetric case of the simpler model, all the approximate results obtained using the saddle point method are confirmed by the rigorous results obtained in this paper. (Note how the conditions of cases 2, 3, 6, 8 and the special situation of case 4 of Section 5 of Radcliffe and Rass [20] relate to the condition  $\alpha_1^*(\max(c_1, c_2)) \geq \alpha_2(\max(c_1, c_2))$  used in the present paper.) In fact, the rigorous results also cover cases where the saddle point method could not be applied because of the lack of analyticity of a certain function,  $f(\lambda)$ , at its minimum.

**6. Some further results and the extension to the general reducible case.** The results obtained so far are subject to the restriction that  $\alpha_1^*(\max(c_1, c_2)) \geq \alpha_2(\max(c_1, c_2))$ . We now turn our attention to the case where  $\alpha_1^*(\max(c_1, c_2)) < \alpha_2(\max(c_1, c_2))$ . This can only occur if  $\rho(\Gamma_{22}) > 1$ .

The results for types in group 1 are unaltered.

The saddle point method cannot be used for group 2 types since it is easy to establish from the results of Section 5 of Radcliffe and Rass [20] that the function  $f(\lambda)$  used in that paper is not analytic at its minimum if  $\alpha_1^*(\max(c_1, c_2)) < \alpha_2(\max(c_1, c_2))$ . In fact, this minimum occurs where  $f(\lambda) = c^*$  with  $c^* > \max(c_1, c_2)$  satisfying the condition that  $\alpha_1^*(c^*) = \alpha_2(c^*)$ . Note that  $c^*$  is uniquely defined, and the value of  $\lambda$  at which this minimum occurs is  $\lambda = \alpha_1^*(c^*) = \alpha_2(c^*)$ .

If  $c_2 > c_1$ , then there exists a wave solution amongst group 2 types only at each speed  $c \geq c_2 = c_0$  which is unique modulo translation; the wave solution at speed  $c_2$  being the one of major interest. If  $c_1 \geq c_2$ , then we are concerned with wave solutions amongst both groups of types. No such solution exists at any speed unless there exists a  $c > 0$  such that  $\alpha_1(c) = \alpha_2(c)$ . If such a  $c$  exists, then the infimum of the speeds at which wave solutions exist amongst both groups of types is necessarily greater than  $c^*$ .

For types in group 2, the methods of this paper now only give bounds on the possible speed of propagation. We say that the speed of propagation lies between  $c_\alpha$  and  $c_\beta$ , where  $c_\alpha < c_\beta$ , if

- (i) for any  $c > c_\beta$ , the solution  $w_i(\mathbf{s}, t)$  tends uniformly to zero in the region  $|\mathbf{s}| \geq ct$ ,
- (ii) for any  $c < c_\alpha$ , the solution  $w_i(\mathbf{s}, t)$  is bounded away from zero uniformly in the region  $|\mathbf{s}| \leq ct$  for  $t$  sufficiently large.

The results are summarized in the following theorem.

**Theorem 4.** *If  $\rho(\mathbf{\Gamma}_{22}) > 1$  with  $\alpha_1^*(\max(c_1, c_2)) < \alpha_2(\max(c_1, c_2))$ , then*

- (i) *The asymptotic speed of propagation of group 1 types is  $c_1$ , where  $c_1 = 0$  if  $\rho(\mathbf{\Gamma}_{11}) \leq 1$ .*
- (ii) *The asymptotic speed of propagation of group 2 types is at least  $\max(c_1, c_2)$ ; and at most  $c^*$ , where  $c^*$  is such that  $\alpha_1^*(c^*) = \alpha_2(c^*)$ .*

*Proof.* (i) This follows from Radcliffe and Rass [18].

(ii) The result that the speed of propagation is at least  $\max(c_1, c_2)$  follows from Theorems 2 and 3. If  $\rho(\mathbf{\Gamma}_{11}) \leq 1$ , it follows in an identical manner to Corollary 3 part (ii) of Theorem 1 that the asymptotic speed

of propagation is at most  $c^*$ . If  $\rho(\mathbf{\Gamma}_{11}) > 1$ , we use a similar argument to Corollary 4 part (ii) to obtain the result.  $\square$

The results of this and the previous sections may be extended to cover a general reducible epidemic. No additional mathematics is required. The results are therefore merely stated.

Consider only those groups of types in which an infection can be caused by the initial infection, perhaps through a series of infections. This will give a submatrix  $\mathbf{\Gamma}^*$  of the infection matrix  $\mathbf{\Gamma}$ . By reordering the types, we can ensure that the sub-matrix  $\mathbf{\Gamma}^*$  is expressed in normal form, (see [11, p. 75])

$$\mathbf{\Gamma}^* = \begin{pmatrix} \mathbf{\Gamma}_{1,1} & 0 & \cdots & 0 & 0 & \cdots & 0 \\ 0 & \mathbf{\Gamma}_{2,2} & \cdots & 0 & 0 & \cdots & 0 \\ \vdots & \vdots & \ddots & \vdots & \vdots & \vdots & \vdots \\ 0 & 0 & \cdots & \mathbf{\Gamma}_{g,g} & 0 & \cdots & 0 \\ \mathbf{\Gamma}_{g+1,1} & \mathbf{\Gamma}_{g+1,2} & \cdots & \mathbf{\Gamma}_{g+1,g} & \mathbf{\Gamma}_{g+1,g+1} & \cdots & 0 \\ \cdots & \cdots & \cdots & \cdots & \cdots & \cdots & \cdots \\ \mathbf{\Gamma}_{s,1} & \mathbf{\Gamma}_{s,2} & \cdots & \mathbf{\Gamma}_{s,g} & \mathbf{\Gamma}_{s,g+1} & \cdots & \mathbf{\Gamma}_{s,s} \end{pmatrix},$$

where  $\mathbf{\Gamma}_{ii}$  is a nonreducible,  $n_i \times n_i$  matrix for  $i = 1, \dots, s$ ; and for each  $i = g+1, \dots, s$ , there exists a  $j < i$  such that  $\mathbf{\Gamma}_{i,j} \neq 0$ . A corresponding partition is made for  $\mathbf{V}(c, \lambda)$ .

For  $k, t$  such that  $\mathbf{\Gamma}_{kt} \neq 0$ , we define

$$\Delta_{kt} = \min \left\{ \Delta_{ij} \text{ for } i = \sum_{r=1}^{k-1} n_r + 1, \dots, \sum_{r=1}^k n_r \right. \\ \left. \text{and } j = \sum_{r=1}^{t-1} n_r + 1, \dots, \sum_{r=1}^t n_r \right\}.$$

We restrict  $p_{ij}(\mathbf{r})$  so that, for such  $k, t$ ,  $\Delta_{kt} \geq \min\{\Delta_{kk}, \Delta_{tt}\}$ . We impose equivalent conditions on the contact distributions and the infection from outside to those imposed for the simple reducible case.

If  $\rho(\mathbf{\Gamma}_{ii}) > 1$ , define

$$c_i = \inf\{c > 0 : \rho(\mathbf{V}_{ii}(c, \lambda)) = 1 \text{ for some } \lambda \in (0, \Delta_{ii})\}.$$



Then for  $c \geq c_i$ , we define  $\lambda = \alpha_i(c)$  to be the smallest positive root of  $\rho(\mathbf{V}_{ii}(c, \lambda)) = 1$ . This is the only root if  $c = c_i > 0$ . In this case we define  $\alpha_i^*(c_i) = \alpha_i(c_i)$ . When  $c > c_i$  there is a second positive root, which is defined to be  $\alpha_i^*(c)$ . There is no root if  $c < c_i$ .

When  $\rho(\mathbf{\Gamma}_{ii}) \leq 1$ , we define  $c_i = 0$ . For each  $c > 0$ , there is a single positive root of  $\rho(\mathbf{V}_{ii}(c, \lambda)) = 1$  which is defined to be  $\alpha_i^*(c)$ . In this case  $\alpha_i(c)$  is defined to be zero.

Now define

$$c_i^0 = \max\{c_j \text{ over all } j \text{ such that there exists a sequence} \\ j = i_1 < i_2 \cdots < i_r = i \text{ and } \mathbf{\Gamma}_{i_k i_t} \neq 0 \text{ for } k = 1, \dots, r-1\}.$$

Note that  $c_i^0 = c_i$  for  $i = 1, \dots, g$ .

For  $i = 1, \dots, g$ , define  $c_i^* = c_i$ . For  $i > g$ , define

$$c_i^* = \inf\{c : c > c_i^0 \text{ and for every sequence } 1 \leq i_1 < \cdots < i_r = i : \\ \mathbf{\Gamma}_{i_j i_{j+1}} \neq 0 \text{ for } j = 1, \dots, r-1, \\ \alpha_{i_k}(c) < \alpha_{i_t}^*(c) \text{ for all } k, t \text{ such that } 1 \leq t < k \leq r\}.$$

Then the speed  $c$  of propagation of group  $i$  types is such that  $c_i^0 \leq c \leq c_i^*$ . Note that for  $i = 1, \dots, g$ , the speed of propagation is  $c_i$ .

**7. The final size.** We now prove the pandemic theorem. This gives a lower bound for  $v_i(\mathbf{s}) = 1 - \lim_{t \rightarrow \infty} x_i(\mathbf{s}, t)$ , the proportion of individuals of type  $i$  at position  $\mathbf{s}$  who eventually suffer the epidemic. Note that  $v_i(\mathbf{s})$  exists since  $x_i(\mathbf{s}, t)$  is monotone decreasing in  $t$  and bounded below.

Since  $H_i(\mathbf{s}, t)$  is monotone increasing in  $t$  and bounded above,  $a_i(\mathbf{s}) = \lim_{t \rightarrow \infty} H_i(\mathbf{s}, t)$  exists. If  $a_i(\mathbf{s}) \equiv 0$  for all  $\mathbf{s} \in \mathbf{R}^N$ , then the infectives from outside do not directly infect type  $i$  susceptibles. Note that  $a_i(\mathbf{s}) > 0$  for  $\mathbf{s}$  in some open set in  $\mathbf{R}^N$  and for some  $i = 1, \dots, n_1$ .

**Theorem 3** (The Pandemic Theorem). (i) *If  $\rho(\mathbf{\Gamma}_{11}) \leq 1$  and  $\rho(\mathbf{\Gamma}_{22}) > 1$ , then  $v_i(\mathbf{s}) \geq \eta_i$  for all  $\mathbf{s} \in \mathbf{R}^N$  and  $i = n_1 + 1, \dots, n$ , where  $y_i = \eta_i$  for  $i = n_1 + 1, \dots, n$ , is the unique positive solution to*

$$-\log(1 - y_i) = \sum_{j=n_1+1}^n \gamma_{ij} y_j, \quad i = n_1 + 1, \dots, n.$$

(ii) If  $\rho(\mathbf{\Gamma}_{11}) > 1$ , then  $v_i(\mathbf{s}) \geq \eta_i$  for all  $\mathbf{s} \in \mathbf{R}^N$  and  $i = 1, \dots, n$ , where  $y_i = \eta_i$  for  $i = 1, \dots, n$  is the unique positive solution to

$$-\log(1 - y_i) = \sum_{j=1}^n \gamma_{ij} y_j, \quad i = 1, \dots, n.$$

*Proof.* (i) This follows in an identical manner to Corollary 4 to Theorem 5 of our paper [18].

(ii) The result that  $v_i(\mathbf{s}) \geq \eta_i$  for all  $\mathbf{s} \in \mathbf{R}^N$  and  $i = 1, \dots, n_1$  also follows in an identical manner to Corollary 4 to Theorem 5 of our paper [18]. It remains only to show that  $v_i(\mathbf{s}) \geq \eta_i$  for all  $\mathbf{s} \in \mathbf{R}^N$  and  $i = n_1 + 1, \dots, n$ .

Consider equation (2.1) with  $w_i(\mathbf{s}, t) = -\log(1 - v_i(\mathbf{s}, t))$ , i.e.,

$$\begin{aligned} & -\log(1 - v_i(\mathbf{s}, t)) \\ &= \sum_{j=1}^n \int_{\mathbf{R}^N} \int_0^t v_j(\mathbf{s} - \mathbf{r}, t - \tau) p_{ij}(\mathbf{r}) \gamma_{ij}(\tau) d\tau d\mathbf{r} + H_i(\mathbf{s}, t). \end{aligned}$$

Taking the limit as  $t \rightarrow \infty$  and using monotone convergence (for details see Theorem 5 of our paper [16])

$$-\log(1 - v_i(\mathbf{s})) = \sum_{j=1}^n \gamma_{ij} \int_{\mathbf{R}^N} v_j(\mathbf{s} - \mathbf{r}) p_{ij}(\mathbf{r}) d\mathbf{r} + a_i(\mathbf{s})$$

for  $\mathbf{s} \in \mathbf{R}^N$  and  $i = 1, \dots, n$ .

Now  $a_i(\mathbf{s}) \geq 0$  for  $\mathbf{s} \in \mathbf{R}^N$  and  $i = 1, \dots, n_1$ . Also,  $v_i(\mathbf{s}) \geq \eta_i > 0$  for  $\mathbf{s} \in \mathbf{R}^N$  and  $i = 1, \dots, n_1$ . Hence, for  $i = n_1 + 1, \dots, n$

$$\begin{aligned} -\log(1 - v_i(\mathbf{s})) &\geq \sum_{j=n_1+1}^n \gamma_{ij} \int_{\mathbf{R}^N} v_j(\mathbf{s} - \mathbf{r}) p_{ij}(\mathbf{r}) d\mathbf{r} \\ &\quad + \sum_{j=1}^{n_1} \gamma_{ij} \eta_j. \end{aligned}$$

Let  $v_i = \inf_{\mathbf{s}} v_i(\mathbf{s})$ . Then for  $i = n_1 + 1, \dots, n$ , we obtain

$$-\log(1 - v_i(\mathbf{s})) \geq \sum_{j=n_1+1}^n \gamma_{ij} v_j + \sum_{j=1}^{n_1} \gamma_{ij} \eta_j$$

for  $\mathbf{s} \in \mathbf{R}^N$ . Hence,

$$-\log(1 - v_i) \geq \sum_{j=n_1+1}^n \gamma_{ij} v_j + \sum_{j=1}^{n_1} \gamma_{ij} \eta_j.$$

Now  $\sum_{j=1}^{n_1} \gamma_{ij} \eta_j > 0$  for some  $i$  from  $n_1 + 1, \dots, n$ . Also  $\Gamma_{22}$  is nonreducible.

From Theorem 1 of our paper [16], the equations

$$-\log(1 - v_i) = \sum_{j=n_1+1}^n \gamma_{ij} v_j + b_i, \quad i = n_1 + 1, \dots, n,$$

where at least one  $b_i > 0$ , have a unique solution which is positive and is continuous and monotone increasing in each  $b_i$ .

Thus,  $v_i \geq \eta_i$ ,  $i = n_1 + 1, \dots, n$ , where  $y_i = \eta_i$ ,  $i = n_1 + 1, \dots, n$ , is the unique positive solution to

$$-\log(1 - y_i) = \sum_{j=n_1+1}^n \gamma_{ij} y_j + \sum_{j=1}^{n_1} \gamma_{ij} \eta_j, \quad i = n_1 + 1, \dots, n.$$

Since  $v_i = \inf_{\mathbf{s}} v_i(\mathbf{s})$ , we obtain  $v_i(\mathbf{s}) \geq \eta_i$  for  $\mathbf{s} \in \mathbf{R}^N$  and  $i = n_1 + 1, \dots, n$ .

Thus  $v_i(\mathbf{s}) \geq \eta_i$ ,  $i = 1, \dots, n$ , where  $y_i = \eta_i$ ,  $i = 1, \dots, n$ , is the unique positive solution to

$$-\log(1 - y_i) = \sum_{j=1}^n \gamma_{ij} y_j, \quad i = 1, \dots, n.$$

This completes the proof of the theorem.  $\square$

Consider the general reducible epidemic as described in Section 6, so that there are exactly  $s$  groups in which infection occurs. The extension of Theorem 3 to such an epidemic is now recorded. Let  $\mathbf{v}_i(\mathbf{s})$  be the vector of final sizes of the epidemic at position  $\mathbf{s}$  for types in group  $i$  where  $\mathbf{\Gamma}^*$  corresponding to the  $s$  groups is written in normal form as in Section 6.

Then  $\mathbf{v}_i(\mathbf{s}) \geq \eta_i$  where  $\mathbf{y}_i = \eta_i$ ,  $i = 1, \dots, s$ , is a particular solution of

$$\begin{pmatrix} \mathbf{u}_1 \\ \vdots \\ \mathbf{u}_s \end{pmatrix} = \mathbf{\Gamma}^* \begin{pmatrix} \mathbf{y}_1 \\ \vdots \\ \mathbf{y}_s \end{pmatrix}$$

with  $-\log(1 - \{\mathbf{u}_1\}_j) = \{\mathbf{y}_1\}_j$ .

This particular solution is the unique solution [16, Theorem 1] satisfying the following conditions:

- (i) For each  $i = 1, \dots, g$ ,  $\eta_i > \mathbf{0}$  if  $\rho(\mathbf{\Gamma}_{ii}) > 1$  and  $\eta_i = \mathbf{0}$  if  $\rho(\mathbf{\Gamma}_{ii}) \leq 1$ ;
- (ii) Successively, for each  $i = g+1, \dots, s$ ,  $\eta_i > \mathbf{0}$  if  $\rho(\mathbf{\Gamma}_{ii}) > 1$  and/or there exists  $j < i$  such that  $\mathbf{\Gamma}_{ij} \neq \mathbf{0}$  and  $\eta_j > \mathbf{0}$ ; otherwise,  $\eta_i = \mathbf{0}$ .

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