Qualitative Analyses of Communicable Disease Models*

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ABSTRACT

Deterministic communicable disease models which are initial value problems for a system of ordinary differential equations are considered, where births and deaths occur at equal rates with all newborns being susceptible. Asymptotic stability regions are determined for the equilibrium points for models involving temporary immunity, disease-related fatalities, carriers, migration, dissimilar interacting groups, and transmission by vectors. Epidemiological interpretations of all results are given.

1. INTRODUCTION

The spread of a communicable disease involves not only disease-related factors such as the infectious agent, mode of transmission, incubation period, infectious period, susceptibility, and resistance, but also social, cultural, economic, demographic, and geographic factors. Insight into communicable disease processes can be obtained by analyzing models which contain some of these factors. Here some new differential equation models are formulated and theorems about equilibrium points and asymptotic stability regions are obtained by using modern qualitative methods for differential equations. Terminology, notation, and assumptions are given in this section. A section on previous results is included, because new formulations of the basic models are used which lead to more meaningful communicable disease interpretations and because the treatment here unifies

scattered results and compares various models. References to other communicable disease models are given in the last section.

An epidemic is an occurrence of a disease in excess of normal expectancy, while a disease is called endemic if it is habitually present; however, communicable disease models of all types are often referred to as epidemic models, and the study of disease occurrence is called epidemiology. A basic concept in epidemiology is the existence of thresholds; these are critical values for quantities such as population size or vector density that must be exceeded in order for an epidemic to occur. In this paper the infectious contact number, which is the average number of contacts of an infective during his infectious period, is identified as the threshold quantity which determines the behavior of the infectious disease.

Communicable disease models involving differential equations were considered and threshold theorems were obtained by Kermack and McKendrick [18, 19]. Both deterministic and stochastic models are described in the book by N. T. J. Bailey [1]. A survey of epidemic results up to 1967 was given by K. Dietz [7]. Deterministic threshold models are considered in the monograph by P. Waltman [26]. The APHA handbook on communicable diseases [2] is a good source of information on specific diseases.

The population or community under consideration is divided into disjoint classes which change with time t. The susceptible class consists of those individuals who can incur the disease but are not yet infective. The infective class consists of those who are transmitting the disease to others. The removed class consists of those who are removed from the susceptible-infective interaction by recovery with immunity, isolation, or death. The fractions of the total population in these classes are denoted by $S(t), I(t)$, and $R(t)$, respectively.

If recovery does not give immunity, then the model is called an *SIS* model, since individuals move from the susceptible class to the infective class and then back to the susceptible class upon recovery. If individuals recover with immunity, then the model is an *SIR* model. If individuals do not recover, then the model is an *SI* model. In general, *SIR* models are appropriate for viral agent diseases such as measles, mumps, and smallpox, while *SIS* models are appropriate for some bacterial agent diseases such as meningitis, plague, and venereal diseases, and for protozoan agent diseases such as malaria and sleeping sickness.

In our communicable disease models, the following assumptions are made:

1. The population considered has constant size N which is sufficiently large so that the sizes of each class can be considered as continuous variables instead of discrete variables. If the model is to include vital

dynamics, then it is assumed that births and deaths occur at equal rates and that all newborns are susceptible. Individuals are removed by death from each class at a rate proportional to the class size with proportionality constant δ, which is called the daily death removal rate. The average lifetime is $1/\delta$.

2. The population is uniform and homogeneously mixing. The daily contact rate λ is the average number of contacts per infective per day. A contact of an infective is an interaction which results in infection of the other individual if he is susceptible. Thus the average number of susceptibles infected by an infective per day is λS, and the average number of susceptibles infected by the infective class with size NI per day is λNIS. The daily contact rate λ is fixed and does not vary seasonally. The type of direct or indirect contact adequate for transmission depends on the specific disease.

3. Individuals recover and are removed from the infective class at a rate proportional to the number of infectives with proportionality constant γ, called the daily recovery removal rate. The latent period is zero (it is defined as the period between the time of exposure and the time when infectiousness begins). Thus the proportion of individuals exposed (and immediately infective) at time t_0 who are still infective at time $t_0 + t$ is $\exp(-\gamma t)$, and the average period of infectivity is $1/\gamma$.

The removal rate from the infective class by both recovery and death is $\gamma + \delta$, so that the death-adjusted average period of infectivity is $1/(\gamma + \delta)$. Thus the average number of contacts (with both susceptibles and others) of an infective during his infectious period is $\sigma = \lambda/(\gamma + \delta)$, which is called the infectious contact number. Since the average number of susceptibles infected by an infective during his infectious period is σS, the quantity σS is called the infective replacement number.

2. PREVIOUS RESULTS

In this example we formulate an initial value problem (IVP) using the class sizes first, and then change to the IVP involving the fractions of the total population in each class. The IVP for a simple *SIS* model with vital dynamics is

$$[NS(t)]' = -\lambda NIS + \gamma NI + \delta N - \delta NS,$$

$$[NI(t)]' = \lambda NIS - \gamma NI - \delta NI, \qquad (2.1)$$

$$NS(0) = NS_0 > 0, \qquad NI(0) = NI_0 > 0, \qquad NS(t) + NI(t) = N,$$

where λ is positive. The $-\lambda NIS$ term gives the rate of movement from the

susceptible class of size $NS(t)$ to the infective class of size $NI(t)$. The $-\gamma NI$ term gives the rate at which infectives recover (without immunity) and return to the susceptible class. The δN term corresponds to the newborn susceptibles, while $-\delta NS$ and $-\delta NI$ correspond to deaths in the susceptible and infective classes, respectively.

If we divide each equation in (2.1) by the population size N, then the IVP is

$$S'(t) = -\lambda IS + \gamma I + \delta - \delta S,$$

$$I'(t) = \lambda IS - \gamma I - \delta I, \tag{2.2}$$

$$S(0) = S_0 > 0, \qquad I(0) = I_0 > 0, \qquad S(t) + I(t) = 1,$$

where λ is positive. Note that the IVP (2.2) involves only the daily contact and removal rates and not the population size N. This model might be appropriate for bacterial agent diseases such as meningitis, streptococcal sore throat, and tuberculosis. In this paper, all parameters in the differential equations are nonnegative, and only nonnegative solutions are considered, since negative solutions have no epidemiological significance.

Since $S(t)$ can always be found from $I(t)$ by using $S(t) = 1 - I(t)$, it is sufficient to consider the IVP for $I(t)$. The differential equation for $I(t)$ with $S = 1 - I$ is

$$I'(t) = [\lambda - (\gamma + \delta)]I - \lambda I^2, \tag{2.3}$$

which can be solved to obtain the unique solution

$$I(t) = \begin{cases} \dfrac{e^{kt}}{\lambda(e^{kt}-1)/k + 1/I_0}, & k \neq 0 \\[2ex] \dfrac{1}{\lambda t + 1/I_0}, & k = 0 \end{cases} \tag{2.4}$$

where $k = \lambda - (\gamma + \delta)$. The asymptotic behavior of $I(t)$ for large t is found from the explicit solution (2.4).

THEOREM 2.1

The solution $I(t)$ of (2.3) approaches $1 - 1/\sigma$ as $t \to \infty$ if $\sigma = \lambda/(\gamma + \delta) > 1$ and approaches 0 as $t \to \infty$ if $\sigma \leqslant 1$.

BIOCOROLLARY 2.1

In a disease without immunity with any initial infective fraction, the infective fraction approaches a constant endemic value if the infectious contact number exceeds one; otherwise, the infective fraction approaches zero.

One advantage of precise threshold results such as Theorem 2.1 is that the effects of changes in certain parameter values on the asymptotic behavior can be determined. Note that the infective replacement number σS is 1 at the equilibrium point. A threshold result for an SI model is obtained from Theorem 2.1 by taking the removal rate γ to be zero in the equations (2.2). If the death rate δ is also zero, then the model is the SI model considered by N. T. J. Bailey [1, p. 20].

Instead of assumption 2 in Sec. 1, it is sometimes assumed that susceptibles become infectious at a rate proportional to the product of the number of susceptibles and the number of infectives, with proportionality constant β. By comparing the resulting IVP with the IVP (2.1), we see that $\beta = \lambda/N$, and thus the assumption that β is constant implies that the daily contact rate λ is proportional to the population size N. Although the daily contact rate would probably increase if the population within a fixed region increased (i.e., the population density increased), the daily contact rates might be the same for a large population in a large region and a small population in a small region. Thus it seems best to carefully separate the daily contact rate λ and the population size N, as we have done in assumption 2. Moreover, threshold statements such as Theorem 2.1 involving the infectious contact number seem more realistic than population size threshold statements which result from the alternate assumption above. Population size threshold theorems given in previous publications [1, 11, 12, 18, 19] can be converted easily to infectious contact number threshold theorems.

Although the asymptotic behavior is similar for the SIS models with and without vital dynamics, this is not true for SIR models. The IVP for an SIR model without vital dynamics is

$$S'(t) = -\lambda IS,$$

$$I'(t) = \lambda IS - \gamma I,$$

$$R'(t) = \gamma I, \qquad\qquad (2.5)$$

$$S(0) = S_0 > 0, \qquad I(0) = I_0 > 0, \qquad R(0) \geqslant 0,$$

$$S(t) + I(t) + R(t) = 1,$$

where λ and γ are positive. Since $R(t)$ can always be found from $S(t)$ and $I(t)$ by using $R(t) = 1 - S(t) - I(t)$, it is sufficient to consider the IVP in the SI plane. The solution curves $I = 1 - S + [\log(S/S_0)]/\sigma$ in the SI plane are

found from

$$\frac{dI}{dS} = -1 + \frac{1}{\sigma S},\qquad(2.6)$$

where $\sigma = \lambda/\gamma$ is the infectious contact number. By analyzing these curves [13, 26] the following result is obtained.

THEOREM 2.2

Let $(S(t), I(t))$ be the solutions of (2.5). If $\sigma S_0 \leqslant 1$, then $I(t)$ decreases to zero as $t \to \infty$; if $\sigma S_0 > 1$, then $I(t)$ first increases up to a maximum value equal to $1 - 1/\sigma - [\log(\sigma S_0)]/\sigma$ and then decreases to zero as $t \to \infty$. The susceptible fraction $S(t)$ is a decreasing function, and the limiting value $S(\infty)$ is the unique root in $(0, 1/\sigma)$ of the equation

$$1 - S(\infty) + \frac{\log[S(\infty)/S_0]}{\sigma} = 0.$$

BIOCOROLLARY 2.3

In a disease without vital dynamics where recovery gives immunity, if the initial infective replacement number is greater than one, then the infective fraction increases up to a peak and then decreases to zero; otherwise, the infective fraction decreases to zero. The infection spread stops because the infective replacement number becomes less than one when $S(t)$ becomes small; however, the final susceptible population is not zero.

The IVP for an *SIR* model with vital dynamics is

$$S'(t) = -\lambda IS + \delta - \delta S,$$

$$I'(t) = \lambda IS - \gamma I - \delta I,$$

$$R(t) = 1 - S(t) - I(t),\qquad(2.7)$$

$$S(0) = S_0 > 0,\qquad I(0) = I_0 > 0,\qquad R(0) \geqslant 0,$$

where λ and δ are positive. The asymptotic behavior of (2.7) in the triangle D bounded by the S and I axes and the line $S + I = 1$ was determined in [12] using Liapunov's direct method and is a special case of Theorem 4.1.

THEOREM 2.3.

Let $(S(t), I(t))$ be a solution of the differential equations in (2.7). If $\sigma > 1$, then $D - \{(S, 0) : 0 \leqslant S \leqslant 1\}$ is an asymptotic stability region (ASR) for the

equilibrium point (EP) $(1/\sigma, \delta(\sigma-1)/\lambda)$, *where* $\sigma = \lambda/(\gamma+\delta)$. *If* $\sigma \leqslant 1$, *D is an ASR for the EP* $(1,0)$.

BIOCOROLLARY 2.3

In a disease with vital dynamics where recovery gives immunity, if the infectious contact number exceeds one, then the susceptible and infective fractions eventually approach constant positive endemic values except in the trivial case when there are no infectives initially. If the infectious contact number is less than one, then the infective fraction approaches zero and the removed fraction approaches zero (due to death of removed individuals), so that the entire population is eventually susceptible (due to the continuous birth of new susceptibles).

By comparing Theorems 2.2 and 2.3 and their biocorollaries, it is clear that the asymptotic behaviors for *SIR* models without and with vital dynamics are very different. The *SIR* model without vital dynamics might be appropriate for describing an epidemic outbreak during a short time period, whereas the *SIR* model with vital dynamics would be appropriate over a longer time period. Viral agent diseases such as measles, chicken pox, mumps, influenza, and smallpox may have occasional large outbreaks in certain communities and yet be endemic at a low level in larger population groups.

Note from Theorems 2.1 and 2.3 that the infectious contact number threshold criterion for determining if a disease with vital dynamics remains endemic is the same for diseases without and with immunity; however, the infective fraction approached asymptotically for large time is higher for diseases without immunity than for diseases with immunity. The values $I(\infty)$ and $I(\infty)+R(\infty)$ are reasonable measures of the intensities of infectious diseases of *SIS* and *SIR* types, respectively. The infective fraction for some diseases such as measles, chicken pox, and mumps varies periodically because of seasonal changes in the daily contact rates. Models involving recurrent or periodic epidemics have been considered [1, 11, 21, 30]. Although numerical and approximate solutions of the asymptotic behavior of an *SIR* model with periodic daily contact rate have been found, a precise analysis has not been done.

3. MATHEMATICAL PRELIMINARIES

The communicable disease models which we will consider involve two dimensional autonomous, nonlinear (quadratic) systems of ordinary differential equations, and consequently the methods of phase plane analysis can be applied. See [16, 3] for a discussion of phase plane methods, almost linear systems, and the Poincaré-Bendixson theory.

We now formulate a theorem motivated by a survey paper of W. A.

Coppel [6], which we use several times to eliminate the possibility of limit cycles. The method of proof using Green's theorem has been used by other authors. In our applications of the theorem, D is a rectangle or triangle in the first quadrant, and $B(x,y)$ is found by first assuming that it is of the form $x^i y^j$ and then finding i and j such that

$$\frac{\partial}{\partial x}(BP) + \frac{\partial}{\partial y}(BQ)$$

has the same sign throughout D.

THEOREM 3.1

Assume that P and Q are continuously differentiable in an open connected region D, that no solution path of

$$x'(t) = P(x,y),$$

$$y'(t) = Q(x,y)$$

leaves D, and that D contains at least one EP. *If there exists a $B(x,y)$ which is continuously differentiable in D and such that*

$$\frac{\partial}{\partial x}(BP) + \frac{\partial}{\partial y}(BQ)$$

has the same sign throughout D, then there are no closed paths (periodic solutions) in D.

Proof. Note that closed paths must contain at least one EP, so that if D contains no EP, then there are no closed paths in D. Suppose that there is a closed path Γ with interior R containing at least one of the EP in D. Since no path leaves D, R is contained in D. By the assumption that

$$\frac{\partial}{\partial x}(BP) + \frac{\partial}{\partial y}(BQ)$$

has the same sign throughout D and Green's theorem,

$$0 \neq \int\int_R \left[\frac{\partial}{\partial x}(BP) + \frac{\partial}{\partial y}(BQ)\right] dA = \int_\Gamma BP\,dy - BQ\,dx$$

$$= \int_\Gamma B\left(\frac{dx}{dt}\frac{dy}{dt} - \frac{dy}{dt}\frac{dx}{dt}\right) dt = 0,$$

which is a contradiction.

4.   AN SIRS MODEL WITH TEMPORARY IMMUNITY.

In this model with vital dynamics, recovery gives temporary immunity. This model might be appropriate for smallpox, tetanus, influenza, cholera, and typhoid fever. One conclusion in this section is that temporary instead of permanent immunity does not change the threshold criterion, but it does raise the infective level approached asymptotically for large time. Assume that the rate at which removed individuals lose their immunity and return to the susceptible class is proportional to the number of removed individuals with proportionality constant α, called the daily loss of immunity rate. The average period of immunity is $1/\alpha$, with permanent immunity when $\alpha = 0$. The IVP is

$$S'(t) = -\lambda IS + \delta - \delta S + \alpha R$$

$$= -\lambda IS + (\delta + \alpha) - (\delta + \alpha)S - \alpha I,$$

$$I'(t) = \lambda IS - \gamma I - \delta I, \tag{4.1}$$

$$R(t) = 1 - S(t) - I(t),$$

$$S(0) = S_0 > 0, \qquad I(0) = I_0 > 0, \qquad R(0) = R_0 \geqslant 0,$$

where λ, $\gamma + \delta$ and $\delta + \alpha$ are positive. The assumption $\gamma + \delta > 0$ means that there must be some flow out of the infective class, and $\delta + \alpha > 0$ means that there must be some flow into the susceptible class. Let $\sigma = \lambda/(\gamma + \delta)$, and let D be the triangle $S \geqslant 0$, $I \geqslant 0$, $S + I \leqslant 1$. Typical SI plane portraits found by numerical integration of (4.1), showing solution paths approaching the EP, are given in Fig. 1 and 2 for infectious contact number less than and greater than one, respectively.

THEOREM 4.1

Let $(S(t), I(t))$ be a solution of (4.1). If $\sigma > 1$, then $D - \{(S,0): 0 \leqslant S \leqslant 1\}$ is an ASR (asymptotic stability region) for the EP (equilibrium point)

$$\left(\frac{1}{\sigma}, \frac{(\delta + \alpha)(\sigma - 1)}{\lambda + \alpha\sigma} \right). \tag{4.2}$$

If $\sigma \leqslant 1$, then D is an ASR for the EP $(1,0)$.

BIOCOROLLARY 4.1

In a disease where recovery gives temporary immunity, if the infectious contact number exceeds one, then the susceptible and infective fractions approach constant endemic values for large time; otherwise, the susceptible fraction increases as the infective fraction decreases to zero until eventually the entire population is susceptible.
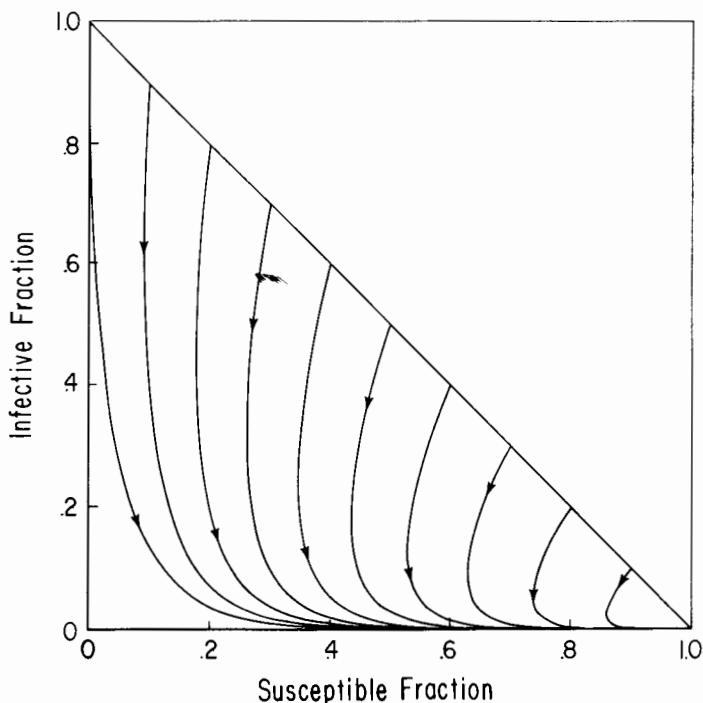
FIG. 1. Typical *SI* plane solution paths for an *SIR* model with vital dynamics and temporary immunity when the infectious contact number is 0.5. The parameter values in (4.1) are $\lambda = 0.1$, $\gamma = 0.2$, $\delta = 0.0001$, and $\alpha = 0.02$.

Proof. The EP in the *SI* plane are $(1,0)$ and the point given by (4.2). If the EP $(1,0)$ is translated to the origin, the characteristic roots of the linearization of the system of differential equations are $-\delta - \alpha$ and $(\gamma + \delta)(\sigma - 1)$. If $\sigma < 1$, both roots are real and negative, so that $(1,0)$ is an attractive node; if $\sigma > 1$, the roots are of opposite sign, so that $(1,0)$ is a saddle point. More information about the local phase portraits near the EP can be found. No solution path leaves D, since $I = 0$ is a solution path, implies $S'(t) = \delta + \alpha(1 - I) > 0$, and $I + S = 1$ implies $I'(t) + S'(t) = -\gamma$

If $\sigma > 1$, then the EP (4.2) is in the interior of D. If this EP is trans to the origin, then the characteristic roots of the linear part of the differential equations both have negative real part, so that (4.2) is an attractor. By Theorem 3.1 with $B = 1/I$, there are no limit cycles in D. The S axis is the attractive line for the saddle point $(1,0)$, while the repulsive direction has slope $-1 + \gamma/(\lambda + \delta)$, which is into the triangle. Thus D minus the S axis is an ASR for the EP (4.2) for $\sigma > 1$.
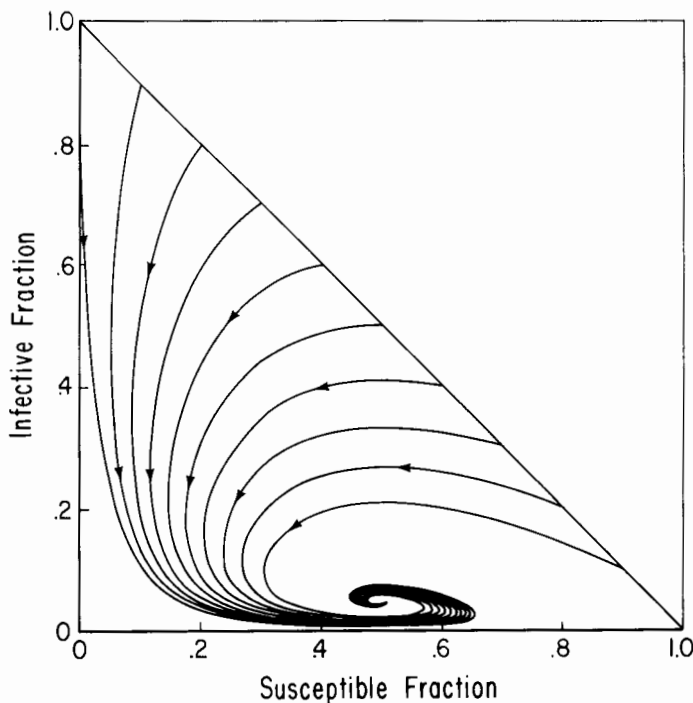
FIG. 2. Typical *SI* plane solution paths for an *SIR* model with vital dynamics and temporary immunity when the infectiour contact number is 2. The parameter values used in (4.1) are $\lambda = 0.4$, $\gamma = 0.2$, $\delta = 0.0001$, and $\alpha = 0.02$.

If $\sigma < 1$, the $(1,0)$ is the only EP in D, and consequently every solution path in D must approach $(1,0)$. If $\sigma = 1$, then the above method does not apply. However, $\sigma = 1$ implies $I'(t) = \beta I(S-1) \leq 0$ in D, with equality only if $I = 0$. Thus all solution paths approach $I = 0$, but since $I = 0$ is a solution path, all solution paths must approach $(1,0)$. Hence D is an ASR for $(1,0)$ if $\sigma \leq 1$.

5. AN SIS MODEL WITH SOME DISEASE FATALITIES

In this SIS model with vital dynamics, we let $NR(t)$ be the number of people who have died due to the disease. This model might be appropriate for plague, tuberculosis, or syphilis. A disease where recovery does not give immunity usually remains endemic; however, we will show that if there are disease-related fatalities, then the disease eventually disappears, leaving a positive susceptible fraction. Assume that the rate of removal of infectives

by death due to the disease is proportional to the number of infectives. The IVP is

$$S'(t) = -\lambda IS + \gamma I + \delta(I + S) - \delta S = -\lambda I(S - 1/\sigma),$$

$$I'(t) = \lambda IS - \gamma I - \delta I - \eta I, \tag{5.1}$$

$$R(t) = 1 - S(t) - I(t),$$

$$S(0) = S_0 > 0, \qquad I(0) = I_0 > 0, \qquad R(0) \geqslant 0,$$

where λ and η are positive. The parameter η is called the daily disease-related death rate. Let $\sigma = \lambda/(\gamma + \delta)$, $\xi = \eta/(\gamma + \delta)$, and let D be the triangle bounded by the axes and $S + I = 1$. A typical SI plane portrait is given in Fig. 3.
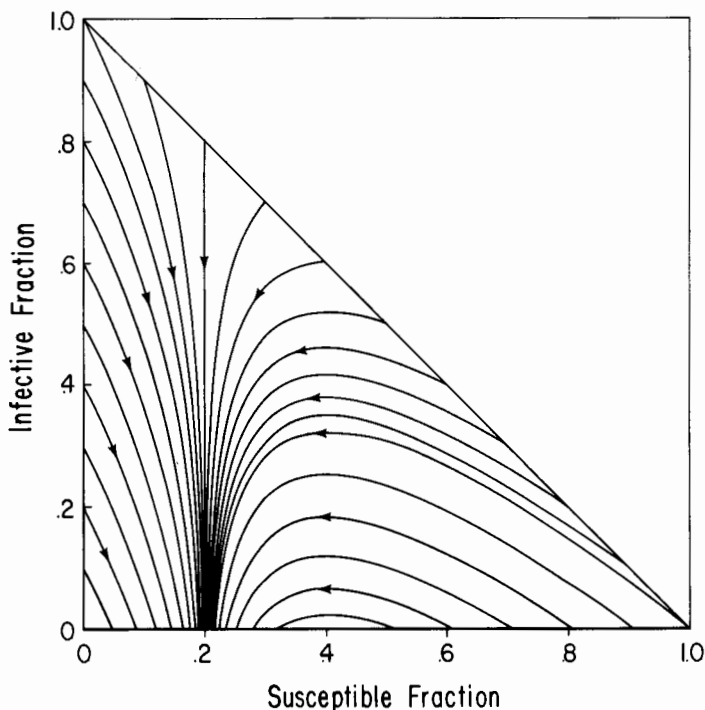

FIG. 3. Typical SI plane portrait for an SIS model with some disease fatalities. The parameter values used in (5.1) are $\lambda = 1.0$, $\gamma = 0.2$, $\delta = 0.0001$, and $\eta = 0.2$.

THEOREM 5.1

Let (S, I) be the solution of (5.1). If $1 < \sigma S_0 < 1 + \xi$, then $I(t)$ decreases to zero as $t \to \infty$; if $\sigma S_0 > 1 + \xi$, then $I(t)$ first increases up to a maximum value equal to

$$1 - \frac{1+\xi}{\sigma} + \frac{\xi}{\sigma} \log \frac{\xi}{\sigma S_0 - 1} \tag{5.2}$$

and then decreases to zero as $t \to \infty$. The susceptible fraction decreases, and the limiting value $S(\infty)$ is the unique root in $(1/\sigma, (1+\xi)/\sigma)$ of

$$1 - S(\infty) + \frac{\xi}{\sigma} \log \frac{\sigma S(\infty) - 1}{\sigma S_0 - 1} = 0. \tag{5.3}$$

If $0 \leqslant \sigma S_0 \leqslant 1$, then $I(t)$ decreases to zero as $t \to \infty$, and $S(\infty)$ is the root in $(0, 1/\sigma)$ of (5.3).

BIOCOROLLARY 5.1

In a disease with no immunity where the disease causes death of some of the infectives, the disease always eventually disappears and the final susceptible fraction is positive.

This biocorollary seems reasonable because the fraction R is increasing inasmuch as the disease is fatal to a fixed proportion of the infectives, so that the reproducing population $(S + I)$ decreases until the birth of new susceptibles is insufficient to keep the disease endemic. This model is probably unrealistic in a human population, since for a potentially fatal disease, preventive measures would probably be taken which would invalidate one of the model assumptions such as homogeneous mixing. It might be appropriate for an epizootic in an animal population such as tularemia in rabbits.

Proof. In the model (5.1), every point on the S axis is an EP. If $\sigma S_0 > 1$, then the translation $U = S - 1/\sigma$ yields

$$U'(t) = -\lambda I U,$$

$$I'(t) = \lambda I U - \eta I, \tag{5.4}$$

which is essentially the same as (2.3), and all of the conclusions there carry

over. If $\sigma S_0 \leqslant 1$, then I is decreasing unless I is zero so that $I(\infty) = 0$. Also, $S(t)$ is increasing and bounded by $1/\sigma$ (since $S = 1/\sigma$ is a solution path), so that $S(\infty)$ exists. Indeed, $S(\infty)$ is the root of (5.3) in $(0, 1/\sigma)$.

6. AN SIR MODEL WITH CARRIERS

A carrier is an individual who carries and spreads the infectious disease, but has no clinical symptoms. Models of *SIS* type involving carriers were considered in [11]. Carriers are a mode of transmission in diseases with immunity such as hepatitis, polio, diptheria, typhoid fever, and cholera. Here we assume that the number of carriers is constant in an *SIR* model with vital dynamics (models where C changes with time are possible.) The IVP is

$$S'(t) = -\lambda(I + C)S + \delta - \delta S,$$

$$I'(t) = \lambda(I + C)S - \gamma I - \delta I, \tag{6.1}$$

$$R(t) = 1 - S(t) - I(t),$$

$$S(0) = S_0 > 0, \qquad I(0) = I_0 > 0, \qquad R(0) \geqslant 0,$$

where λ, C, and δ are positive. Note that the term λCS could correspond to either a constant number CN of carriers or an inanimate carrier such as a polluted water supply with contact rate parameter λCN. Let $\sigma = \lambda/(\gamma + \delta)$, and let D be the triangle bounded by the axes and $S + I = 1$. The only EP in D is (S^*, I^*), where

$$I^* = \frac{[\sigma - 1 - C\lambda/\delta] + \{[\sigma - 1 - C\lambda/\delta]^2 + 4CN\lambda\sigma/\delta\}^{1/2}}{2\lambda/\delta}$$

and $S^* = 1 - \lambda I^*/\delta\sigma$. Typical *SI* plane portraits would be similar to those in Fig. 2.

THEOREM 6.1

For the differential equations in (6.1), the triangle D is an ASR for the EP (S^*, I^*).

BIOCOROLLARY 6.1

In a disease with carriers and vital dynamics where recovery gives immunity, the disease always remains endemic.

Proof. If the EP (S^*, I^*) is translated to the origin, the characteristic roots of the linear part of the differential equation system have negative real parts, so that (S^*, I^*) is an attractor. No solutions leave D, and there are no limit cycles, by Theorem 3.1 with $B = 1/(SI)$. Thus every solution path in D approaches (S^*, I^*).

7. AN SIS MODEL WITH MIGRATION

Communicable diseases sometimes spread across countries and continents and around the world. Some models for spatial spread have been analyzed [1, 17, 23]. A fascinating question is whether a disease could remain endemic by traveling geographically around a region or around the world. In this *SIS* model with vital dynamics, we assume that individuals immigrate and emigrate between two communities at equal rates. One conclusion is that migration can keep the disease endemic in two population groups, even though without migration the disease would eventually disappear in one of the groups. We assume that a constant proportion θ of individuals in each community move to the other community per unit time. The IVP is

$$I_1'(t) = \lambda_1 I_1(1 - I_1) - \gamma_1 I_1 - \delta_1 I_1 + \theta (I_2 - I_1)/N,$$

$$I_1(0) = I_{10} > 0, \qquad S_1 + I_1 = 1, \tag{7.1}$$

$$I_2'(t) = \lambda_2 I_2(1 - I_2) - \gamma_2 I_2 - \delta_2 I_2 + \theta (I_1 - I_2)/N_2,$$

$$I_2(0) = I_{20} > 0, \qquad S_2 + I_2 = 1,$$

where λ_1, λ_2, and θ are positive. Let $\sigma_i = \lambda_i/(\gamma_i + \delta_i)$, and let D be the rectangle bounded by the I_1 and I_2 axes and the lines $I_1 = 1$ and $I_2 = 1$. Typical $I_1 I_2$ plane portraits would be similar to Fig. 4 and 5. Let $a = \lambda_1 - \gamma_1 - \delta_1 - \theta/N_1$, $b = \theta/N_1$, $c = \theta/N_2$, and $d = \lambda_2 - \gamma_2 - \delta_2 - \theta/N_2$.

THEOREM 7.1

For the differential equations in (7.1), if $a + d < 0$ and $ad - bc \geqslant 0$, then D is an ASR for the origin; otherwise, there is a unique EP in the interior of D, and D minus the origin is an ASR for this EP.

BIOCOROLLARY 7.1

For a disease without immunity in two communities with migration, the behavior can be unusual when one of the infectious contact numbers is near 1. For example, if one infectious contact number slightly exceeds 1 and one is less than 1, then migration can cause the disease to eventually disappear in both populations. If one infectious contact number is significantly greater than 1 ($a > 0$) and one is below, then migration causes the disease to remain endemic in both populations.

Proof. To find the EP in the rectangle D, we need to find the intersection points of two parabolas. If we set the right side of the equation for $I_1'(t)$ equal to zero, we obtain

$$I_2 = \frac{N_1}{\theta}\left[\left(\gamma_1 + \delta_1 + \frac{\theta}{N_1} - \lambda_1\right)I_1 + \lambda_1 I_1^2\right]. \tag{7.2}$$
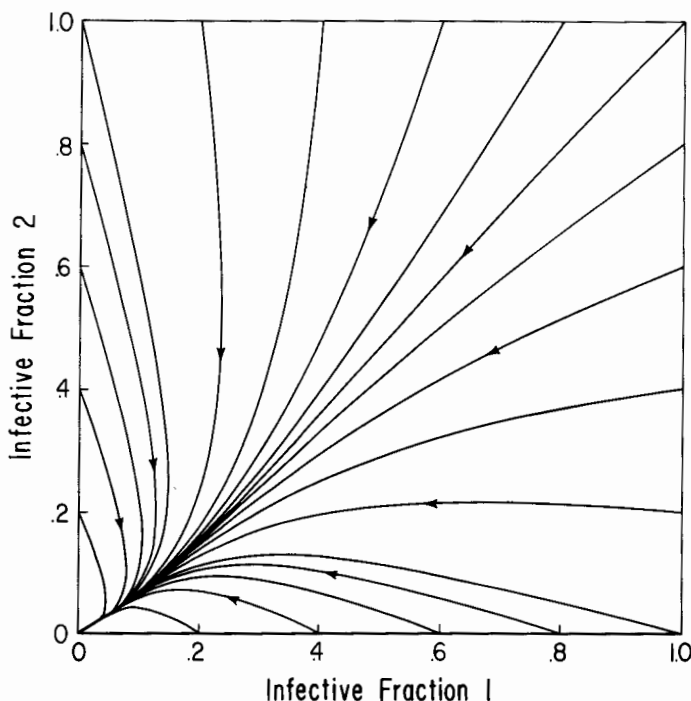

FIG. 4.   Typical $I_1 I_2$ plane portrait for an *SIS* model with two dissimilar groups using the values $N_1 = N_2$, $\lambda_{11} = 0.3$, $\lambda_{12} = \lambda_{21} = 0.1$, $\lambda_{22} = 0.2$, $\gamma_1 = \gamma_2 = 0.4$, $\delta_1 = 0.0005$, and $\delta_2 = 0.001$ in (8.1).
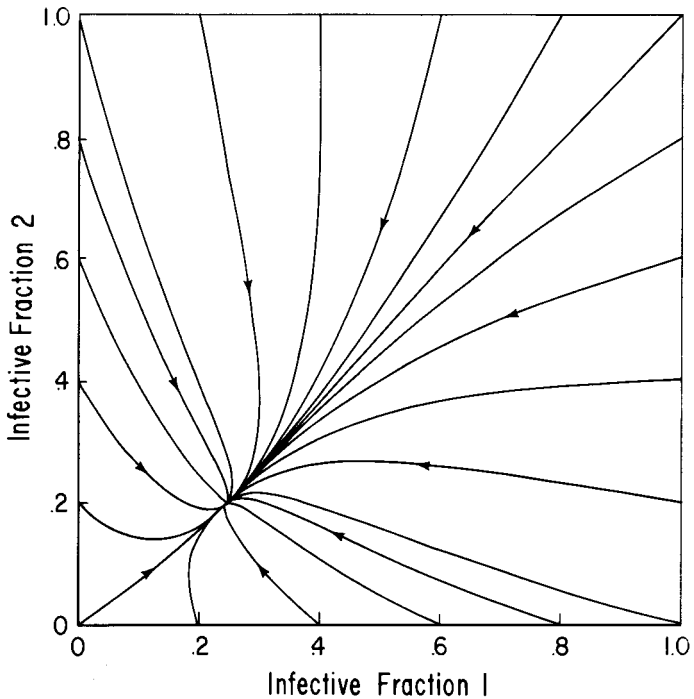
FIG. 5. Typical $I_1 I_2$ plane solution paths for an *SIS* model with two dissimilar groups using the values $N_1 = N_2$, $\lambda_{11} = 0.45$, $\lambda_{12} = \lambda_{21} = 0.1$, $\lambda_{22} = 0.375$, $\gamma_1 = \gamma_2 = 0.4$, $\delta_1 = 0.0005$, and $\delta_2 = 0.001$ in (8.1).

The zero in addition to the origin of this parabola is $1 - 1/\sigma - \theta/\lambda_1 N_1$. The right branch of (7.2) intersects the line $I_2 = 1$ at a point in $[0, 1)$. The second parabola is symmetrically similar with subscripts 1 and 2 interchanged throughout. If $a + d < 0$ and $ad - bc > 0$, then the origin is an attractor and is the only EP (intersection of the parabolas) in D. Since no solutions leave D, D is an ASR for the origin. Note that $a < 0$ and $d < 0$ in this case.

If $ad - bc < 0$, then the origin is a saddle point with repulsive direction into D and attractive direction not in D. If $a + d > 0$ and $ad - bc > 0$, then the origin is a repeller. In both of these cases, the parabolas intersect at exactly one EP in the interior of D. No solutions leave D, the index of this EP is $+1$, and there are no limit cycles in D by theorem 3.1 with $B = 1/(I_1 I_2)$. Thus the EP is an attractor, and D minus the origin is an ASR for this EP. This EP could be found numerically for particular parameter values. The case when $ad - bc = 0$ is resolved by analysis of the solution paths in the regions of D bounded by the parabolas.

8.   AN SIS MODEL FOR TWO DISSIMILAR GROUPS

A communicable disease model may be more realistic if it assumes that there are several interacting groups, each with different parameter values, where the subdivision is based on age or social behavior. Typically daily contact rates are higher for preschool and school children than for adults or for old people. Models with dissimilar groups are necessary, since changing a model by joining dissimilar groups together can change the asymptotic behavior (i.e., whether the disease remains endemic or fades away). Here we consider an SIS model with two dissimilar interacting groups and vital dynamics. The IVP is

$$I_1'(t) = (\lambda_{11}I_1 + \lambda_{12}N_2I_2/N_1)(1 - I_1) - \gamma_1 I_1 - \delta_1 I_1,$$

$$(8.1)$$

$$I_1(0) = I_{10} > 0, \qquad S_1 + I_1 = 1,$$

with similar equations for the other population group; here λ_{12}, λ_{21}, and $\gamma_1 + \delta_1 + \gamma_2 + \delta_2$ are positive.

The parameter λ_{ij} is the average number of contacts per day of an infective in the jth group with individuals in the ith group. Since $1/(\gamma_j + \delta_j)$ is the death-adjusted average infectious period for an individual in group j, $\sigma_{ij} = \lambda_{ij}/(\gamma_j + \delta_j)$ is the average number of group i individuals (both susceptibles and infectives) contacted by a group j infective during his infectious period. Let D be the region bounded by the axes, $I_1 = 1$, and $I_2 = 1$. Typical I_1I_2 plane portraits are given in Fig. 4 and 5. Let $a = \lambda_{11} - \gamma_1 - \delta_1$, $b = \lambda_{12}N_2/N_1$, $c = \lambda_{21}N_1/N_2$, $d = \lambda_{22} - \gamma_2 - \delta_2$.

THEOREM 8.1

For the differential equations in (8.1), if $a + d < 0$ and $ad - bc \geq 0$, then D is an ASR for the origin; otherwise, there is a unique EP in the interior of D, and $D - \{(0,0)\}$ is an ASR for this EP.

BIOCOROLLARY 8.1

For a disease without immunity in two dissimilar groups, if the infectious contact numbers (σ_{11} and σ_{22}) are less than 1 in both groups, then the interaction can cause the disease to remain endemic in both groups. If one infectious contact number is below 1 and one is above 1, then the interaction causes the disease to remain endemic in both groups.

The proof of theorem 8.1 is similar to the proof of theorem 7.1 except that the equilibrium points are at the intersection points of two hyperbolas. Lajmanovich and Yorke [20] consider a model for gonorrhea in a non-homogeneous population which is actually an *SIS* model for n dissimilar groups and consequently could be used for other diseases. Theorem 8.1 is a special case of their result, with more details.

9. SIS MODELS WITH VECTORS

If a communicable disease exists in two species and individuals in each species can be infected only by the other, then it is called a host-vector disease. Some host-vector diseases without immunity are malaria (mosquitoes), filariasis (mosquitoes), onchocerciasis (black flies), and plague (fleas). Since mosquitoes do not recover from malaria during their lifetime, their recovery rate γ is zero, so that the vector part of the malaria model is actually an *SI* model. Gonorrhea with men and women as the two populations could be interpreted as a host-vector *SIS* disease [20]. The model (8.1) for two dissimilar groups is a host-vector model if λ_{11} and λ_{22} are zero. This model was analyzed in [12], where the EP in the interior of D was found explicitly.

THEOREM 9.1.

For the differential equations in (8.1) with $\lambda_{11} = \lambda_{22} = 0$, if $\sigma_{12}\sigma_{21} \leqslant 1$, then D is an ASR for the origin. If $\sigma_{12}\sigma_{21} > 1$, then D minus the origin is an ASR for the EP

$$\left(\frac{\sigma_{12}\sigma_{21} - 1}{\sigma_{12}\sigma_{21} + \lambda_{21}N_1/(\gamma_2 + \delta_2)N_2}, \quad \frac{\sigma_{12}\sigma_{21} - 1}{\sigma_{12}\sigma_{21} + \lambda_{12}N_2/(\gamma_1 + \delta_1)N_1} \right). \quad (9.1)$$

BIOCOROLLARY 9.1

For a host-vector disease with no immunity, if the product of the two infectious contact numbers is less than one, then the disease eventually disappears; otherwise, the disease remains endemic.

Some diseases have both human and nonhuman hosts such as monkeys, rodents, pigs, etc. The IVP for a host-vector-host model (i.e., with two hosts)

is

$$I_1'(t) = \lambda_{12} N_2 I_2 (1 - I_1) / N_1 - \gamma_1 I_1 - \delta_1 I_1,$$

$$I_1(0) = I_{10} > 0, \qquad S_1 + I_1 = 1,$$

$$I_2'(t) = (\lambda_{21} N_1 I_1 + \lambda_{23} N_3 I_3)(1 - I_2) / N_2 - \gamma_2 I_2 - \delta_2 I_2, \qquad (9.2)$$

$$I_2(0) = I_{20} > 0, \qquad S_2 + I_2 = 1,$$

$$I_3'(t) = \lambda_{32} N_2 I_2 (1 - I_3) / N_3 - \gamma_3 I_3 - \delta_3 I_3,$$

$$I_3(0) = I_{30} > 0, \qquad S_3 + I_3 = 1,$$

Let D be the region $0 \leqslant I_1, I_2, I_3 \leqslant 1$, and let

$$p = \gamma_1 + \delta_1 + \gamma_2 + \delta_2 + \gamma_3 + \delta_3,$$

$$q = (\gamma_1 + \delta_1)(\gamma_2 + \delta_2) + (\gamma_2 + \delta_2)(\gamma_3 + \delta_3) + (\gamma_1 + \delta_1)(\gamma_3 + \delta_3)$$

$$- \lambda_{23} \lambda_{32} - \lambda_{21} \lambda_{12},$$

$$r = (\gamma_1 + \delta_1)(\gamma_2 + \delta_2)(\gamma_3 + \delta_3) - \gamma_1 \lambda_{23} \lambda_{32} - \gamma_3 \lambda_{21} \lambda_{12}.$$

THEOREM 9.2

For the differential equations in (9.2), *if* $p > 0$, $r > 0$, *and* $pq > r$, *then* D *is an ASR for the origin. Otherwise, there is a unique EP in the interior of* D, *and* D *minus the origin is an ASR for this EP.*

Theorem 9.2 follows from the result of Lajmanovich and Yorke [20] and the Routh-Hurwitz criteria.

10. OTHER COMMUNICABLE DISEASE MODELS

Another type of communicable disease model is the *SEIR* model, where E is a class of exposed individuals, who are in the latent period. Various assumptions regarding the length of the latent and infective periods lead to delay differential equations, functional differential equations, and integral equations [28, 4, 14, 10, 15, 27, 29, 5, 26]. One obvious question is whether the solution behaviors for these models are essentially different from those of the ordinary differential equation models considered here. Computer simulation models for various diseases have been used [8]. Clearly, deterministic, stochastic, and simulation models are interrelated, and conclusions resulting

from one type of model have implications for the analogous models of the other types [22].

The control of communicable diseases is an important practical problem. If communicable disease models can be developed so that epidemiologists have some confidence in their predictive ability, then these models can be used in the cost-effectiveness evaluation of various control measures. Models involving control of diseases by vaccination have been considered [8, 25, 24, 13, 9].

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