ROCKY MOUNTAIN
JOURNAL OF MATHEMATICS
Volume 24, Number 1, Winter 1994

THE INFLUENCE OF DISEASES ON LOTKA-VOLTERRA SYSTEMS

EZIO VENTURINO

Dedicated to Paul Waltman on the occasion of his 60th birthday

ABSTRACT. Single species population dynamics have been studied since the past century, in the first researches of Verhulst. The first interacting species model was proposed by Volterra and then also studied by Lotka. On the other hand, the classical model that considers epidemics in a population was proposed by Kermack and McKendrick. Although the two fields have been the subject of widespread research in recent years, hardly any work has been done to study the effect of a disease on an environment where two competing species are present.

Here we analyze multiple modifications of the basic Lotka-Volterra model, to account for a disease spreading among one of the two species. We choose the simplest epidemiological models, the SI and SIS, where only susceptibles and infectives are counted. We analyze two different types of incidences, simple mass action and the standard incidence. The results seem to indicate that either the disease dies out, leaving only "neutral" cycles of the Lotka-Volterra system or one species disappears and all individuals in the other one eventually become infected. For some particular choices of the parameters, however, endemic equilibria in which both populations survive seem to arise.

1. Introduction. The study of interacting species had already begun in the first part of the century. It has received a renewed interest in the past 15 years in the mathematical literature. Epidemiology models are routinely used nowadays to understand the spread of infectious diseases with the goal to determine vaccination policies to possibly eradicate them.

To our knowledge, with the exception of [**5**], no model has been proposed to merge the two phenomena. Very recently, epidemic models which account for a varying population size have been proposed [**11**]. The population dynamics introduced in these models are immigration,

Received by the editors on March 3, 1993.

deaths, and births proportional to population sizes. In [**4**] these are modified by allowing a carrying capacity, i.e., introducing a modification of the logistic equation.

In this paper we propose and investigate several simple models for studying the spread of diseases among competing species. It is our goal to consider one of the simplest possible predator-prey models, the so-called Lotka-Volterra. We want to couple it with the basic epidemiology models, namely the SI and the SIS.

The Lotka-Volterra model has the drawback of exhibiting neutral type oscillations around the equilibrium point, but from our point of view is attractive because it minimizes the number of parameters we have to deal with. For the same reason, we discard from this investigation epidemiology models with more "stages" for the disease, i.e., SIRS or SEIRS.

To keep matters simple, we account for the two interacting species and with one extra variable we consider the infected individuals in one of the two species. We do not allow infected individuals both among prey and predatory, but we study the two cases independently; in Sections 3 and 4 the disease spreads among the prey, in Sections 5 and 6 it affects only the predators. Two different types of ways in which individuals contract the disease are assumed. In the literature they are known as mass action and standard incidence. The former states that new infectives are "generated" by random encounters at a rate proportional to the population size of infectives and susceptibles. The latter assumes, in addition, that the contact rate is not constant but inversely proportional to the total population size. We discuss the former in Sections 3 and 5 and the latter in Sections 4 and 6. Overall, eight models are studied in the paper. The basic conclusion we can draw is that they inherit essentially the dynamics of the Lotka Volterra system. What is useful in the formulation turns out to be disappointing in the analysis, because no dramatic effects on the long term behavior of the system, with the exception of two models, seem to arise form the introduction of the disease in the predator-prey model. This perhaps suggests either that more computational work needs to be performed to simulate these models, or that more sophisticated models need to be introduced.

The paper is organized as follows. In the next section we formulate the basic models. In Sections 3 to 6 we examine the SI and SIS models, for each combination of incidences and species affected by the disease, as described earlier. A final discussion concludes the paper.

As far as the notation is concerned, we use capital letters to denote the number of individuals in each population, R being the prey (rabbits), F the predators (foxes), U the infected prey and V the infected predators. The corresponding lower case letters denote the perturbations of these variables about equilibria.

All the coefficients in the models will be positive real numbers, with the exception of h, the "value of predation upon infected prey" which will be analyzed respectively in the two cases corresponding to the usual predator-prey interaction, and to predation which causes harm to the predator. In the latter case, we are implicitly assuming that, by catching an infected prey, the predatory gets the disease and dies of it.

Coefficients directly linked with the spread of the disease will be denoted by greek letters; the ones related to the predator-prey model by latin letters.

2. Modeling the disease spread among the prey. The Lotka-Volterra model for predator-prey interaction is given by:

$$\begin{aligned} \dot{R} &= aR - cRF \\ \dot{F} &= -bF + dRF. \end{aligned} \tag{2.1}$$

The assumptions underlying this model are as follows. The habitat for the prey is assumed to be unlimited, so that in absence of predators the prey will reproduce exponentially. The predators survive only on the prey. In the absence of food, their number will decrease exponentially. Interactions among individuals in the two populations result in loss for the prey and a gain for the predators, denoted respectively by the coefficients c and d.

The simple SIS model in epidemiology is

$$\begin{aligned} \dot{S} &= -\lambda(N)IS + \gamma I + \mu - \mu S \\ \dot{I} &= \lambda(N)IS - \gamma I - \mu I \end{aligned} \tag{2.2}$$

where S and I denote the fractions of susceptibles and infectives in the population of size N, so that $S + I = 1$. The incidence function

$\lambda(N)$ is usually modeled either as a constant, $\lambda(N) \equiv \lambda$, thus leading to a simple mass action, or as a nonlinear function, $\lambda(N) \equiv \lambda/N$, the standard incidence [**4, 11**]. The parameters γ and μ denote respectively the recovery rate from infective to susceptible, and the mortality (and birth) rate. If $\gamma = 0$, the model becomes a simple SI model.

Our task here is to combine the preceding models. We assume in this section that the disease spreads only among the prey; U denotes the total number of infected prey. To keep the model simple we assume that infected prey do not reproduce, and that there is no disease related mortality. Finally, we model the incidence rate by a simple mass action.

Under these assumptions we are led to

$$\begin{aligned} \dot{R} &= R(a - cF - \lambda U) + \gamma U \\ \dot{U} &= U(\lambda R - gF - \gamma) \\ \dot{F} &= F(-b + dR - hU) \end{aligned} \tag{2.3}$$

with suitable initial conditions. We will consider at first only the SI model, i.e., suppose $\gamma = 0$. Notice that the coefficient h is unrestricted in sign. For $h < 0$ we will get a positive value for predation, corresponding to the usual Lotka-Volterra model.

This case allows both the case $0 < -h < d$, which says that infected prey have less nutritional value than sound ones, and the case $-h > d$, which expresses the fact that sick prey are easier to catch. Also, for $h > 0$ we get a more interesting model, suggesting that a predator which comes in contact with an infected prey might become sick from eating diseased prey and eventually die of it. In such a case we also assume that these sick predators are unable to hunt, so that we can remove them from consideration, to keep the model simple.

A second model for the spread of the disease among the prey is obtained from the same above considerations, by replacing the mass action incidence by a nonlinear function. In this case, however, we need to take into account also the total number N of prey, susceptible or infected; by letting X and Y denote the fractions of susceptible and infective prey, we then have

$$N = R + U, \qquad X = R/N, \qquad Y = U/N. \tag{2.4}$$

The major difference of the present formulation with respect to the former one, is the fact that we need to take into account the dynamics

of the whole prey population. The model has a redundant equation:

$$\begin{aligned}\dot{R} &= R(a - cF - \lambda U/N)\\ \dot{U} &= U(\lambda R/N - gF)\\ \dot{F} &= F(-b + dR - hU)\\ \dot{N} &= \dot{R} + \dot{U} = R(a - cF) - gUF.\end{aligned} \tag{2.5}$$

Dividing the first two equations by N and using the definitions of X and Y, we are led to

$$\begin{aligned}\dot{X} &= X[a - cF - \lambda Y - (aX - cFX - gYF)]\\ \dot{Y} &= Y[\lambda X - gF - (aX - cFX - gYF)]\\ \dot{F} &= F(-b + dXN - hYN)\\ \dot{N} &= N(aX - cFX - gYF).\end{aligned}$$

On these variables, the restrictions are:

$$X + Y = 1, \qquad X, Y, N, F \geq 0.$$

One of the fractions of the prey subpopulations can then be ignored. Further simplifications lead to:

$$\begin{aligned}\dot{Y} &= Y(1 - Y)(r - zF)\\ \dot{F} &= F[-b + dN - (d + h)YN]\\ \dot{N} &= N[a - cF - Y(a + zF)]\end{aligned} \tag{2.6}$$

with

$$0 \leq Y \leq 1; \qquad N, F \geq 0; \qquad z = g - c \geq 0, \quad r = \lambda - a.$$

Notice that the requirement $z \geq 0$ is natural, since it amounts to requiring that infected prey are more likely to succumb to an attack than sound individuals are.

Proceeding in a similar fashion, we can also obtain the SIS version of the above model:

$$\begin{aligned}\dot{Y} &= Y[(1 - Y)(r - zF) - \gamma]\\ \dot{F} &= F[-b + dN - (d + h)YN]\\ \dot{N} &= N[a - cF - Y(a + zF)]\end{aligned} \tag{2.7}$$

with

$$0 \leq Y \leq 1, \qquad N, F \geq 0.$$

3. Analysis of the models with mass action incidence. We consider at first (2.3) with $\gamma = 0$. The first step consists of finding the equilibria $E_i \equiv (R_i, U_i, F_i)$. These are:

$$\begin{aligned} &E_0 = (0,0,0) && E_2 = (0, U_2, 0) \\ &E_1 = \left(\frac{b}{d}, 0, \frac{a}{c}\right) && E_3 = \left(\frac{g(ah+b\lambda)}{\lambda(ch+dg)}, \frac{adg-cb\lambda}{\lambda(ch+dg)}, \frac{ah+b\lambda}{ch+dg}\right) \end{aligned} \tag{3.1}$$

Linearization about the origin shows easily that it behaves essentially like a saddle, with increasing R, decreasing F, and U remaining constant.

To understand the behavior around E_2, let us study a perturbation of this equilibrium point. Let

$$r = R - R_2 \equiv R, \qquad u = U - U_2, \qquad f = F - F_2 \equiv F.$$

Substitution into (2.3) and dropping of higher order terms leads to the system

$$\dot{\underline{x}} = A\underline{x} \tag{3.2}$$

where $\underline{x} = (r, u, f)$ and

$$\begin{aligned} &A_{11} = a - \lambda U_2 && A_{21} = \lambda U_2 && A_{23} = -gU_2 \\ &A_{33} = -(b + hU_2) && A_{ij} = 0 \end{aligned} \tag{3.3}$$

for any other pair of indices $i, j = 1, 2, 3$. The eigenvalues of A are $0, -(b + U_2 h)$, $a - \lambda U_2$. Both F and R tend exponentially to zero if $h > -b/U_2$, $a < \lambda U_2$, the former being always verified if $h > 0$. Under these conditions, U tends exponentially to the arbitrary value U_2.

Proposition 1. *If*

$$h > -b/U_2, \qquad a < \lambda U_2 \tag{3.4}$$

the equilibrium point E_2 is locally asymptotically stable.

Notice that $(0, U_2, 0)$ is actually a neutrally stable line of equilibrium points, since one of the above eigenvalues is zero.

For E_1, let

$$r = R - R_1, \qquad u = U - U_1 \equiv U, \qquad f = F - F_1$$

to get (3.2) with

$$\begin{aligned} &A_{12} = -\lambda b/d \qquad A_{13} = -bc/d \qquad A_{22} = \frac{b\lambda}{d} - \frac{ga}{c} \\ &A_{31} = ad/c \qquad A_{32} = -ah/c. \end{aligned} \tag{3.5}$$

$A_{ij} = 0$ otherwise. The eigenvalues are $b\lambda/d - ga/c$, and the pure imaginary values are $\pm(ab)^{1/2}i$. If $bc\lambda < adg$, U approaches zero exponentially, i.e., the disease extinguishes and the model exhibits the neutrally stable cyclic behavior proper of the Lotka-Volterra system.

The equilibrium E_3 is feasible if $h > 0$ provided $adg > bc\lambda$. if $h < 0$, it is in the two cases $h > -b\lambda/a > -gd/c$ or $h < -b\lambda/a < -gd/c$.

The linearization procedure in this case yields the characteristic polynomial

$$\begin{aligned} P(\mu) &= +\mu^3 + a_1\mu + a_0 \\ a_1 &= -ghF_3U_3 + cdF_3R_3 + \lambda^2 U_3R_3 \\ a_0 &= -U_3F_3R_3\lambda(gd + hc). \end{aligned} \tag{3.6}$$

For $h > 0$, it follows that $a_0 < 0$ so that the equilibrium E_3 cannot be stable. If $h < 0$, again instability occurs if $h > -gd/c$. For the remaining feasible case, $h < -b\lambda/a < -gd/c$, we look at the explicit solutions of the cubic equation. Observe that $a_1 > 0$ and therefore

$$Q \equiv \frac{a_1^3}{27} + \frac{a_0^2}{4} > 0$$

so that the cubic has one real root and two complex conjugate ones; combining this information with the fact that in the above representation of $P(\mu)$ the coefficient of the quadratic term is zero, we immediately obtain that the real root has sign differing from the sign of the real part of the other roots. Hence instability follows again.

Proposition 2. *The equilibrium point E_3 is always unstable.*

In conclusion, model (2.3) with $\gamma = 0$ does not sustain an equilibrium in which the disease is endemic with both species surviving. The only possible stable equilibria are given by the disappearance of all predators, all prey becoming infected, or by the disappearance of the disease, leading to neutrally stable cycles of the type found in the Lotka-Volterra model.

We now turn to the analysis of (2.3) with $\gamma \neq 0$. The equilibria E_0 and E_1 found earlier are equilibria here also. E_0 is again a saddle, and E_1 shows local neutral stability under the relaxed condition $\lambda b/d < ag/c + \gamma$.

The remaining equilibria are found from the quadratic:

$$q(R) \equiv -\lambda(ch + gd)R^2 + [(ag + c\gamma)h + g(\lambda b + \gamma d)]R - g\gamma b = 0. \tag{3.7}$$

For $h > 0$, its discriminant is positive and the coefficients in (3.7) have two variations in sign, i.e., the two roots for R are both feasible. For $h < 0$ the discriminant of (3.7) is in turn a trinomial of second degree in h, with discriminant $16g^3ab\lambda D$,

$$D = (ag + c\gamma)(\gamma d - \lambda b) + abg\lambda. \tag{3.8}$$

For $D < 0$, (3.7) again has two real roots, for $D > 0$, we need to take h external to the interval of the roots of the quadratic equation obtained from the discriminant of (3.7). By combining this analysis with Descartes' rule applied to (3.7), we are led to the following table. Here $h_1 = -(gD+D^{1/2})/(ag+c\gamma)^2 < 0$, $h_2 = (-gD+D^{1/2})/(ag+c\gamma)^2$, $h_3 \equiv -gd/c$, $h_4 \equiv -g(b\lambda + d\gamma)/(ag + \gamma c)$, and $R_{2,3}$ or R_3 in a certain interval denote that for h in that interval, there are respectively two and one feasible root for (3.7). For $D > g^{-2}$, only the following situations can occur

$$\begin{aligned}
&h_3 < R_3 < h_4 < R_{2,3} < h_1 < h_2 < R_{2,3} < 0\\
&h_4 < R_3 < h_3 < R_{2,3} < h_1 < h_2 < R_{2,3} < 0\\
&h_3 < R_3 < h_1 < h_4 < h_2 < R_{2,3} < 0\\
&h_1 < h_3 < h_4 < h_2 < R_{2,3} < 0\\
&h_1 < h_4 < h_3 < h_2 < R_{2,3} < 0
\end{aligned} \tag{3.9}$$

$$h_1 < h_4 < h_2 < R_3 < h_3 < R_{2,3} < 0$$
$$h_4 < R_3 < h_1 < h_2 < R_3 < h_3 < R_{2,3} < 0$$
$$h_4 < R_3 < h_1 < h_3 < h_2 < R_{2,3} < 0.$$

For $0 < D < g^{-2}$, we have instead:

$$\begin{aligned} &h_4 < R_3 < h_3 < R_{2,3} < h_1 < 0 \\ &h_3 < R_3 < h_4 < R_{2,3} < h_1 < 0 \\ &h_3 < R_3 < h_1 < h_4 < 0 \\ &h_4 < R_3 < h_1 < h_3 < 0 \end{aligned} \tag{3.10}$$

with other combinations leading only to infeasible solutions. Finally, notice that if $D < 0$, then there are always two feasible roots, $R_{2,3}$.

The equilibria are then easily found to be

$$E_{2,3} = (R_{2,3}(dR_{2,3} - b)/h, (\lambda R_{2,3} - \gamma)/g). \tag{3.11}$$

We need to ensure also that the other two variables are feasible, $U_{2,3} \geq 0$, $F_{2,3} \geq 0$, giving

$$R_{2,3} \geq \max(b/d, \gamma/\lambda) \equiv \widetilde{M}. \tag{3.12}$$

Detailed examination of these conditions leads to the following picture:

If $b\lambda > \gamma d$, E_3 is feasible and E_2 is not if

$$h > 0, \quad \frac{dag}{c} > \lambda b - \gamma d \quad \text{or}$$
$$h < 0, \quad \frac{dag}{c} < \lambda b - \gamma d.$$

If $b\lambda > \gamma d$, both E_2 and E_3 are feasible if

$$h > 0, \quad \frac{dag}{c} < \lambda b - \gamma d \quad \text{and}$$
$$\frac{b\lambda}{ch}(gd + ch) + (b\lambda - d\gamma) < \frac{adg}{c} + \frac{d^2 g\gamma}{ch},$$

or in case all three previous inequalities are reversed.

Finally, if $b\lambda < \gamma d$, $h > 0$ implies E_3 feasible.

In all other cases the two points E_2 and E_3 will be infeasible.

An alternative, clearer picture of the situation is obtained geometrically. Solving the second equation (2.3) for F in terms of R and substituting into the first equation yields the hyperbola

$$U = R\frac{c\lambda R - (c\gamma + ga)}{g(\gamma - \lambda R)}.$$

This is positive for $R \geq 0$ only for

$$\frac{\gamma}{\lambda} \leq R \leq \frac{\gamma c + ga}{\lambda c}.$$

For $h > 0$, one intersection with the remaining equation, which gives the straight line $dR - hU = b$, is guaranteed if we require

$$\frac{b}{d} < \frac{\gamma c + ga}{\lambda c}.$$

If $h < 0$, the intersection is guaranteed by the converse condition

$$\frac{b}{d} > \frac{\gamma c + ga}{\lambda c}.$$

In this case, however, there could be no intersections, or also two intersections, if the slope of the hyperbola at the point $R^{**} = (\gamma c + ga)/(\lambda c)$ is larger than the slope of the straight line, i.e.,

$$-c/g\left[1 + \frac{\gamma c}{ga}\right] \geq d/h.$$

These two feasible solutions are in the interval $[R^*, R^{**}]$, provided that $R^* < b/d \leq R^{**}$, where

$$R^* = (\gamma + \alpha)/\lambda$$

denotes the abscissa of the point where the line is tangent to the hyperbola, and

$$\alpha = \left[\frac{-h\gamma ag}{ch + dg}\right]^{1/2}.$$

To ensure that the two roots exist, we can just require that the line at R^* be above the hyperbola,

$$g(d\gamma - b\lambda) - h(c\gamma - ag) < 0.$$

For the stability analysis for $h > 0$, evaluating the characteristic polynomial at the origin shows a real positive eigenvalue. E_i is then unstable. The same situation occurs if

$$0 > h > -\frac{g^2 dF_i R_i}{\lambda c R_i^2 + g\gamma U_i} \equiv K_i. \tag{3.13}$$

If $h < K_i < 0$, we need a more detailed analysis of the eigenvalues, but we omit the discussion.

In conclusion, the SIS model applied to the Lotka-Volterra system does not seem to lead to stable equilibria where all three subpopulations survive, in the most interesting case $h > 0$.

If h is negative, more complicated situations for studying the stability of the equilibria arise.

4. Analysis of the models with standard incidence. We sketch the study of (2.6) and then perform a more detailed investigation for (2.7). It is immediately seen that four equilibria $P_i \equiv (N_i, F_i, Y_i)$, $i = 0, 1, 2, 3$, exist for (2.6). P_0 is just the origin and it cannot be stable since one of the eigenvalues is $a > 0$. For the remaining points, we have

$$P_1 = \left(\frac{b}{d}, 0, \frac{a}{c}\right)$$
$$P_2 = (N_2, 1, 0)$$
$$P_3 = \left(\frac{bz\lambda}{rdg + hcr - haz}, \frac{az - rc}{z\lambda}, \frac{r}{z}\right).$$

Linearizing about P_1, we get explicitly the eigenvalues

$$r - \frac{az}{c}, \qquad i\sqrt{ab}, \qquad -i\sqrt{ab}.$$

The parameters r and z have been defined in terms of the original parameters of the system. For z nonnegativity is a natural requirement,

but for r no such restriction exists. Thus, r and h are the only parameters unrestricted in sign.

P_1 is then a "stable" equilibrium for $r < az/c$. The disease dies out exponentially, and the system exhibits neutral type oscillations around (N_1, F_1).

P_2 represents actually a line of equilibria, since N_2 is arbitrary. (3.2) in this case yields

$$\begin{aligned} &A_{12} = -N_2 g && A_{13} = -N_2 a \\ &A_{22} = -(b + N_2 h) && A_{33} = -r \\ &A_{ij} = 0 \text{ otherwise.} \end{aligned} \tag{4.1}$$

The eigenvalues are $0, -(b+N_2h), -r$. The equilibrium line is therefore stable if $h > -b/N_2$, $r > 0$. It is unstable in any other case.

The feasibility requirements for P_3 are $0 \leq r \leq az/c$ and $h \leq rdg/(az - cr)$. The linearized system has the eigenvalues which can be written in terms of complicated expressions of the parameters of the system, p_1, p_2, as

$$p_1 + p_2, \qquad -\frac{1}{2}(p_1 + p_2) + \frac{\sqrt{3}}{2} i(p_1 - p_2),$$
$$-\frac{1}{2}(p_1 + p_2) - \frac{\sqrt{3}}{2} i(p_1 - p_2).$$

This is enough to show that stability cannot be achieved, since the first two eigenvalues have real parts with opposite signs.

In summary, the only nontrivial equilibrium point P_3 is always unstable. The disease dies out locally and the system has the neutral equilibrium P_1 for $r < az/c$. The line of equilibrium point P_2 is locally stable for $h, r > 0$. For h negative, it is stable if $h > -b/N_2$, $r > 0$. In such cases the predators die out and every prey ultimately contracts the disease.

We now analyze the SIS model with standard incidence. Again we find the equilibria P_0 and P_1, with P_0 a saddle. The analysis for P_1 gives slightly different eigenvalues:

$$r - \gamma - \frac{az}{c}, \qquad +i\sqrt{ab}, \qquad -i\sqrt{ab}.$$

The neutral stability requirement here is more restrictive, namely, $r > az/c + \gamma$. Apart from these, there are three other equilibria

$$P_4 = \left(0, 1 - \frac{\gamma}{r}, 0\right)$$
$$P_i = \left(\frac{b}{d(1-Y_i) - hY_i}, Y_i, \frac{r(1-Y_i) - \gamma}{z(1-Y_i)}\right), \qquad i = 5, 6$$

where Y_i is either solution of the quadratic equation

$$P(Y) \equiv \lambda z Y^2 + [cr - az + z(\gamma - \lambda)]Y + c(\gamma - r) + az = 0. \tag{4.2}$$

The equilibrium P_4 is trivial in the sense that both predators and prey die out, but the disease remains endemic. In order for it to be feasible, we need to require

$$0 \leq 1 - \frac{r}{\gamma} \leq 1.$$

This is verified if and only if $r \geq \gamma$. The matrix corresponding to the linearized system about P_4 is lower triangular and its eigenvalues are immediately obtained

$$\frac{a\gamma}{r}, \qquad -b, \qquad \gamma - r.$$

P_4 cannot be a stable equilibrium since the first eigenvalue is positive.

The feasibility analysis for P_5 and P_6 tells us that Y_5 and Y_6 are real if

$$\Delta \equiv [cr - az + z(\gamma - \lambda)]^2 - 4\lambda z(c(\gamma - r) + az) \geq 0. \tag{4.3}$$

Given the above condition, since the coefficient of Y^2 in $P(Y)$ is positive, to get two positive roots we need to require only $P(0) > 0$, $P'(0) < 0$, i.e.,

$$cr - az < \min(c\gamma, z(\lambda - \gamma)). \tag{4.4}$$

For the roots to be less than one, it is enough that the sum of the coefficients be positive. In turn, this yields $(z + c)\gamma > 0$, which is obviously verified.

Also, the condition $N_i \geq 0$, $i = 5, 6$, gives

$$h \leq d\left(\frac{1}{Y_i} - 1\right) \tag{4.5}$$

and $F_i \geq 0$, $i = 5, 6$, yields

$$r \geq \gamma/(1 - Y_i). \tag{4.6}$$

Notice that if (4.4) is not verified, but $P'(0) \leq 0$ and $P(0) \leq 0$, then only P_6 is feasible.

Let us analyze the stability of these equilibria. Linearization about P_i, $i = 5, 6$, yields the system (3.2), again where $\underline{x} = (n, y, f)$ and

$$\begin{aligned} &A_{12} = -(a + zF_i)N_i && A_{13} = -(c + zY_i)N_i \\ &A_{22} = -(r + zF_i)Y_i && A_{23} = -zY_i(1 - Y_i) \\ &A_{31} = F_i(d(1 - Y_i) - hY_i) && A_{32} = -(d + h)N_iF_i \\ &A_{ij} = 0 \text{ otherwise.} \end{aligned} \tag{4.7}$$

Notice that all A_{ij} are negative, in particular A_{31} is, by virtue of the feasibility condition (4.5). The Routh-Hurwitz test is necessary and sufficient for a cubic polynomial. The characteristic polynomial here is

$$\begin{aligned} \tilde{Q}(u) = \mu^3 - A_{22}\mu^2 + \mu(A_{23}A_{32} + A_{13}A_{31}) \\ + A_{31}(A_{13}A_{22} - A_{12}A_{23}). \end{aligned}$$

The criterion then becomes

$$\begin{gathered} -A_{22} > 0, \\ \tilde{\Delta} = -A_{22}(A_{23}A_{32} + A_{13}A_{31}) - A_{31}(A_{13}A_{22} - A_{12}A_{23}) > 0, \\ A_{31}(A_{13}A_{22} - A_{12}A_{23})\tilde{\Delta} > 0. \end{gathered}$$

The first condition is obviously true. The second one can be reduced to the following statement, in terms of the parameters of the model

$$a(d(1 - Y_i) - hY_i) + zF_id > Y_ir(d + h) + 2zF_iY_i(d + h). \tag{4.8}$$

The third one can be reduced in a similar way to

$$cr + czF_i + zrY_i + 2z^2F_iY_i > za(1 - Y_i) + z^2F_i. \tag{4.9}$$

These conditions are stated in terms of the coordinates of the equilibrium points, but these in turn are given in terms of the original parameters of the model. In summary, we have eight free parameters in (2.7). To be feasible and stable, the equilibria P_5 and P_6 require conditions (4.3)–(4.9) to be satisfied. Since these are six, there is always the possibility of satisfying them since we have more free parameters.

It thus follows that either P_5 or P_6 is a nontrivial equilibrium where the disease persists endemically, at the level of the fraction Y_i of infectives.

Also, observe that $N_i > N_1$, $i = 5, 6$, since this statement reduces to the condition $h > -d$, and it will be true if $h > 0$. In a similar way the inequality $F_i < F_1$, $i = 5, 6$, reduces to

$$\gamma c \geq (1 - Y_i)(rc - az),$$

which in turn is satisfied, using (4.4). Thus this model shows that an equilibrium where the two competing species both survive is possible. This equilibrium is stable and the levels of the two populations adjust so that the prey will increase with respect to the normal level attained at the equilibrium of the Lotka-Volterra model. The predators instead will move to a lower level than the one attained in the Lotka-Volterra model.

This is a remarkable result since we are assuming that the disease spreads only among the prey, but in a certain sense it allows us to "control" the size of the predators.

5. The epidemic among predators with mass action incidence. In this section we analyze the SIS model constructed by assuming that the disease spreads among the predators, where V denotes the size of the infected predators

$$\begin{aligned} \dot{R} &= R(a - cF - \eta V) \\ \dot{F} &= F(-b + dR - \delta V) + \nu V \\ \dot{V} &= V(\delta F + eR - \nu). \end{aligned} \tag{5.1}$$

We are assuming that infected predators still can catch prey, at a different rate η than the sound ones. The parameter η can be thought to be less than c, if the disease affects the ability in hunting of the

predators, or larger than c, if we want to emphasize that the interactions with infected predators cause the prey to die for the disease even if they are not caught. All the parameters in this model are nonnegative.

For the SI case, the equilibria are

$$Q_0 = (0,0,0) \qquad Q_2 = (b/d, a/c, 0)$$
$$Q_1 = (0,0,V_1) \qquad Q_3 = \left(R_3, -\frac{e}{\delta}R_3, \frac{a\delta d + ceb}{\delta(d\eta - ce)}\right)$$

with

$$R_3 = \frac{b\eta + a\delta}{d\eta - ce}.$$

Here Q_0 is again a saddle, since one eigenvalue is $a > 0$. The line of trivial equilibria Q_1 where no species survives has interest only because the disease in the system remains endemic. Local stability holds if $V_1 \geq a/\eta$; the trajectories lie in the $V = V_1$ plane, where V_1 is an arbitrary value.

The equilibrium Q_2 corresponds to the neutrally stable equilibrium for the Lotka-Volterra system, but it is always unstable, one eigenvalue being $(ad\delta + ceb)/(cd)$. The orbits may spiral around it in the RF plane, but along the V axis they are repelled away from it.

Finally, the equilibrium Q_3 is never feasible, due to the relationship between F_3 and R_3.

For the SIS model, there are two other equilibria, apart from Q_0 and Q_2:

$$Q_i = \left(\frac{\nu - \delta F_i}{e}, F_i, \frac{a - cF_i}{\eta}\right), \qquad i = 4, 5,$$

and F_i solves the quadratic:

$$Q(F) \equiv \delta(ec - d\eta)F^2 + F[\nu(d\eta - ec) - e(\delta a + b\eta)] + \nu ea = 0. \tag{5.2}$$

Here Q_0 is again a saddle, but Q_2 may be a stable equilibrium, if $\nu > (a\delta d + ebc)/(cd)$.

Rather than carrying out the feasibility analysis for Q_4 and Q_5 by algebraic means as done in Section 3 for the SIS model, we can obtain it geometrically as follows. It is easily observed that, by solving for R in terms of F from the equation for $\dot{V} = 0$, we obtain

$R = (\nu - \delta F)/e$. Substitution into the equation for $\dot{F} = 0$ gives the hyperbola $V(F) = (ebF)/[e(\nu - \delta F)] - dF$, which must be intersected with the straight line $a - cF - \eta V = 0$. The hyperbola is positive for $\nu/\delta \geq F \geq \max[0, (\nu - b/d)/\delta]$. In order to obtain one feasible solution, however, the additional condition $a/c \geq \max[0, (\nu - b/d)/\delta]$ must be satisfied. Observe also that two feasible solutions are never simultaneously possible.

For the stability analysis it is easy to see that the constant term of the characteristic polynomial is negative, so that a positive eigenvalue exists. Hence, Q_i, $i = 4, 5$, is always unstable.

6. Disease in the predators with standard incidence. In this final section we reformulate the model (5.1) by introducing the fractions of the total predator population $M = F + V$; then let $W = V/M$ and $Z = F/M = 1 - W$. The system to be investigated is

$$\begin{aligned} \dot{R} &= R[a - cM(1 - W) - \eta MW] \\ \dot{W} &= W[(1 - W)(b + \delta + (e - d)R) - \nu] \\ \dot{M} &= M[(dR - b)(1 - W) + eRW]. \end{aligned} \tag{6.1}$$

Again, here all the parameters are nonnegative. The SI model, for $\nu = 0$, has the equilibria

$$\begin{aligned} T_0 &= (0, 0, 0), & T_2 &= (0, 1, M_2) \\ T_1 &= \left(\frac{b}{d}, 0, \frac{a}{c}\right), & T_3 &= \left(\frac{\delta + b}{d - e}, \frac{be + d\delta}{\delta(d - e)}, M_3\right) \end{aligned}$$

with

$$M_3 = \frac{-a\delta(d - e)}{\delta(ec - \eta d) + eb(c - \eta)}.$$

Again T_0 and T_1 are unstable.

The equilibrium T_2 is again trivial, but interesting, because it shows that every predator eventually becomes infected. It is stable if $M_2 > a/\eta$. The trajectories lie in the plane $M = M_2$, with M_2 arbitrary. The prey die out and all predators become infected.

T_3 is always infeasible since $W_3 \leq 1$ yields

$$\frac{e(b + \delta)}{\delta(d - e)} \leq 0,$$

which cannot be satisfied since $R_3 \geq 0$ gives $d \geq e$.

The analysis of the SIS model with standard incidence yields the following equilibria, apart from T_0 and T_1

$$T_4 = \left(0, 1 - \frac{\nu}{b+\delta}, 0\right),$$
$$T_i = \left(\frac{b(1-W_i)}{d(1-W_i)+eW_i}, W_i, \frac{a}{c(1-W_i)+\eta W_i}\right); \qquad i = 5, 6,$$

where W_i is either root of the quadratic

$$N(W) \equiv \delta(e-d)W^2 + W[be + \delta d + (e-d)(\nu-\delta)] + d(\nu-\delta) - be = 0. \tag{6.2}$$

Again, the origin is a saddle. The real eigenvalue relative to the linearization about T_1 is

$$\delta + \frac{be}{d} - \nu.$$

It follows that T_1 can be "stable" provided that $\nu > \delta + be/d$. The trajectories approach the equilibrium along the W axis but then in the RM plane will describe neutrally stable cycles around it.

To ensure feasibility for T_4, we need to impose $1 \geq W_4 \geq 0$. The former inequality is trivial, but the latter gives $\nu < b + \delta$.

The matrix of the linearized system about T_4 is lower triangular so that the eigenvalues are immediately given by

$$a, \qquad -b - \delta + \nu, \qquad \frac{b\nu}{b+\delta}.$$

It follows that the first eigenvalue is always positive. The equilibrium is thus unstable.

Let T_5 denote the smallest of the two roots of (6.2). It is real and positive if we impose

$$[be + \delta d + (e-d)(\nu-\delta)]^2 \geq 4d\delta(e-d)(\nu-\delta)$$

and we require

$$d > e \quad \text{and} \quad be > d(\nu - \delta). \tag{6.3}$$

In such a situation indeed the coefficient of W in (6.2) is negative, so that both roots W_i, $i = 5, 6$, are positive. If (6.3) is not satisfied, however, then only T_6 may still be feasible. This happens if

$$e > d \quad \text{and} \quad d(\nu - \delta) > be. \tag{6.4}$$

To ensure feasibility of T_i, $i = 5, 6$, we need also to ensure that $W_i \leq 1$. It is easy to establish that this condition is equivalent to imposing that the sum of the coefficients of (6.2) be nonnegative. In turn, this yields

$$\nu e > 0,$$

which is obviously true. The remaining conditions $R_i \geq 0$, $M_i \geq 0$, $i = 5, 6$, are also immediately seen to hold without any restriction on the parameters of the model.

The alternative geometric interpretation gives that $M \geq 0$ is satisfied from the first (6.1), solving for M in terms of W. From the second and third equations we obtain two other hyperbolae which must be intersected. The latter gives positive values for W in $[0, 1]$ only in the case $e < d$ and $b + \delta - \nu > 0$. It will be positive only for $0 \leq W \leq \alpha$, with $\alpha = 1 - \nu/(b + \delta)$. In the same case the former is positive for any W in $[0, 1]$. To ensure an intersection, it is sufficient to require $(b + \delta - \nu)/(d - e) > b/d$.

Looking now at the stability analysis, upon linearization we find the following matrix for $\underline{x} = (r, w, m)$

$$\begin{aligned}
&A_{12} = (c - \eta) M_i R_i && A_{13} = -[c(1 - W_i) + \eta W_i] R_i \\
&A_{21} = (e - d)(1 - W_i) W_i && A_{22} = -W_i [b + \delta + R_i (e - d)] \\
&A_{31} = [d(1 - W_i) + e W_i] M_i && A_{32} = M_i [(e - d) R_i + b] \\
&A_{ij} = 0 \text{ otherwise.}
\end{aligned}$$

From these, the characteristic polynomial can easily be obtained. The Routh-Hurwitz criterion then gives

$$\begin{gathered}
-A_{22} > 0, \\
\overline{\Delta} = -A_{22}[A_{13}A_{31} + A_{12}A_{21}] - A_{13}[A_{22}A_{31} - A_{21}A_{32}] > 0, \\
A_{13}[A_{22}A_{31} - A_{21}A_{32} > 0.
\end{gathered} \tag{6.5}$$

Notice that

$$A_{13} < 0, \qquad A_{31} > 0. \tag{6.6}$$

Now suppose that $e \geq d$, so that only T_6 is feasible. Then we also have

$$A_{22} \leq 0, \qquad A_{21} \geq 0, \qquad A_{32} \geq 0 \tag{6.7}$$

so that the first of (6.5) is satisfied. The second condition is then reduced to

$$A_{12} > -A_{13}A_{32}/A_{22}, \tag{6.8}$$

which in turn becomes, in terms of the original parameters of the model

$$(d-e)(c-\eta)W_6^2 - \delta d(c-\eta)W_6 - ecb > 0. \tag{6.9}$$

In this case it turns out that the third condition of the Routh-Hurwitz criterion is automatically satisfied.

Now, for the case $e < d$, we obtain two feasible points T_5 and T_6; from the first of (6.5),

$$R_i > \frac{b+\delta}{d-e}, \qquad i = 5, 6. \tag{6.10}$$

Since now, in place of (6.7), we have

$$A_{21} < 0, \qquad A_{32} < 0, \qquad A_{13} < 0, \qquad A_{31} > 0, \tag{6.11}$$

the second condition gives again inequality (6.8). In this case it can be written as (6.9); we can rewrite it also as follows

$$W_i > \frac{c(b+(e-d)R_i)}{\delta(\eta-c)}. \tag{6.12}$$

Then for $c > \eta$ it is obviously satisfied since the right hand side is negative. Conversely, if $c < \eta$, (6.12) becomes a necessary condition for stability.

In this case also, it is easily checked that the third condition is automatically verified, on using the relations (6.11).

In conclusion the above conditions give the cases when the equilibria T_5 and T_6 are feasible and stable. A suitable choice of the parameters of the model will satisfy all of them, since there are less conditions than parameters in the model.

7. Discussion. Eight different models for the study of the spreading of diseases among predator-prey systems have been proposed and analyzed for the two cases when a positive and a negative value on the predation of infected individuals is assumed. In all of them, trivial equilibria corresponding to the neutral equilibrium point of the Lotka-Volterra system arise, and show either instability or neutral type stability, in the sense that the disease dies out and the trajectories approach the neutrally stable limit cycles in the RF plane.

A second type of trivial equilibria is given by the line $R = 0$, $F = 0$ and the number of infectives being arbitrary. This is usually a stable equilibrium corresponding to extinction of both species and to the fact that every individual in one of them gets infected.

Apart from these there are other equilibria which are nontrivial. It turns out, however, that in the models where the incidence is represented by a mass action term, either they are infeasible or unstable, at least in the most interesting case of negative value on predation ($h > 0$). When we use standard incidence, if we consider the SI epidemic model, the nontrivial equilibrium is again either infeasible or unstable. For the SIS case we obtain, however, nontrivial stable equilibria. These are the most interesting cases because they show a radically different behavior of these models from the previous ones and from the original Lotka-Volterra. The long term behavior of the population sizes adjusts so that the predators decrease their size, with respect to the equilibrium of the Lotka-Volterra system, while the prey increase. Thus, if the disease spreads among the prey, it can act as a control on the predator population size. If, instead, the disease is supposed to affect the predators, the equilibria T_5 and T_6 correspond to population sizes for both species, which are at a lower level than the corresponding ones given by the Lotka-Volterra model. This is easily seen from their definition.

In conclusion, this investigation seems to suggest that if we want to control the predator population size, not harming the prey too much, we could introduce a pest on them, which can affect the predators

as well. If the hypothesis for assuming the standard incidence in the spread of the disease are satisfied, then the long term behavior of the system will be as desired, provided that the initial conditions are close enough to the equilibrium.

Acknowledgments. The author wishes to thank the referee for his many suggestions leading to significant improvements in the paper.

REFERENCES

1. S. Busenberg, P. van den Driessche, *Analysis of a disease transmission model in a population with varying size*, J. Math. Biol. **28** (1990), 257–270.

2. L. Cesari, *Asymptotic behavior and stability problems in ordinary differential equations*, Academic Press, New York, 1962.

3. U. D'Ancona, *The struggle for existence*, Brill, Leiden, 1954.

4. L.Q. Gao and H.W. Hethcote, *Disease transmission models with density-dependent demographics*, J. Math. Biol. **30** (1991), 717–731.

5. K.P. Hadeler and H.I. Freedman, *Predator-prey populations with parasitic infection*, J. Math. Biol. **27** (1989), 609–631.

6. H.W. Hethcote, *Three basic epidemiological models*, Biomathematics, vol. 18 (Levin, Hallam and Gross, eds.), Appl. Math. Ecol. (1989), 119–144.

7. H.W. Hethcote, H.W. Stech and P. van den Driessche, *Periodicity and stability in epidemic models*; *A survey*, Differential equations and applications in ecology, epidemics and population problems (1981), 65–82.

8. H.W. Hethcote and S.A. Levin, *Periodicity in epidemiological models*, Biomathematics, vol. 18 (Levin, Hallam and Gross, eds.), Appl. Math. Ecol. (1989), 193–211.

9. A.J. Lotka, *Elements of mathematical biology*, Dover, New York, 1956.

10. A.C. McKendrick, *Applications of mathematics to the medical problems*, Proc. Edinburgh Math. Soc. **44** (1926), 98–130.

11. J. Mena-Lorca and H.W. Hethcote, *Dynamic models of infectious diseases as regulators of population sizes*, J. Math. Biol. **30** (1991), 693–716.

12. P.F. Verhulst, *Recherches mathématiques sur la loi d'accroissement de la population*, Mém. Acad. Roy. Bruxelles **18** (1845), 1.

13. ———, *Recherches mathématiques sur la loi d'accroissement de la population*, Mém. Acad. Roy. Bruxelle **20** (1847), 1.

14. V. Volterra and U. D'Ancona, *La concorrenza vitale tra le specie dell'ambiente marino*, VIIe. Congr. Int. acquicult. et de pêche, Paris (1931), 1–14.

15. H. Von Foerster, *Some remarks on changing populations*, in *The kinetics of cellular proliferation*, Grune & Stratton, New York (1954), 382–407.

Department of Mathematics, University of Iowa, Iowa City, IA 52242