A Predator-Prey Model with Disease Dynamics[*†]

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Abstract

We propose a model to describe the interaction between a diseased fish population and their predators. Analysis of the system is performed to determine the stability of equilibrium points for a large range of parameter values. The existence and uniqueness of solutions is established and solutions are shown to be uniformly bounded for all nonnegative initial conditions. The model predicts that a deadly disease and a predator population cannot co-exist. Numerical simulations illustrate a variety of dynamical behaviors that can be obtained by varying the problem data.

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1 Introduction

We consider a model proposed by Chattopadhyay and Bairagi[4]: a system of three nonlinear ordinary differential equations describing disease and predation among the Tilapia fish of the Salton Sea and their predator, the pelican. This model is of interest because it combines a two compartment epidemic model with a standard predator-prey model.

Since mid-August of 1996, a bacterial outbreak of *Vibrio vulnificus* in the Salton Sea among the Tilapia has led to massive deaths not only among the fish themselves, but also in the pelican population. Studies have indicated that the bacterial infection contributes to low oxygen levels in the tissues of the infected fish. The shortage of oxygen causes the fish to seek oxygen from the sea's surface and leads to a favorable environment for botulism to grow in the tissues of the infected fish[4]. When pelicans prey upon these vulnerable fish, it is likely that they ingest the botulism toxins that eventually contribute to the development of *Avian botulism*. *Avian botulism* is a debilitating neurological disease which usually inflicts death upon its host[8].

Environmental stressors have been targeted as the probable cause of this situation. Extremely high temperature, massive algae growth, high salinity, pollution, and low dissolved oxygen levels are all conditions which can lead to epidemics[8]. In this paper we neglect the initial cause of the outbreak and focus on the dynamics and interactions of the sick and susceptible fish with the pelicans.

The model of Chattopadhyay and Bairagi is based on these assumptions:

(1) The prey are divided into two disjoint classes, susceptible fish, S, and infected fish, I;

(2) In the absence of predation and infection, the fish population grows logistically;

(3) Infected fish are unable to reproduce due to short lifespan and infected fish do not recover;

(4) Infection impairs fish defenses, therefore pelicans eat only infected fish; a Holling type II predation rate is used for infected fish;

(5) Pelicans die at a constant relative rate due to infected fish consumption and therefore die at a constant relative rate overall.

One limitation of their model is that eating infected fish must be more beneficial than harmful. This paper deals with the case where eating infected fish does more harm than good.

In our paper we alter assumptions (4) and (5) to generalize and thus more accurately describe natural dynamics. It seems more likely that the pelicans will consume whatever is available, be it sick or susceptible fish. The infected fish are more likely to be preyed upon by pelicans because the infection has been found to impair fish defense mechanisms as well as forcing the fish to seek oxygen on the sea's surface. Since botulism is an accumulative sickness it is reasonable to presume that the pelicans' death rate is not constant, but rather proportional to the number of sick fish ingested[8]. We develop these assumptions into a model and then find and classify its equilibrium points. Numerical experiments are then carried out to confirm and visualize the equilibrium analysis.

2 The Model and Equations

In the development of our model, we first considered the typical SI model in which two populations of fish exist: susceptible fish, S, and infected fish, I. A typical SI model with an open system of variable size is

$$\dot{S} = \epsilon - \lambda SI - \rho S,$$
$$\dot{I} = \lambda SI - \mu I.$$

where ϵ represents the birth rate, λ the infection rate multiplied by transmission probability, ρ the natural death rate for the susceptible fish, and μ the death rate due to infection. This model assumes that the disease is transmitted by contact between an infected fish and a susceptible fish, and that all susceptible fish are equally susceptible and all infected fish are equally infectious.

2.1 Predation Term

We then attempted to incorporate the standard predator-prey model with Holling Type II predation into our model. A common predator-prey model with Holling Type II predation can be expressed as

$$\dot{N} = rN\left(1 - \frac{N}{K}\right) - \frac{mNP}{\gamma + N},$$
$$\dot{P} = \frac{CmNP}{\gamma + N} - dP.$$

where N denotes the fish population and P the pelicans. In the absence of predation, the fish population, N, grows logistically with carrying capacity K. In this system, m denotes the search rate, C the conversion factor, and γ the half saturation coefficient. These equations only successfully model a system in which a single predator feeds upon a single prey. Considering our new assumptions, namely the assumption that the pelicans eat both infected and susceptible fish, the Holling Type II predation term does not suffice. This caused us to develop a new predation term which accounted for the pelicans' consumption of both infected and susceptible fish.

In the development of our new predation term, we refer to work by C.S. Holling[5] in which he described the dynamics of the consumption of prey by predators as the density of the prey changes. We expand his model to account for a system in which a single predator feeds upon two types of prey, specifically infected and susceptible fish.

In his 1959 paper, Holling devised an experiment in which blindfolded human subjects represented the predator and uniformly sized sandpaper disks corresponded to the prey. The human subject was placed in front of a 3 by 3 foot table to which the sandpaper disks were attached. The experimental process required the "predator" to search for the "prey" by tapping on the table. When a disk was located the "predator" picked it up and placed it in a pile; this represented handling time. The procedure was repeated for a given period of time, and the results were recorded. Holling described these results with the following equation:

$$y = aT_s x,$$

where y is the total number of collected disks, a is the rate of searching multiplied by the probability of finding a disk, T_s is the available search time, and x is the density of the disks. The search time, T_S, is found from taking the total time and subtracting the total handling time, where b is the handling time per fish. This is expressed as

$$T_s = T_t - by.$$

Substitution yields the standard Holling Type II predation formula

$$y = \frac{T_t a x}{1 + abx}.$$

To find a predation term that reflects our assumption that the pelicans consume both infected and susceptible fish, we follow the steps in Holling's experiment. We require that the sandpaper disks consist of two distinct sizes. Studies have indicated that the infected fish are more vulnerable to predation due to their illness[7]; therefore, we let the larger disks correspond to the infected fish, and the smaller disks to the susceptible fish. Recall that in Holling's experiment, the parameter a is the rate of searching times the probability of finding a disk. We consider that the probability of finding a particular disk depends on the area of the disk. This is a reasonable assumption since the chances of finding any particular disk on a table corresponds directly to the size of the disk. The predation rates for susceptible and infected fish are then

$$y_S = a_S T_s S, \qquad y_I = a_I T_s I, \tag{1}$$

where a_S and a_I are the area of each respective disk. To take into account the vulnerability of the infected fish, we make the infected disks larger than the susceptible by a vulnerability factor of v, where $v > 1$. Setting $a = a_S$ we can simplify $a_I = v a_S = va$, which simplifies eqs. 1 to

$$y_S = aT_s S, \qquad y_I = vaT_s I. \tag{2}$$

The susceptible and infected fish are of the same species, so it is reasonable to consider that they require the same amount of handling time, b. Considering T_t to be unit time yields the following expression for search time

$$T_s = 1 - b(y_S + y_I). \tag{3}$$

Substituting eq. 3 into eqs. 2, we obtain the ystem

$$\begin{pmatrix} 1 + abS & ab \\ vab & 1 + vabI \end{pmatrix} \begin{pmatrix} y_S \\ y_I \end{pmatrix} = \begin{pmatrix} aS \\ vaI \end{pmatrix},$$

with solutions

$$y_S = \frac{aS}{1+ab(S+vI)}, \qquad y_I = \frac{vaI}{1+ab(S+vI)}.$$

To aide in scaling, we change the parameters to $\ell = 1/b$ and $\gamma = 1/ab$. This yields the new solutions

$$y_S = \frac{\ell S}{\gamma+S+vI}, \qquad y_I = \frac{v\ell I}{\gamma+S+vI},$$

Our new parameters ℓ and γ now correspond respectively to the amount of handled fish in unit time and the number of searches per unit time.

2.2 Pelican Death Rate

We assume that pelicans die at a rate proportional to the amount of infected fish consumed. This requires the addition of a death rate which incorporates both our new predation term and the natural relative death rate, which can be considered constant. Accordingly, we define the relative death rate of pelicans to be

$$\left(\frac{1}{P}\right)\frac{dP}{dt} = -\left(\psi y_I + d\right).$$

Here d is the natural death rate and ψ is the pelican death rate due to infection per ingested fish.

2.3 Model Formulation

Putting the pieces of our model together we obtain the following differential equations

$$\begin{aligned}
\dot{S} &= rS\left(1-\frac{S+I}{K}\right) - \frac{\ell SP}{\gamma+S+vI} - \lambda SI, \\
\dot{I} &= \lambda SI - \frac{v\ell IP}{\gamma+S+vI} - \mu I, \\
\dot{P} &= \frac{\theta\ell(S+vI)P}{\gamma+S+vI} - dP - \frac{\psi v\ell IP}{\gamma+S+vI}.
\end{aligned}$$

The birth/death rates in the SI model are built into the parameters r and K in $\dot{S}$. Thus, r denotes the maximum growth rate of fish, θ the conversion factor for the pelicans, and all other parameters have been previously defined.

Before analyzing our system, it is convenient to scale our equations. By writing a model in dimensionless form, we are able to reduce the number of parameters. We choose our scale for S and I to be K because we want our maximum population to be 1. We pick our scale for P to be $r\gamma/\ell$, and our timescale to be r. Implementing these scales into our model we are left with the following system in Kolmogorov form[2]:

$$\begin{aligned} \dot{s} &= sf(s,i,p), \\ \dot{i} &= ig(s,i,p), \\ \dot{p} &= ph(s,i,p), \end{aligned} \tag{4}$$

with

$$\begin{aligned} f(s,i,p) &= 1 - s - (1+\beta)i - \frac{\Gamma p}{\Gamma + s + vi}, \\ g(s,i,p) &= \beta(s-\alpha) - \frac{v\Gamma p}{\Gamma + s + vi}, \\ h(s,i,p) &= c\left(\frac{s + v(1-\eta)i}{\Gamma + s + vi} - e\right), \end{aligned}$$

where

$$\beta = \frac{\lambda K}{r}, \quad c = \frac{\theta \ell}{r}, \quad \eta = \frac{\psi}{\theta}, \quad e = \frac{d}{\theta}, \quad \alpha = \frac{\mu}{\lambda K}, \quad \Gamma = \frac{\gamma}{K}.$$

The parameters now have the following physical interpretations:

- β: Rate constant for disease transmission;
- Γ: Half-saturation point of our predation term;
- v: Vulnerability of infected fish relative to susceptible fish;
- α: Non-predation death rate of infected fish relative to the infection rate;
- c: Ratio of population grouth rate of pelicans to that of fish;
- η: Harm for pelicans eating infected fish;
- e: Per capita pelican death rate relative to the growth rate.

3 The Analysis and Mathematical Results

Here we deal with the existence and stability of equilibrium points. Throughout the rest of the paper we assume that $\eta > 1$. This assumption is reasonable considering that eating infected fish is harmful to pelicans. The existence and uniqueness of solutions is established and the solutions are shown to be uniformly bounded.

3.1 Equilibrium Analysis

Setting all derivatives of system 4 equal to zero we find the following five equilibrium points, named by the nontrivial variables:

$$\begin{aligned} \mathbf{T} &\equiv (0,0,0) \\ \mathbf{S} &\equiv (1,0,0) \\ \mathbf{SI} &\equiv \left(\alpha, \tfrac{1-\alpha}{1+\beta}, 0\right) \\ \mathbf{SP} &\equiv \left(\tfrac{\Gamma e}{1-e}, 0, \tfrac{1-e(\Gamma+1)}{(1-e)^2}\right) \end{aligned}$$

The last equilibrium point **SIP** can be found by solving the following system of 3 equations:

$$\begin{pmatrix} \beta+v & v(1+\beta) \\ (1-e) & v(1-\eta-e) \end{pmatrix} \begin{pmatrix} s \\ i \end{pmatrix} = \begin{pmatrix} v+\beta\alpha \\ \Gamma e \end{pmatrix}, \tag{5}$$

$$p = \frac{\beta(\Gamma + s + vi)(s-\alpha)}{v\Gamma}.$$

We use the convention $\mathbb{R}_{0,+} \equiv (\mathbb{R} \cup \{0\})$. Since we are working in $\mathbb{R}^3_{0,+}$ we need to check which parameter values allow the equilibria to exist. It is easy to see that **T** and **S** exist for all parameter values. **SI** exists when $\alpha < 1$. Similarly, **SP** exists when $e < \frac{1}{\Gamma+1}$. Note here that the disease can't persist if the death rate of infected fish is too high. Similarly, the predator can't survive if it's death rate is too high.

3.1.1 Existence of SIP

The interior equilibrium point, $\mathbf{SIP} = (\mathbf{S}, \mathbf{I}, \mathbf{P})$, exists when $\mathbf{S}$, $\mathbf{I}$, $\mathbf{P} \in \mathbb{R}_+$. In order to have $\mathbf{P} > 0$, we must have

$$1 - \mathbf{S} - (1+\beta)\mathbf{I} > 0 \text{ and } \beta(\mathbf{S} - \alpha) > 0$$

Let $D = \{(s,i) \in \mathbb{R}^2_+ \mid s > \alpha \text{ and } s + (1+\beta)i < 1\}$. In order for **SIP** to exist, we must ensure that $(\mathbf{S}, \mathbf{I}) \in D$. The linear system 5 yields unique solutions for $(\mathbf{S}, \mathbf{I})$ when $\eta > 1$. The first part of system 5 gives the equation $(\beta+v)\mathbf{S} + v(1+\beta)\mathbf{I} = v + \beta\alpha$. Since D is convex, and the graph of the last equation enters D at a vertex, then showing $(\mathbf{S}, \mathbf{I}) \in D$ is equivalent to showing that $0 < \mathbf{I} < \frac{1-\alpha}{1+\beta}$. Thus **SIP** exists whenever:

$$E_1 \equiv \frac{\alpha(1+\beta) + v(1-\eta)(1-\alpha)}{(\Gamma+\alpha)(1+\beta) + v(1-\alpha)} < e < \frac{v+\beta\alpha}{(v+\beta\alpha) + \Gamma(v+\beta)} \equiv E_2 \tag{6}$$

$$\alpha < 1 \text{ and } e < \frac{1}{\Gamma+1}$$

The parameters α and e are recognized to be important from the criterion for the existence of equilibria. The existence regions for the equilibria are depicted schematically in Figure 1.

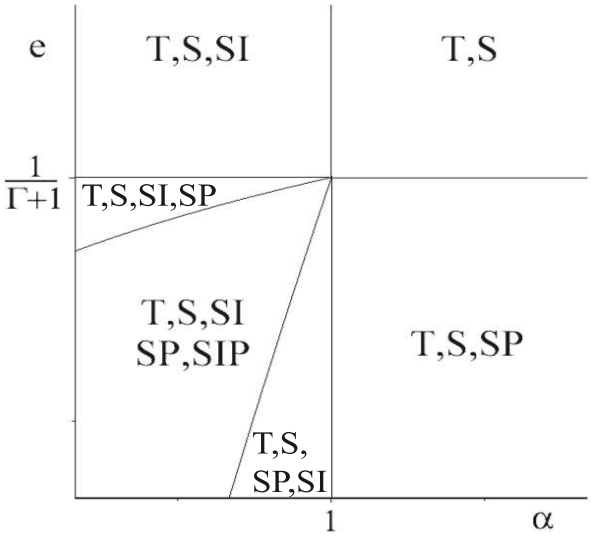

Figure 1: Regions of equilibria existence in (α, e) space

3.2 Stability and Boundedness

Next we find where these equilibria are stable. Standard linearization techniques confirm that **T** is a saddle for all parameter values and that **S** is locally stable whenever $\alpha > 1$ and $e > \frac{1}{\Gamma+1}$.

Claim 1 *The equilibrium **SI** is locally stable whenever $e > E_1$ and $\alpha < 1$.*

The Jacobian for the system at **SI** is

$$J(\mathbf{SI}) = \begin{pmatrix} -\alpha & -\alpha(1+\beta) & sf_p \\ \frac{(1-\alpha)\beta}{1+\beta} & 0 & ig_p \\ 0 & 0 & \frac{(1+\beta)(\alpha-e\Gamma-e\alpha)+v(1-\alpha)(1-\eta-e)}{(1+\beta)(\Gamma+\alpha)+v(1-\alpha)} \end{pmatrix}.$$

The eigenvalues of this matrix are $\lambda_1 = \frac{(1+\beta)(e\Gamma+e\alpha-\alpha)+v(1-\alpha)(\eta+e-1)}{(1+\beta)(\Gamma+\alpha)+v(1-\alpha)}$, and $\lambda_{2,3} = \frac{-\alpha \pm \sqrt{\alpha^2-4(\alpha-\alpha^2)\beta}}{2}$.

$$e > E_1 \iff \lambda_1 < 0.$$

$\Re(\lambda_{2,3}) < 0$ as long as $\alpha^2 - 4(\alpha - \alpha^2)\beta < \alpha^2$ holds; this is clearly true for $\alpha < 1$. Thus we have that **SI** is locally stable under the given conditions.

Claim 2 *The equilibrium point **SP** is locally stable whenever* $\frac{1-\Gamma}{\Gamma+1} < e < \min\{\frac{1}{\Gamma+1}, E_2\}$.

The Jacobian for the system at **SP** is

$$J(\mathbf{SP}) = \begin{pmatrix} \frac{e(\Gamma+\Gamma e+e-1)}{e-1} & sf_i & -\Gamma e \\ 0 & \frac{v(1-\Gamma e-e)+\beta(\alpha-\alpha e-\Gamma e)}{e-1} & 0 \\ \frac{c(1-\Gamma e-e)}{\Gamma} & cph_i & 0 \end{pmatrix}.$$

An eigenvalue of this matrix is $\lambda_1 = \frac{v(1-\Gamma e-e)+\beta(\alpha-\alpha e-\Gamma e)}{e-1}$. The other two eigenvalues are found from the matrix

$$J_{2,2} = \begin{pmatrix} \frac{e(\Gamma+\Gamma e+e-1)}{e-1} & -\Gamma e \\ \frac{c(1-\Gamma e-e)}{\Gamma} & 0 \end{pmatrix}$$

$$e < E_2 \iff \lambda_1 < 0.$$

The Routh-Hurwitz Criterion[2] for a 2×2 matrix states that the eigenvalues will have negative real parts whenever $det(J_{2,2}) > 0$ and $trace(J_{2,2}) < 0$.

$$e < \frac{1}{1+\Gamma} \iff \det(J_{2,2}) > 0,$$

$$e > \frac{1-\Gamma}{1+\Gamma} \iff trace(J_{2,2}) < 0.$$

Since all eigenvalues of $J(\mathbf{SP})$ have negative real part, we have that **SP** is locally stable with the given conditions.

Claim 3 *The interior equilibrium point **SIP** is unstable everywhere that it exists.*

$$J(\mathbf{SIP}) = \begin{pmatrix} \mathbf{S}f_s & \mathbf{S}f_i & \mathbf{S}f_p \\ \mathbf{I}g_s & \mathbf{I}g_i & \mathbf{I}g_p \\ c\mathbf{P}h_p & c\mathbf{P}h_i & 0 \end{pmatrix}$$

Given that $g_p = vf_p$, we have

$$\det(J(\mathbf{SIP})) = (-c\mathbf{SIP}f_p)(vf_sh_i + g_ih_s - vf_ih_s - g_sh_i)$$

After quite a bit of computation, it can be shown that

$$vf_sh_i + g_ih_s - vf_ih_s - g_sh_i = \frac{\eta(1+\beta)}{\Gamma+s+vi} > 0$$

and

$$f_p = \frac{-\Gamma}{\Gamma + s + vi} < 0.$$

Thus $\det(J(\mathbf{SIP})) > 0$. The Routh-Hurwitz criterion for 3×3 matrices says there will be at least one eigenvalue of $J(\mathbf{SIP})$ that will have a non-negative real part. This allows us to conclude that **SIP** is an unstable equilibrium point in the region where it exists.

Figure 2 shows where, in the (α, e) plane, the different equilibria are stable.

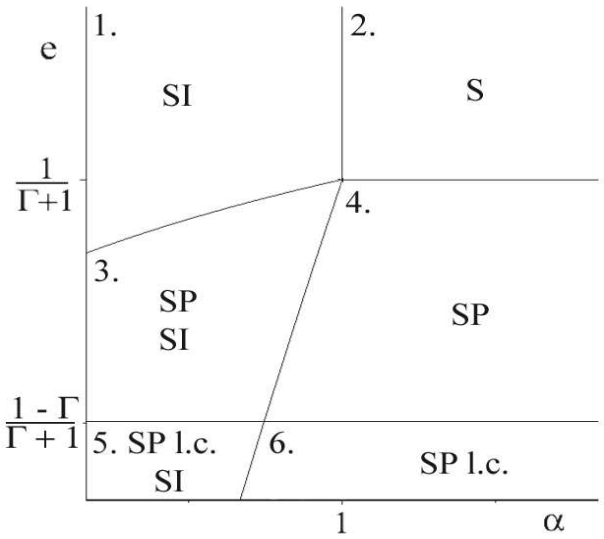

Figure 2: Regions of equilibria stability in (α, e) space. l.c. refers to a limit cycle around the corresponding equilibrium point.

3.2.1 Existence, Uniqueness and Boundedness

Equations 4 can be written in the form

$$\dot{y} \;=\; F(y) \tag{7}$$

where $y = (s, i, p)$, $y_o = (s(0), i(0), p(0))$ and $F(y) = (f(s, i, p), g(s, i, p), h(s, i, p))$.

Lemma 1 *The solution $y(t)$ exists and is unique on an interval $(0, t_f)$ when $y_o \in \mathbb{R}^3_{0,+}$.*

Proof: By assumption, $y_o \in \mathbb{R}^3_{0,+}$. The Jacobian of F is

$$J(F(s,i,p)) = \begin{pmatrix} f + sf_s & sf_i & sf_p \\ ig_s & g + ig_i & ig_i \\ cph_s & cph_i & ch + cph_p \end{pmatrix},$$

and both it and F are continuous because $\Gamma > 0$. Thus, F satisfies a Lipschitz condition on $\mathbb{R}^3_{0,+}$. By the Picard-Lindelof Theorem [10], the solution exists and is unique for some time interval extending forward from the initial point.

Theorem 1 *The solution $y(t)$ is uniformly bounded for $y_o \in \mathbb{R}^3_{0,+}$.*

Proof: Define $k : \mathbb{R}_{0,+} \to \mathbb{R}_{0,+}$ by $k(t) = s(t) + i(t) + \frac{\Gamma}{c}p(t)$. k is well defined and differentiable by **Lemma 1** on some maximal interval $(0, t_f)$. Differentiating k yields

$$\dot{k} = s(1-s) - i(\alpha\beta + s) - \Gamma p\left(\frac{v\eta i}{\Gamma + s + vi} + e\right).$$

For any $\epsilon > 0$,

$$\dot{k} + \epsilon k = s(1 - s + \epsilon) - i(\alpha\beta + s - \epsilon) - \Gamma p\left(\frac{v\eta i}{\Gamma + s + vi} + e - \frac{\epsilon}{c}\right).$$

The maximum value of $s(1 - s + \epsilon)$ on $\mathbb{R}_{0,+}$ is $\frac{(1+\epsilon)^2}{4}$. Therefore

$$\dot{k} + \epsilon k \leq \frac{(1+\epsilon)^2}{4} - i(\alpha\beta - \epsilon) - \Gamma p\left(e - \frac{\epsilon}{c}\right).$$

Choose $0 < \epsilon < \min\{\alpha\beta, ce\}$. Then $\exists\, B > 0$ such that

$$\dot{k} + \epsilon k \leq B \text{ for each } t \in (0, t_f).$$

Let $G(t, y) = B - \epsilon y$, which satisfies a Lipschitz condition everywhere. Clearly $\dot{k}(t) \leq B - \epsilon k(t) = G(t, k(t))$ for all $t \in (0, t_f)$. Let $\dot{r} = G(t, r) = B - \epsilon r$ and $r(0) = k(0) = k_o$. This ordinary differential equation has the solution

$$r(t) = \frac{B}{\epsilon}(1 - e^{-\epsilon t}) + k_o e^{-\epsilon t}.$$

It is clear that $r(t)$ is bounded on $(0, t_f)$. By the comparison theorem[1]:

$$k(t) \leq r(t) = \frac{B}{\epsilon}(1 - e^{-\epsilon t}) + k_o e^{-\epsilon t} \quad \forall t \in (0, t_f).$$

Now suppose $t_f < \infty$. Then $k(t_f) \leq r(t_f) < \infty$, but then the solution exists uniquely for some interval (t_f, t_r) by the Picard-Lindelof Theorem. This contradicts the supposition that $t_f < \infty$. Therefore $k(t)$ must be bounded for all non-negative t, and thus $y(t)$ is uniformly bounded on $\mathbb{R}_{0,+}$.

4 Numerical Results

In this section we use numerical experiments to confirm and visualize the equilibrium analysis. We go through the regions where there are interesting dynamics, and shed new light on their physical meaning. We used a fourth order Runga-Kutta implementation on MATLAB to numerically solve our equations.

4.1 Dynamics of Region 3

In the arc between the two curves E_1 and E_2, both **SI** and **SP** are feasible equilibrium points for various initial conditions. To illustrate the effect that initial conditions have on the convergence toward equilibrium points, we choose a value of α and e which lies within region 3. By fixing all other parameters and choosing two distinct initial conditions on the IP-plane we developed the following systems of graphs.

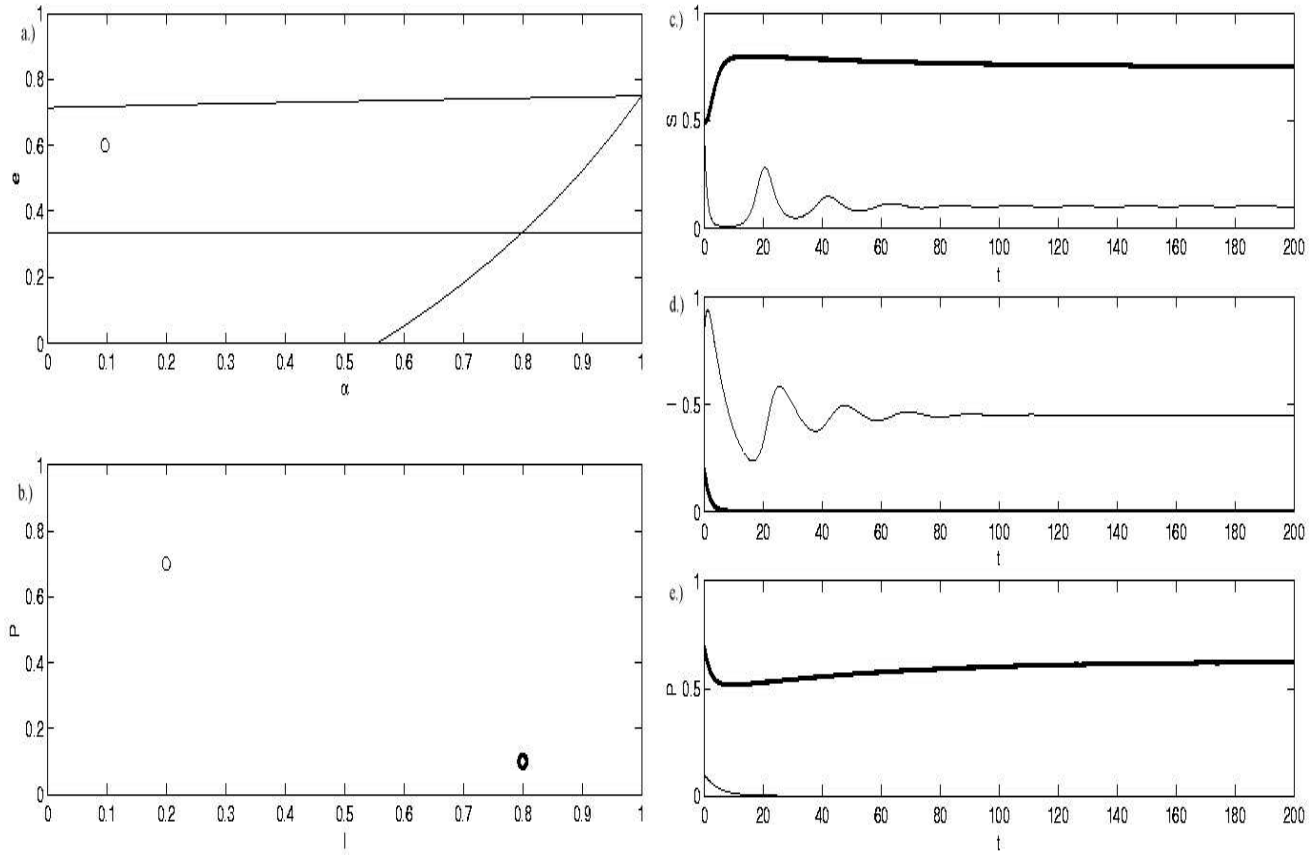

Figure 3: Graphs describing the dynamics of region 3. The (α, e) plane (a), initial conditions (b), and corresponding solutions for $c = 0.2$, $e = 0.6$, $v = 5$, $\Gamma = 0.5$, $\alpha = .1$, $\beta = 1$, $\eta = 1.5$, and initial conditions (0.5, 0.2, 0.7) in normal and (0.5, 0.8, 0.1) in bold.

Since each initial condition converges to separate equilibria, we conclude that the position of the initial conditions determines the equilibrium point to which they converge. We can also infer from our graphs that there must exist a surface in SIP space that seperates the domains of attraction of the **SI** and **SP** equilibrium points. The unstable equilibrium point, **SIP**, is in that surface.

4.2 Dynamics in Region 5

When the parameter e falls below $\frac{1-\Gamma}{\Gamma+1}$ the dynamics of the system change slightly. In order

to illustrate how the system behaves, we chose a point (α, e) which lies in region 5. Maintaining all other parameter values and initial conditions, we obtain the following results.

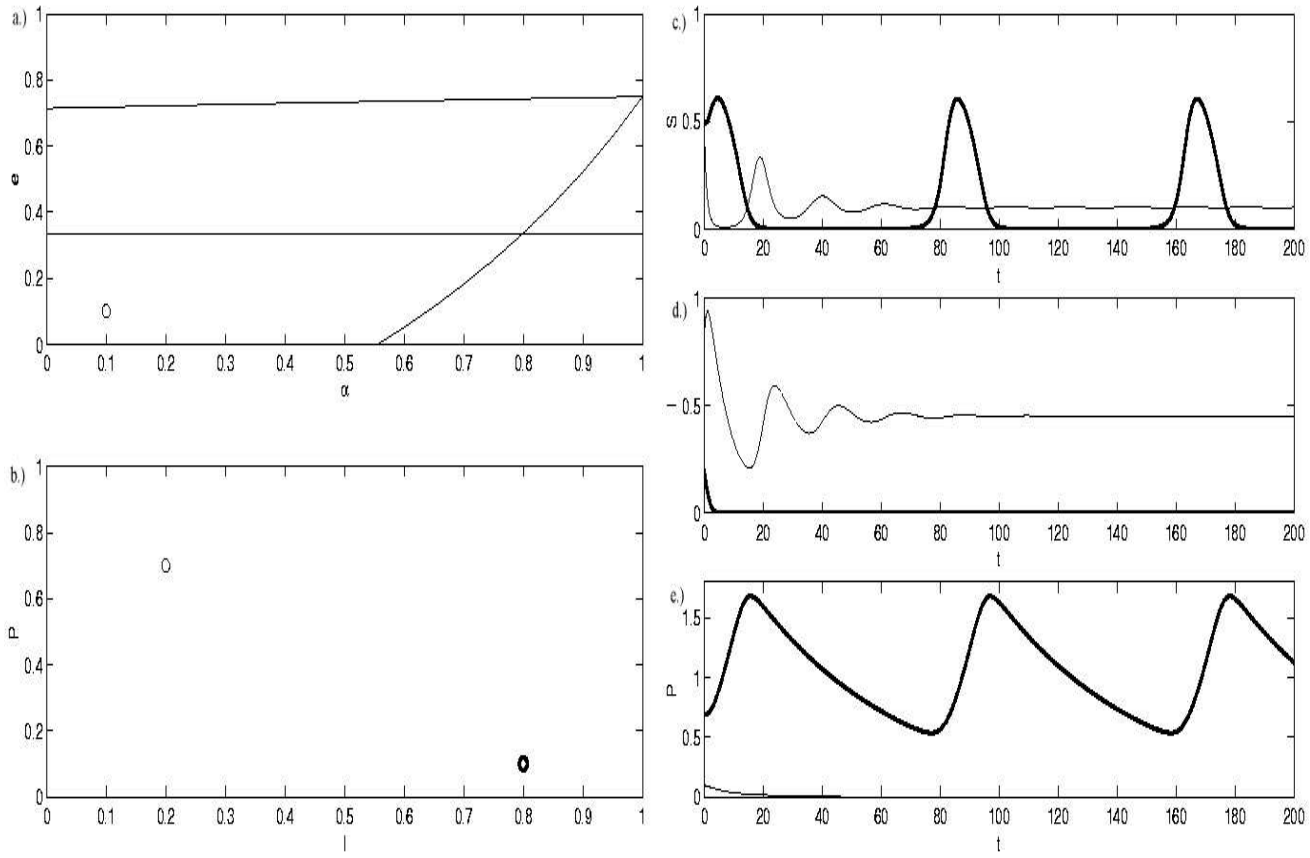

Figure 4: Graphs describing the dynamics in region 5. Choosing $e = 0.1$ and $\alpha = 0.1$ to correspond to a point in region 5. Initial conditions and other parameter values remain unchanged.

Both initial conditions move towards separate equilibrium points similar to the preceding case. However, in this situation one initial condition converges to **SI** whereas the other approaches a limit cycle around **SP**. This still implies the existence of a surface seperating the domains of attraction for the stable behaviors.

4.3 Dynamics in Region 6

Varying the point (α, e) to lie below E_1 and the line $\frac{1-\Gamma}{\Gamma+1}$, we can observe the dynamics within region 6.

In this scenario both initial conditions lead to limit cycles around SP. Our results show that if the infection is very damaging to the sick fish, then they will soon die out and eventually the susceptible fish and pelicans prevail. This is the same behavior that occurs in the equivalent predator-prey system without disease[2].

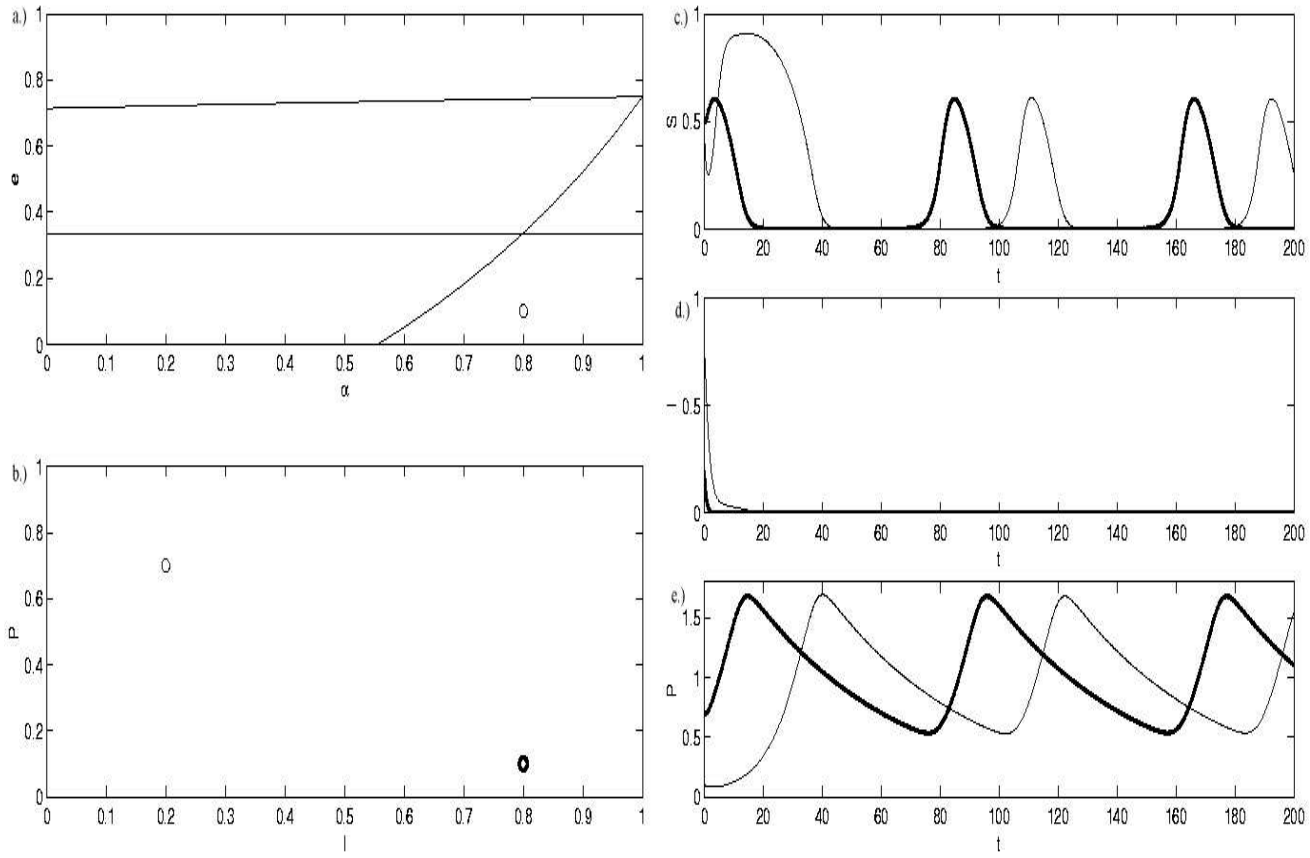

Figure 5: Graphs describing the dynamics in region 6. All parameter and initial condition values are the same as the previous cases. Only the point (α, e) has been modified to be $(0.8, 0.1)$.

4.4 Effect of β

Prior to the first major bird kill, environmental conditions of the sea apparently allowed for the co-existence of pelicans and uninfected fish. This corresponds to a region in which the **SP** equilibrium point is stable. If the environmental state were to change such that the infection rate, β increases, it is possible that the conditions could change so that **SP** is no longer stable. Pollution is one factor which has been shown to increase the infection rate for a variety of fish diseases[6].

To demonstrate the effect of β, let us pick a point, say A, in region 3 and assume that initially **SP** is the stable equilibrium. Let us also assume Γ and e are known and that all other parameters are fixed. As pollution is introduced into the sea, the infection rate increases (this corresponds to changes in β along the vertical axis of the graph), causing the curve E_2 to shift downward thus decreasing the area of region 3. As this occurs, point A is no longer inside region 3 and now exists in region 1, where only **SI** equilibrium is stable, meaning the pelicans will certainly die out. This is consistent with what one would expect to occur in nature. It has been suggested that reducing the number of infected fish may be a means of maintaining

the pelican population[4]. However, according to our results, a change in the initial conditions alone will not suffice because point A will converge to **SI** regardless, as it lies in a region of the parameter space where only **SI** is stable. One would have to alter certain parameter values, such as β, e, or α to return point A to region 3 and possibly adjust the initial conditions such that convergence to **SP** is guaranteed.

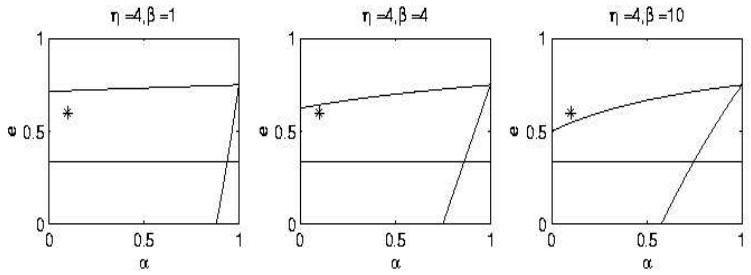

Figure 6: Graphs describing the effect of β. While η is fixed, β varies. Let point A correspond to *, where $\alpha = 0.1$ and $e = 0.6$. The other parameter values are $\Gamma = 0.5$, $v = 5$, $c = 0.2$, and $\eta = 4$.

5 Conclusion

The principle result of the analysis is that, given the harmful effect of infected fish on pelicans ($\eta > 1$), the pelicans cannot coexist with the infected fish; mathematically this corresponds to the lack of a stable **SIP** equilibrium. This lack of coexistence of I and P is caused by the properties that $g_p < 0$, which implies that P is bad for I, and h_i is proportional to $1 - \eta$, meaning that I is bad for P (with $\eta > 1$). Biologically, the lack of IP co-existence can be attributed to direct competition, where the pelicans and infection "prey" upon the susceptible fish. With this direct competition, both "predators" are also harming each other, and only one can prevail. In our analysis, a movement from region 3 to region 1 indicates a definite change in the convergence of equilibrium points. This initiates the start of the epidemic and the eventual extinction of the pelicans. We speculate that this shift is the result of pollution, as the increase in pollution weakens the immunity of the fish making them susceptible to infection. A remedy for this situation would require a restoration of the lake and possible removal of infected fish. This should result in a return to region 3 and **SP** convergence. For purely mathematical interest, one may desire to analyze the situation where it is less harmful for pelicans to consume infected fish (i.e. $\eta < 1$); this may correspond to diseases other than *Avian botulism*.

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