# S. I. S Model for Disease Transmission from Prey to Predator\*

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**Abstract:** A model for disease transmission in a predator-prey system has been proposed and analyzed in this paper. From infected prey, the disease is spreading to susceptible prey and consequently to predator species. The disease does not cause immunity in the prey and predator species. Therefore, SIS (Susceptible-Infected-Susceptible) models are considered for both the species. Conditions for the existence and stability of disease free prey predator system are obtained. The next generation approach is used to obtain the epidemiological threshold quantities  $R_0$ ,  $R_1$  for the model system. Conditions for endemic disease in prey species are discussed. The predator may become extinct due to disease in prey. Disease is not found to be endemic in both the prey and predator species. **Keywords:** Predator, prey, Equilibrium point, carrying capacity, Basic reproduction number, Stability

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#### 1. Introduction

In natural ecosystem, regulatory mechanisms for evolution of biological species are provided by the predator-prey interactions. Not only the disease in the system affects the dynamics of prey predator population, but the prey predator interactions also affect the dynamics of disease<sup>1-4</sup>. Some previous studies of infectious diseases in animal populations focused on the effects of disease-induced mortality or disease-reduced reproduction in the regulation of natural populations (in their natural habitats)<sup>1, 3-5</sup> Reduced population sizes and destabilization of equilibrium into oscillations are caused by the presence of infectious disease in one or both of the populations.

The fact that predators take a disproportionate number of infected preys has been confirmed by earlier studies  $^{1, 3, 6}$ . Infected prey is more vulnerable to predation on account of the fact that the disease may impair their ability

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to protect themselves and expose them to predators<sup>7</sup>. This enhanced vulnerability of infected prey may lead to persistence of the predator (which would otherwise be starved and become extinct). Further, predation of the infected prey may lead to the disappearance of disease which may otherwise be endemic in prey population<sup>4</sup>. Together with no reproduction in infected prey, as predation on infected prey increases, the infection tends to destabilize prey-predator interactions <sup>1</sup>.

Most of the eco-epidemiological studies are restricted to the situations where only prey species can get infection. Few investigations <sup>1, 6, 8</sup> consider the spread of disease from prey to predator through predation of infected prey. For the purpose of eliminating either the disease from predator- prey system or eliminating the menace of the predator by controlling disease in prey, a simplified eco- epidemiological four species prey predator SI model <sup>8</sup> is shown to be useful.

This paper proposes a predator- prey model. The disease is spreading from infected prey to susceptible prey and consequently to the predator species. Both, the prey and the predator species are compartmentalized into susceptible and infected classes. The disease does not cause immunity to the prey and the predator species. Consequently, SIS model is considered for both the prey and the predator.

## 2. Mathematical Model

Let  $N_1(t) = S_1 + I_1$  and  $N_2(t) = S_2 + I_2$  be the density of prey and predator species respectively where  $S_i$ ,  $I_i$  denote the density of susceptible and infected population of specific population. It is assumed that only susceptible prey is capable of reproducing logistically. The infected prey are weakened due to disease and become easier to catch, while susceptible prey can easily escape the predation. The infected prey and predator do not reproduce but still continue to consume resources. The susceptible predator gets food from infected prey for its survival. It may become infected due to interaction with infected prey. Mortality rate of infected predator is higher than that of susceptible predator. These assumptions lead to the following mathematical model:

(1.1) 
$$\frac{dS_1}{dt} = rS_1 \left( 1 - \frac{S_1 + I_1}{K_1} \right) - a_1 S_1 (S_2 + I_2) - c_1 S_1 I_1 + \gamma_1 I_1,$$

(1.2) 
$$\frac{dI_1}{dt} = c_1 S_1 I_1 - a_2 I_1 (S_2 + I_2) - d_1 I_1 - \gamma_1 I_1,$$

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(1.3) 
$$\frac{dS_2}{dt} = -a_2S_2I_1 + (-d_2 + ka_1S_1)S_2 + \gamma_2I_2,$$

(1.4) 
$$\frac{dI_2}{dt} = a_2 S_2 I_1 - d_3 I_2 - \gamma_2 I_2,$$

Various positive parameters in the model are described as

- $a_1$ ,  $a_2$ : Predation rate on the susceptible and infected prey respectively  $(a_2 > a_1)$ 
  - k: Feeding efficiency of predator
  - r: Intrinsic growth rate constant of susceptible prey
  - $K_1$ : Carrying capacity of environment with respect to prey species
  - $c_1$ : Disease incidence rate
- $\gamma_1$ ,  $\gamma_2$ : The recovery rate of infected prey and predator to susceptible respectively
  - $d_1$ : Net death rate of infected prey
- $d_2, d_3$ : Death rate of susceptible and infected predator respectively  $(d_3 > d_2)$

Initial conditions associated with the system are:  $S_1(0) \ge 0$ ,  $S_2(0) \ge 0$ ,  $I_1(0) \ge 0$  and  $I_2(0) \ge 0$ .

# **Mathematical Analysis:**

**Theorem 2.1** All the solutions of the system (1) which initiate in  $R^4_+$  are uniformly bounded.

**Proof:** By theorems of Nagumo <sup>9</sup>, it can be easily proved that  $S_1$ ,  $I_1$ ,  $S_2$  and  $I_2$  remain positive.

Let  $W_1 = S_1 + I_1$ 

Add (1.1) and (1.2) and its simplification gives

$$\frac{d(S_1+I_1)}{dt} \le rS_1\left(1-\frac{S_1}{K_1}\right) - d_1I_1.$$

For arbitrarily chosen  $\eta_1$ , this simplifies to

$$\frac{d(S_1+I_1)}{dt} + \eta_1(S_1+I_1) \le (r+\eta_1)S_1 - \frac{rS_1^2}{K_1} - (d_1-\eta_1)I_1,$$

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$$\frac{dW_1}{dt} + \eta_1 W_1 \le \frac{K_1 (r + \eta_1)^2}{4r} - \frac{r}{K_1} \left( S_1^2 - (r + \eta_1) \frac{K_1}{r} S_1 + \frac{K_1^2 (r + \eta_1)^2}{4r^2} \right) - (d_1 - \eta_1) I_1,$$

$$\frac{dW_1}{dt} + \eta_1 W_1 \le \frac{K_1 (r + \eta_1)^2}{4r} - \frac{r}{K_1} \left( S_1 - (r + \eta_1) \frac{K_1}{2r} \right)^2 - (d_1 - \eta_1) I_1,$$

Choosing  $\eta_1 < d$  and applying the results of (Birkhoff and Rota, 1982<sup>10</sup>), yields

$$0 < W_1(S_1(t), I_1(t)) \le \frac{L_1}{\eta_1} (1 - e^{-\eta_1 t}) + W_1(S_1(0), I_1(0)) e^{-\eta_1 t},$$

as  $t \to \infty$ , it gives

$$0 < S_1 + I_1 \le \frac{L_1}{\eta_1}$$
, Where  $L_1 = \frac{K_1(r + \eta_1)^2}{4r}$ ,

 $\Rightarrow$   $W_1 = S_1 + I_1$  is bounded.

 $\Rightarrow$  From positivity of  $S_1$  and  $I_1$ ,  $0 \le S_1 \le \frac{L_1}{n}$ ;  $0 \le I_1 \le \frac{L_1}{n}$ .

Similarly defining another positive definite function W as

$$W = S_1 + I_1 + S_2 + I_2.$$

Proceeding in the similar manner and choosing  $\eta = \min(d_1, d_2, d_3)$  yields

$$0 < W(S_1, I_1, S_2, I_2) \le \frac{L_1(r+\eta)}{\eta \eta_1} (1 - e^{-\eta t}) + W(S_1(0), I_1(0), S_2(0), I_2(0)) e^{-\eta t}.$$

As  $t \to \infty$ , it gives

$$0 < W \le \frac{L_1(r+\eta)}{\eta \eta_1} = L$$
, where  $L = \frac{L_1(r+\eta)}{\eta \eta_1}$ 

 $\Rightarrow W = S_1 + I_1 + S_2 + I_2$  is bounded.

 $\Rightarrow$  Hence all solutions of (2.1) that initiate in  $R_+^4$  are confined in the region

$$B = \{ (S_1, I_1, S_2, I_2) \in R_+^4 : S_i \le L; I_i \le L; i = 1, 2 \},\$$

The following equilibrium points exist for the system (1): The trivial equilibrium point  $E_0(0,0,0,0)$  and the axial equilibrium point

 $E_1(K_1, 0, 0, 0)$  always exist.

The planar equilibrium point  $E_2(S'_1, I'_1, 0, 0)$  in  $S_1 - I_1$  plane is obtained as  $S_{1}^{'} = \frac{d_{1} + \gamma_{1}}{c_{1}}, I_{1}^{'} = \frac{rS_{1}^{'}(c_{1}K_{1} - d_{1} - \gamma_{1})}{(c_{1}d_{1}K_{1} + rd_{1} + r\gamma_{1})}, S_{1}^{'} + I_{1}^{'} < K_{1}.$ 

The equilibrium point  $E_2(S'_1, I'_1, 0, 0)$  exists provided

(2.1) 
$$(c_1K_1 - d_1 - \gamma_1) > 0.$$

In this case, the predator species is eliminated and the disease persists in the prey species.

Another disease free planar equilibrium point  $E_3(\hat{S}_1, 0, \hat{S}_2, 0)$  on  $S_1 - S_2$ plane exists provided

$$(2.2) K_1 > \frac{d_2}{ka_1}.$$

The equilibrium level densities of susceptible prey and predator are

$$S_1^{"} = \frac{d_2}{ka_1}$$
 and,  $S_2^{"} = \frac{r(ka_1K_1 - d_2)}{ka_1^2K_1}$ .

For non-zero equilibrium point  $E^*(S_1^*, I_1^*, S_2^*, I_2^*)$  of the system (1),  $S_1^*$  is given by the positive root of the quadratic

(2.3a) 
$$f(s) = AS^2 - BS + C = \mathbf{0},$$
  
where 
$$A = ra_2d_3 + a_1c_1d_3K_1 + ka_1(d_3 + \gamma_2)(r + c_1K_1) > 0,$$
$$B = [ra_2d_3K_1 + a_1K_1d_3(d_1 + \gamma_1) + (d_3 + \gamma_2)\{(r + c_1K_1)d_2 + \gamma_1ka_1K_1\}],$$
$$C = \gamma_1d_2K_1(d_3 + \gamma_2),$$

A, B and C being positive, the quadratic has two positive roots if  $B^2 > 4AC$ . Also

(2.3b) 
$$I_1^* = \frac{ka_1(d_3 + \gamma_2)(S_1^* - S_1^*)}{a_2d_3},$$

(2.3c) 
$$S_2^* = \frac{c_1 d_3 (S_1^* - S_1^{'})}{a_2 (d_3 + (S_1^* - S_1^{''})ka_1)},$$

(2.3d) 
$$I_2^* = \frac{ka_1(S_1^* - S_1^*)S_2^*}{d_3}$$

It is clear from (2.3b) and (2.3c) that  $S_1^* \ge S_1^*$  and  $S_1^*$ . Further,  $S_1^* + I_1^* \le K_1$ , which implies that  $S_1^* \le K_1 - I_1^*$ . Hence,  $S_1^* \in (\bar{S}, \hat{S})$  where  $\bar{S} = \max\left(\frac{d_2}{ka_1}, \frac{d_1 + \gamma_1}{c_1}\right)$  and  $\hat{S} = \frac{K_1 a_2 d_3 + (d_3 + \gamma_2) d_2}{a_2 d_3 + (d_3 + \gamma_2) ka_1}$ . Further, the root  $S_1^*$  of

the quadratic is unique provided  $f(\overline{S})f(\hat{S}) < 0$ . This will also ensure the uniqueness of positive equilibrium point  $E^*(S_1^*, I_1^*, S_2^*, I_2^*)$ .

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The Basic Reproduction Number is defined as the average number of secondary cases when one infective is introduced into a completely susceptible host population. Let  $R_0$  is the basic reproduction number for isolated prey population and  $R_1$  is the basic reproduction in the prey population when both prey and predator are present in the system. Using next generation approach <sup>11</sup>, the epidemiological threshold quantities  $R_0$  and  $R_1$  for the system (1) are computed as

$$R_0 = \frac{c_1 K_1}{d_1 + \gamma_1}$$
 and  $R_1 = \frac{c_1 S_1^{"}}{d_1 + \gamma_1 + a_2 S_2^{"}}$ ,

It may be noted that  $R_1 < R_0$ . Also, the greater vulnerability of prey will reduce  $R_1$ .

The variational matrix V of given system (1) is given by

$$V = \begin{pmatrix} a_{11} & -\frac{rS_1}{K_1} - c_1S_1 + \gamma_1 & -a_1S_1 & -a_1S_1 \\ c_1I_1 & a_{22} & -a_2I_1 & -a_2I_1 \\ ka_1S_2 & -a_2S_2 & a_{33} & \gamma_2 \\ 0 & a_2S_2 & a_2I_1 & -d_3 - \gamma_2 \end{pmatrix},$$

where

$$a_{11} = r - \frac{2rS_1}{K_1} - a_1(S_2 + I_2) - c_1I_1 - \frac{rI_1}{K_1}$$
$$a_{22} = c_1S_1 - d_1 - \gamma_1 - a_2(S_2 + I_2)$$
$$a_{33} = -a_2I_1 + (-d_2 + ka_1S_1)$$

**Theorem 2.2** The trivial equilibrium point  $E_0(0,0,0,0)$  is a saddle point with unstable manifold along  $S_1$  direction.

**Proof**: From variational matrix V, the characteristic equation about  $E_0(0,0,0,0)$  is obtained as

$$(\lambda - r)(\lambda + d_1 + \gamma_1)(\lambda + d_2)(\lambda + d_3 + \gamma_2) = 0.$$

All the eigenvalues are negative except the one along  $S_1$  i.e.  $\lambda = r$ . Hence result.

**Theorem 2.3** The axial equilibrium  $E_1(K_1, 0, 0, 0)$  is locally asymptotically stable provided

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(2.4)  $R_0 < 1 \text{ and } ka_1K_1 < d_2.$ 

**Proof**: The characteristic equation about  $E_1(K_1, 0, 0, 0)$  is obtained as

$$(\lambda + r)(\lambda - c_1K_1 + d_1 + \gamma_1)(\lambda + d_2 - ka_1K_1)(\lambda + d_3 + \gamma_2) = 0.$$

All the eigenvalues are negative provided

$$c_1 K_1 < d_1 + \gamma_1$$
 and  $k a_1 K_1 < d_2$ 

Hence result.

When the perturbations are taken in  $S_1 - I_1$  plane only, then the equilibrium point  $E_1(K_1, 0, 0, 0)$  is locally asymptotically stable for  $R_0 < 1$ . However in the presence of predator population the stability depends upon the dynamics of predator also as is evident from (2.4). In fact, when the feeding efficiency of predator is sufficiently low then the disease will die out from both the prey and predator under condition (2.4) and the equilibrium point  $E_1(K_1, 0, 0, 0)$  is locally asymptotically stable.

The equilibrium  $E_1(K_1, 0, 0, 0)$  is saddle point when

(2.5) 
$$K_1 > \max\left(\frac{d_1 + \gamma_1}{c_1}, \frac{d_2}{ka_1}\right)$$

Further, when  $E_1(K_1, 0, 0, 0)$  is locally asymptotically stable, it is observed from (2.1) and (2.2) that the equilibrium points  $E_2(S_1, I_1, 0, 0)$  and  $E_3(S_1, 0, S_2, 0)$  do not exist. Further, the stability of  $E_1$  also excludes the existence of  $E^*(S_1, I_1, S_2, I_2)$ .

**Theorem 2.4** If  $R_0 > 1$ , then  $E_2(S_1, I_1, 0, 0)$  locally asymptotically stable provided

(2.6) 
$$ka_1S'_1 - \frac{a_2d_3}{(d_3 + \gamma_2)}I'_1 - d_2 < 0.$$

**Proof**: From variational matrix V, the characteristic equation about  $E_2(S_1, I_1, 0, 0)$  is obtained as

$$\left\{\lambda^{2} + \left(\frac{rS_{1}'}{K_{1}} + \frac{\gamma_{1}I_{1}'}{S_{1}'}\right)\lambda + c_{1}I_{1}'\left(\frac{rS_{1}'}{K_{1}} + c_{1}S_{1}' - \gamma_{1}\right)\right\}\left(\lambda^{2} + P\lambda + Q\right) = 0,$$

where

$$P = a_2 I_1' - ka_1 S_1' + d_2 + d_3 + \gamma_2,$$
  
$$Q = (d_2 - ka_1 S_1)(d_3 + \gamma_2) + d_3 a_2 I_1'.$$

Since the expression  $(c_1S_1' - \gamma_1) > 0$  is always positive, the first quadratic factor gives eigenvalues with negative real part. Therefore, the system around  $E_2(S_1', I_1', 0, 0)$  is locally asymptotically stable provided (2.6) is satisfied.

It may be concluded from theorem 2.4 that the disease may be endemic in prey population subject to the condition (2.6) when the basic reproduction number  $R_0$  is above the threshold. In this case, the predator species goes to extinction.

The condition of instability of  $E_2(S_1, I_1, 0, 0)$  if it exists, is given by

(2.7) 
$$ka_1S_1' - \frac{a_2d_3}{(d_3 + \gamma_2)}I_1' - d_2 > 0.$$

**Theorem 2.5** The disease free equilibrium state  $E_3(\hat{S}_1, 0, \hat{S}_2, 0)$ , is locally asymptotically stable provided

$$c_1\hat{S}_1 - a_2\hat{S}_2 - d_1 - \gamma_1 < 0$$
.

**Proof:** From variational matrix V, the characteristic equation about  $E_3(\hat{S}_1, 0, \hat{S}_2, 0)$  is obtained as

$$\left\{\lambda^{2} + \left(\frac{rS_{1}^{"}}{K_{1}} + a_{1}S_{2}^{"}\right)\lambda + ka_{1}^{2}S_{1}^{"}S_{2}^{"}\right\}(c_{1}S_{1}^{"} - a_{2}S_{2}^{"} - d_{1} - \gamma_{1} - \lambda)(-d_{3} - \lambda) = 0.$$

Since the quadratic factor always yields two eigenvalues with negative real part, the condition for stability is

(2.8) 
$$c_1 \hat{S}_1 - a_2 \hat{S}_2 - d_1 - \gamma_1 < 0$$

This completes the proof.

Its simplification gives  $R_1 < 1$ .

If the feeding efficiency of predator population is sufficiently high so as  $K_1 > \frac{d_2}{ka_1}$  then the predator population persist. When the basic reproduction number  $R_1$  is below the threshold, then the disease dies out and prey and

predator population go to their usual persistent equilibrium values and the equilibrium point  $E_3(\hat{S}_1, 0, \hat{S}_2, 0)$  is stabilized.

The greater vulnerability of prey to predation may be responsible for persistence of disease free prey predator population.

The disease free equilibrium state  $E_3(\hat{S}_1, 0, \hat{S}_2, 0)$  if it exist, is unstable if

(2.9) 
$$c_1 \hat{S}_1 - a_2 \hat{S}_2 - d_1 - \gamma_1 > 0$$

**Theorem 2.6** The non-zero equilibrium point  $E^*(S_1^*, I_1^*, S_2^*, I_2^*)$ , if exists, is not locally asymptotically stable.

**Proof:** The characteristic equation about  $E^*$  is obtained as

(2.10) 
$$\lambda^4 + A_0 \lambda^3 + A_1 \lambda^2 + A_2 \lambda + A_3 = 0,$$

where

$$\begin{split} A_{0} &= d_{3} + \gamma_{2} + \frac{\gamma_{1}I_{1}}{S_{1}} + \frac{rS_{1}}{K_{1}} + \frac{\gamma_{2}I_{2}}{S_{2}}, \\ A_{1} &= \left(d_{3} + \gamma_{2} + \frac{\gamma_{2}I_{2}}{S_{2}}\right) \left(\frac{\gamma_{1}I_{1}}{S_{1}} + \frac{rS_{1}}{K_{1}}\right) + \left(cS_{1} - \gamma_{1} + \frac{rS_{1}}{K_{1}}\right) + ka_{1}^{2}S_{1}S_{2}, \\ A_{2} &= c_{1}\left(c_{1}S_{1} - \gamma_{1}\right) \left(d_{3} + \gamma_{2} + \frac{\gamma_{2}I_{2}}{S_{2}}\right) I_{1} + \left\{c_{1}\left(d_{3} + \gamma_{2}\right) - \gamma_{2}a_{2}\right\} \frac{rS_{1}I_{1}}{K_{1}} \\ &+ \left[\frac{r\gamma_{2}S_{1}I_{2}}{K_{1}S_{2}} + ka_{1}^{2}S_{1}S_{2} + \left\{\left(a_{1} - c\right) - \frac{r}{K_{1}}\right\} ka_{1}S_{1}I_{2} - \frac{d_{3}a_{2}^{2}I_{1}I_{2}}{d_{3} + \gamma_{2}}\right] \left(d_{3} + \gamma_{2}\right) \\ &+ \frac{c_{1}\gamma_{2}rS_{1}I_{1}I_{2}}{K_{1}S_{2}} + (\gamma_{1}ka_{1} - d_{3}a_{2})I_{1}S_{2}, \end{split}$$

$$A_{3} = -\left\{ \left( c_{1}d_{3}a_{1} + \frac{d_{3}ra_{2}}{K_{1}} + \frac{rka_{1}a_{2}I_{1}^{*}}{K_{1}} + \frac{Drka_{1}}{K_{1}} \right) S_{2}^{*} + \left( \frac{ra_{2}}{K_{1}} + c_{1}a_{1} \right) I_{2}^{*} \right\} a_{2}S_{1}^{*}I_{1}^{*}$$
$$- \frac{\gamma_{1}a_{2}^{2}d_{3}I_{1}^{*2}(S_{2} + I_{2})}{S_{1}} - (cS_{1}^{*} - \gamma_{1}) \left\{ a_{2}I_{1}^{*} + (d_{3} + \gamma_{2}) \right\} ka_{1}a_{2}I_{1}^{*}S_{2}^{*}.$$

As the constant term  $A_3$  is always negative, at least one eigenvalue of the equation (2.10) will be positive, therefore  $E^*(S_1^*, I_1^*, S_2^*, I_2^*)$  is not locally asymptotically stable.

It is observed that  $E_2(S_1, I_1, 0, 0)$ , if it exist, is locally asymptotically stable provided  $E_3(\hat{S}_1, 0, \hat{S}_2, 0)$  is unstable and vice-versa. However, it is possible for some choice of parameters that both  $E_2(S_1, I_1, 0, 0)$  and  $E_3(\hat{S}_1, 0, \hat{S}_2, 0)$  exist and locally stable.

It is also observed that when both  $E_2(S_1, I_1, 0, 0)$  and  $E_3(\hat{S}_1, 0, \hat{S}_2, 0)$  are stable then non-zero equilibrium point  $E^*(S_1^*, I_1^*, S_2^*, I_2^*)$  always exist.

## 3. Numerical simulations

Numerical simulations have been carried out to investigate dynamics of the proposed model in the 3-D system. Computer simulations have been performed on MATLAB for different set of parameters.

Consider the following set of parametric values:

(3.1) 
$$r = 0.1, K_1 = 50, \gamma_1 = 0.25, \gamma_2 = 0.3, d_1 = 0.7, d_2 = 0.6, d_3 = 0.6, c_1 = 0.01, a_1 = 0.8, a_2 = 0.9, k = 0.012.$$

The system (1) has equilibrium point  $E_1(50, 0, 0, 0)$  for the data set (3.1). It is locally asymptotically stable as the computed value of  $R_0$  is 0.5882 < 1 and the value of feeding efficiency k of predator population is sufficiently low and the condition  $K_1 < \frac{d_2}{ka_1}$  is satisfied. The solution trajectories in phase space  $S_1 - I_1 - S_2$  are drawn in Fig 1 for different initial values.

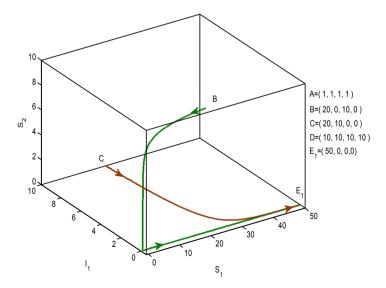


Fig 1a: Phase plot depicting the stability of equilibrium point  $E_1$  in phase space  $S_1 - I_1 - S_2$ 

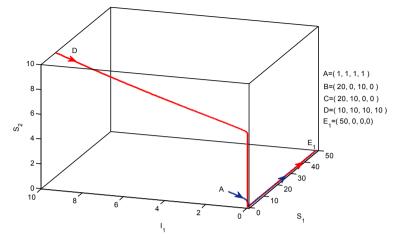


Fig 1b: Phase plot depicting the stability of equilibrium point  $E_1$  in phase space  $S_1 - I_1 - S_2$ 

Now consider the following set of parametric values:

(3.2) 
$$r = 0.59, K_1 = 250, \gamma_1 = 0.27, \gamma_2 = 0.3, d_1 = 0.7, d_2 = 0.45, d_3 = 0.6, c_1 = 0.001, a_1 = 0.07, a_2 = 0.09, k = 0.1.$$

point  $E_1(250, 0, 0, 0)$ For this the set. is unstable and  $E_3(64.2857, 0, 6.2612, 0)$ is locally asymptotically stable as feeding efficiency k = 0.1 is sufficiently large such that  $K_1 > \frac{d_2}{ka_1} (= 64.2857)$  and  $R_1 = 0.0419 < 1$ . Fig. 2 shows the stability of the equilibrium point  $E_3(64.2857, 0, 6.2612, 0)$ . Further, the equilibrium point  $E_2(S_1, I_1, 0, 0)$  does not exist in this case as  $K_1 < \frac{(d_1 + \gamma_1)}{c_1} (= 970)$ .

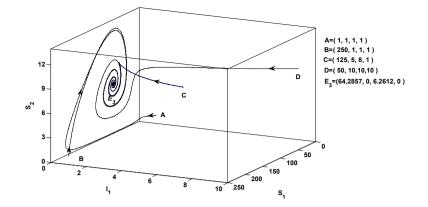


Fig 2: Phase plot depicting the stable behavior of the equilibrium point  $E_3$ 

Consider the following data with other parameter as in (3.2): (3.3)  $K_1 = 500, c_1 = 0.025, a_1 = 0.01, a_2 = 0.03, k = 0.1.$ 

The equilibrium point  $E_1(500, 0, 0, 0)$  is unstable and  $E_2(38.8, 28.3133, 0, 0)$  exists as infection rate  $c_1$  in prey population is high enough so that  $K_1 > \frac{(d_1 + \gamma_1)}{c_1}$  and  $E_2(38.8, 28.3133, 0, 0)$  becomes locally stable as condition (2.5) is satisfied and  $R_0 = 12.8866 > 1$ . The stability of the equilibrium point  $E_2(38.8, 28.3133, 0, 0)$  is shown in Fig 3. Further, it was observed that the equilibrium point  $E_3(\hat{S}_1, 0, \hat{S}_2, 0)$  does not exist in this case as  $K_1 < \frac{d_2}{ka_1} (= 4500)$ .

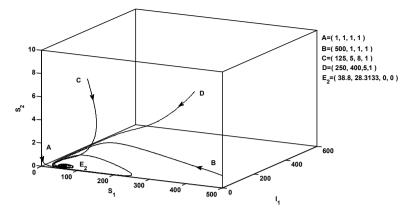


Fig 3: Phase plot depicting the stable behavior of the equilibrium point  $E_2$ 

Now we examine the case when  $E_2$  is stable and  $E_3$  is unstable. For this take following data set of parameters.

(3.4) 
$$r = 0.02, K_1 = 100, \gamma_1 = 0.01, \gamma_2 = 0.3, d_1 = 0.01, d_2 = 0.11, d_3 = 0.15, c_1 = 0.002, a_1 = 0.01, a_2 = 0.02, k = 0.33.$$

As  $K_1$  is greater than computed value 33.3333 of  $\frac{d_2}{ka}$  and 10 of  $\frac{(d_1 + \gamma_1)}{c}$  in this case, the equilibrium point  $E_1(100, 0, 0, 0)$  is unstable and planar equilibrium  $E_{2}(10, 15, 0, 0)$ points both the and exists.  $E_3(33.3333, 0, 1.3333, 0)$ The equilibrium point  $E_3(33.3333, 0, 1.3333, 0)$  is found to be unstable as it satisfies the condition (2.8) i.e.  $c_1 S_1^{"} - a_2 S_2^{"} - d_1 - \gamma_1 = 0.02 > 0$  and  $E_2(10, 15, 0, 0)$  is found to be locally asymptotically stable as condition (2.6) is satisfied i.e.  $ka_1S_1' - \frac{a_2d_3}{(d_2 + \gamma_2)}I_1' - d_2 = -0.1770 < 0$  and  $R_0 = 10 > 1$ . The solution trajectories in phase space  $S_1 - I_1 - S_2$  are drawn in Fig 4 for different initial values. All these trajectories converge to  $E_2(10, 15, 0, 0)$ . Starting with initial value in the neighborhood of the point  $E_3(33.3333, 0, 1.3333, 0)$ , the solution trajectory approaches to the equilibrium point  $E_2(10, 15, 0, 0)$ . This confirms the instability of  $E_3(33.3333, 0, 1.3333, 0)$ .

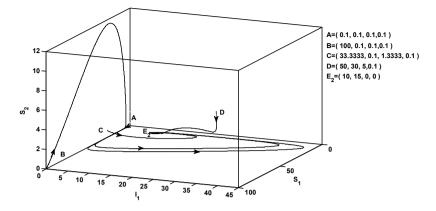


Fig 4: Phase plot depicting the stability of  $E_2$  and instability of  $E_3$ 

Now we take the case when  $E_3$  is stable and  $E_2$  unstable, for this consider following set of data

(3.5) 
$$r = 0.75, K_1 = 45, \gamma_1 = 0.29, \gamma_2 = 0.3, d_1 = 0.6, d_2 = 0.9, d_3 = 0.95, c_1 = 0.02, a_1 = 0.3, a_2 = 0.5, k = 0.73.$$

As  $K_1$  is greater than computed value 4.3379 of  $\frac{d_2}{ka_1}$  and 44.5 of  $\frac{(d_1 + \gamma_1)}{c_1}$  in this case, the equilibrium point  $E_1(45,0,0,0)$  is unstable and system (1) has two planar equilibrium points, say  $E_2(44.5,0.2764,0,0)$  and  $E_3(4.3379,0,2.2590,0)$ . The equilibrium point  $E_3(4.3379,0,2.2590,0)$  is found to be stable as it satisfies the condition (2.8)) i.e.  $c_1S_1^r - a_2S_2^r - d_1 - \gamma_1 = -1.9327 < 0$  and  $E_2(44.5,0.2764,0,0)$  is found to be unstable as  $R_0 = 1.0112 > 1$  and condition (2.6) is not satisfied i.e.  $ka_1S_1' - \frac{a_2d_3}{(d_3 + \gamma_2)}I_1' - d_2 = 8.6899 > 0$ . The solution trajectories in phase space  $S_1 - I_1 - S_2$  are drawn in Fig 5 for different initial values. All these trajectories approach to  $E_3(4.3379,0,2.2590,0)$ . Convergence of solution trajectory to equilibrium  $E_3(4.3379,0,2.2590,0)$  with initial value near  $E_2(44.5,0.2764,0,0)$  also confirms the instability of  $E_2(44.5,0.2764,0,0)$ .

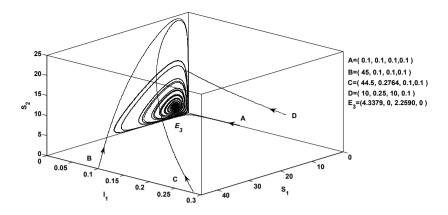


Fig 5: Phase plot depicting the stability of  $E_3$  and instability of  $E_2$ 

The following data is selected for which all the equilibrium points exist:

(3.6) 
$$r = 0.6, K_1 = 50, \gamma_1 = 0.2, \gamma_2 = 0.3, d_1 = 0.6, d_2 = 0.3, d_3 = 0.35, c_1 = 0.2, a_1 = 0.2, a_2 = 0.3, k = 0.25.$$

In this case, the equilibrium point  $E_1(50,0,0,0)$  is unstable. It is observed that both the equilibrium points  $E_2(4.0, 3.4074, 0, 0)$  and  $E_3(6.0, 0, 2.64, 0)$  are locally asymptotically stable. The point  $E_2(4.0, 3.4074, 0, 0)$  is stable because

of  $ka_1S_1' - \frac{a_2d_3}{d_3 + \gamma_2}I_1' - d_2 = -0.6504 < 0$  and  $R_0 = 12.5 > 1$ . Also,

 $E_3(6.0, 0, 2.64, 0)$  is locally asymptotically stable as  $R_1 = 0.7538 < 1$ . Both the equilibrium points have their own domains of attractions. Trajectories with different initial conditions converge to different equilibrium points (See Fig 6).

The instability of non-zero equilibrium point  $E^*(7.2908, 0.3995, 1.8523, 0.3416)$  can be seen from Fig 6.

It is also noticed that when even a very small perturbation is taken from  $E^*(7.2908, 0.3995, 1.8523, 0.3416)$  to E(7.2908, 0.3995, 1.85, 0.3416), along z-direction, the trajectory converges to  $E_2(4.0, 3.4074, 0, 0)$ . Whereas, when the perturbation is taken to F(7.2908, 0.3995, 1.86, 0.3416), the trajectory of the system converges to  $E_3(6.0, 0, 2.64, 0)$ .

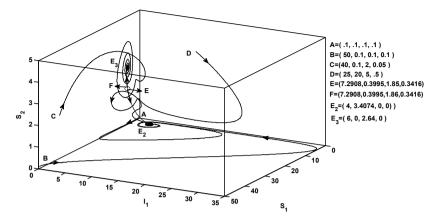


Fig 6: Phase plot depicting the stable behaviour of  $E_2$ ,  $E_3$ 

### 5. Discussion

A predator-prey model has been proposed and analyzed in this paper. From the infected prey, the disease is spreading to susceptible prey and consequently to the predator species. The predator-prey species are categorized into susceptible and infected classes. It is assumed that the disease does not cause immunity in the prey and predator species. The SIS model system admits four boundary equilibrium points and one non-zero interior equilibrium point. The dynamic behavior of the system around each equilibrium point has been studied and threshold values for basic Reproduction numbers  $R_0$  and  $R_1$  are computed. It has been observed the predator population becomes extinct and the disease dies out from the prev population for sufficiently small feeding efficiency of predator when the basic reproduction number  $R_0$  in prey population is below the threshold. If the basic reproduction number  $R_0$  in the prey population is above the threshold, the disease in prev population approaches endemic level and predator population becomes extinct. Since the logistic growth for infected prey is not considered, prey population,  $S'_1 + I'_1$  will never reach its carrying capacity. Disease in prey may be responsible for elimination of predator.

In case the feeding efficiency of predator population is sufficiently high so that  $K_1 > \frac{d_2}{ka_1}$  then the predator population persists. When the basic reproduction number *R* is below the threshold, then the disease dies out and

reproduction number  $R_1$  is below the threshold, then the disease dies out and prey and predator population both go to their persistent equilibrium values

and the equilibrium point  $E_3(\hat{S}_1, 0, \hat{S}_2, 0)$  is stabilized. The difference in death rates of infected and susceptible species may be responsible for instability of the equilibrium point  $E^*(S_1^*, I_1^*, S_2^*, I_2^*)$ . Therefore, the disease may not be endemic in prev predator system

### References

- 1. R. M. Anderson and R. M. May, The invasion, persistence, and spread of infectious diseases within animal and plant communities, *Phillos. Trans. R. Soc. Lond.*, **314** (1986) 533-570.
- 2. E. Venturino, The influence of diseases on Lotka –Volterra system, *Rky. Mt. J. Math.*, **24** (1984) 381-402.
- 3. J. Chattopadhyay and O. Arino, A predator-prey model with disease in the prey, *Nonlinear Anal.*, **36**(1999) 747-766.
- 4. H. W. Hethcote, Wendi Wang, L. Han and Z. Ma, A predator-prey model with infected prey, *Theoretical Population Biology.*, **66**(2004) 259-268.
- 5. R. M. Anderson and R. M. May, Regulation and stability of host-parasite population interactions I: regulatory processes, *J. Anim. Ecol.*, **47** (1978) 219-247.
- 6. K. P. Hadeler and H. I. Freedman, Predator-prey populations with parasitic infection, *J. Math. Biol.*, **27**(1989) 609-631.
- A. P. Dobson, The population biology of parasite-induced changes in host behavior, Q. Rev. Biol., 63(1988) 139-165.
- 8. E. Venturino, On epidemics crossing the species barrier in interacting population models, *Varahmihir J. Math. Sci.*, **6** (2006) 247-263.
- 9. N. Nagumo, Uber die lage der integralkurven gewonlicher di\_erentialgleichunger, *Proc. Phys. Math. Soc. Japan*, **24** (1942) 551.
- 10. G. Birkhoff and G. C. Rota, Ordinary differential equation, Ginn, Boston (1982).
- 11. Castillo-Chavez, S. Blower, P. Driessche, D. Kirschner and A. A. Yakubu, Mathematical Approaches for Emerging and Reemerging Infectious Diseases: An Introduction, Springer-Verlag, New York, 2002.