Article

Stability analysis of a predator-prey model using Takagi-Sugeno method

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Abstract

The current study is based on a predator-prey model with infection that affects only predator species. Predators are divided into two categories such as the susceptible predator and the infected predator, which are feeding on prey species. The Takagi-Sugeno (T-S) based fuzzy impulsive control model was used to explore the stability of the Lotka-Volterra predator-prey system. Numerical simulation provides global stability and the fuzzy solution.

Keywords T-S model; stability; Lotka-Volterra; predator-prey system; eco-epidemiology.

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1 Introduction

Disease's impact on ecosystems is a significant topic from both a mathematical and an ecological point of view. As a result, ecologists and academicians have been focusing more and more on the creation of key tools, as well as experimental ecology, to characterize how ecological species are infected (Zhao and Zhang, 2013; Zhang and Liu, 2023). Lotka and Volterra made the first breakthrough in contemporary mathematical ecology for a predator-prey competing species.

The study of disease transmission in animals is known as epidemiology. Following Lotka and Volterra's (Volterra, 1926) pioneering work on the predator-prey model, the latest mathematical ecology has attained an essential position in analytical biology (Maitiet al., 2007; Li and Zhu, 2009; Haque, 2009; Shakil et al., 2015).

Maximum models for the transmissible diseases are framed based on (Kermack and Kendrick, 1927)famous work. The first to combine the fields, ecology and epidemiology are Anderson and May (Anderson and May, 1986), they constructed a predator-prey model in which predator species were infected by disease. In the following period many authors (Haque and Venturino, 2006; Baek, 2010; Haque, 2011;Wang et al., 2013; Elenaet al., 2013; Ferrarini, 2015; Liu and Liu, 2020; Kumari and Mohan, 2021) investigated and developed different predator-prey models in existence of disease. Recently, there has been an extensive

improvement in a fishery's bio-economic management in presence of some infection (Bairagi et al., 2021; Hu et al., 2022).

The consequence of infection in predator-prey model with disease in prey has been investigated enormously in last few years by many researchers. In 1986, the infectious disease transmission into a predator-prey model has been introduced, assuming that the infection is only transmitted inside the prey species (Anderson and May, 1986). The traditional Lotka-Volterra predator-prey model, in which infection spreads among either the prey or the predator (Greenhalgh and Haque, 2007). However, a very less study has been done with infection in predator (Tseng et al., 2001; Wang et al., 2015).

Venturino examined the local properties of eco-epidemic models in predator-prey systems with disease only in the predator population. The infectious disease in the Holling Tanner predator-prey model recorded a number of interesting results (Greenhalgh and Haque, 2007). They concluded that disease in any species can be used as a biological control.

Almost all the physical dynamical systems in real life cannot be represented by linear differential equations. The non-linear model is analyzed with the help of Takagi-Sugeno Fuzzy model. The fuzzy model proposed by Takagi and Sugeno is described by fuzzy IF-THEN rules which represent local input-output relations of a nonlinear system. The main feature of a Takagi-Sugeno fuzzy model is to express the local dynamics of each fuzzy implication (rule) by a linear system model. It develops a systematic approach to generate fuzzy rules from a given input-output data set. Until recently, less work has been done on the stability of Lotka-Volterra predator-prey system with fuzzy impulsive control. The T-S method is very useful as it is less time consuming and easy to solve complex systems. We can easily analyze the stability of complex systems using T-S method (Lin et al., 2022).

In this paper, we look at predator-prey model system with eco-epidemiological implications with three species: prey, susceptible predator, and infected predator, in which disease solely affects the predator population.

2 Model Formation

Our mathematical model is based on the following assumptions:

- When there's no predator, the prey population expands operational with a per capita constant r growth rate and a carrying capacity of the environment $c = r/e$, where e represents the prey's intra-specific competition.Thus,

$$\frac{dx}{dt} = rx\left(1 - \frac{x}{c}\right) \tag{1}$$

where $x(t)$denotes the prey population at time.

- The whole predator population $z(t)$ is split into two classes in the presence of injurious infection, among one is the susceptible predator $s(t)$ and the other is the infected predator $i(t)$. As a result, the overall predator population density at time *t*i s –

$$z(t) = s(t) + i(t) \tag{2}$$

- We believe that the disease is solely affecting the predator species, and that the prey population is unaltered. The population of diseased predators does not recover or develop immune system.
- For the susceptible predator and the infected predator, the predation rate or searching efficiency constants are m and n, respectively, because the susceptible predator is more effective than the infected predator; therefore, we assume that the prey is eaten by the susceptible predator based on the basic mass action occurrence. According to the Holling type - II functional response, the diseased

predator eats the prey. We assume that the susceptible predator has no handling time, and the infected predator has a non-zero handling time, which obviously depicts a better ecological situation than assuming both predators have the same predation rate.

- We presume that the disease spreading is governed by the basic rule of mass action.
- Let D is the predator's natural death rate, B is the predator's birth rate, and γ is the predator's disease-related mortality rate.

We propose the following model utilizing a set of nonlinear differential equations based on the above assumptions:

$$x' = rx - ex^2 - mxs - \frac{nxi}{A+x} \quad (3)$$

$$s' = f_1 mxs - \beta is + (B - D)s \quad (4)$$

$$i' = \beta is + \frac{f_2 nxi}{A+x} - \gamma i \quad (5)$$

here all the parameters $r, e, m, n, \gamma, \beta,$ are positive and $(B - D)$can be either sign. $A$is the half saturation constant, $f_1, f_2$is the food conversion rate such that $0 < f_1, f_2 < 1$, βis the rate of transmission of a force of infection.

A matrix differential equation is stated as follows to analyze the system's stability:

$\dot{x} = Ax + \phi(x)$ (6)

where

$$\dot{x}=\begin{pmatrix} \dot{x}(t) \\ \dot{s}(t) \\ i(t) \end{pmatrix},\ A = \begin{bmatrix} r & 0 & 0 \\ 0 & (B-D) & 0 \\ 0 & 0 & -\gamma \end{bmatrix},\phi(x) = \begin{bmatrix} -ex^2 - mxs - \frac{nxI}{A+x} \\ f_1 mx - \beta is \\ \beta is + \frac{f_2 nxi}{A+x} \end{bmatrix}$$

3 T-S Fuzzy Model With Impulsive Effects

Let $\dot{x} = f(x(t))$, here the state variable is $x(t) \in R^n$, and $f \in C[R^n, R^n]$ fulfills the condition $f(0) = 0$, is a compact vector field defined in $W \subseteq R^n$. Using the techniques proposed by Tanaka and Wang (Tanaka et al., 2001; Wang et al., 2012). We can build a fuzzy model for system (3-5) as shown below:

3.1.1 Control Rule *i* (*i*=1, 2,...*r*):

IF $z_1(t)$ is M_{i1}, $z_2(t)$ is M_{i2} ... and $z_p(t)$ is M_{ip} THEN $\dot{x} = A_i x(t)$, where r is no. of T-S fuzzy rules, $z_1(t)$, $z_2(t)$, ... , $z_p(t)$are the premise variables, each $M_{ij}$is a fuzzy set and $A \subseteq R^{n*n}$ is a constant matrix .

Thus, the non-linear equations can be transformed into the following linear equation.

If $x(t)$ is $M_i$then,

$\dot{x}\ (t) = A_i x(t), t \neq \tau_j$ (7)

$\Delta(x) = K_{ij} x(t), t = \tau_j, i = 1,2,3, \dots r, j = 1,2, \dots$ (8)

where

$$A_i = \begin{bmatrix} r - z_1 - z_2 - z_3 & 0 & 0 \\ f_1 z_2 & -z_4 + (B - D) & 0 \\ f_2 z_3 & z_4 & -\gamma \end{bmatrix}$$

and $z_1,\ z_2,\ z_3,\ z_4$ are related to the values of $x(t),\ s(t),\ i(t)$ (here $z_1 = ed_1,\ z_2 = md_2,\ z_3 = \frac{nd_3}{A+d_1},\ z_4 = \beta d_3$). $M_i,\ X(t),\ A_i \in R^{3*3}$, r is the number of the IF-THEN rules, K_{ij} denotes the control of the j_{th} impulsive instant, $\Delta(x)\ |_{t=\tau_j} = x(\tau_j - \tau_{j-1})$.

With center-average deffuzifier, the T-S fuzzy impulsive system may be written as:

$\dot{x}(t) = \sum_{i=1}^{r} h_i\big(z(t)\big)\big(A_i x(t)\big);\ \ t \neq \tau_j$ (9)

$\Delta(x) = \sum_{i=1}^{r} h_i\big(z(t)\big)K_{ij};\ \ t = \tau_j$ (10)

where,

$h_i(z(t)) = \omega_i(z(t))/\sum_{i=1}^{r}\omega_i(z(t)),\ \omega_i\big(z(t)\big) = \prod_{j=1}^{p} M_{ij}(z(t))$.

Obviously, $h_i\big(z(t)\big) \geq 0,\ \sum_{i=1}^{r} h_i\big(z(t)\big) = 1, i = 1, 2, \ldots r$.

4 Stability Analysis

Now we'll look at the impulsive fuzzy system's numerous stability (9) by considering the following theorems.

4.1 Theorem

Assume that λ_i is maximum eigen value of $[A_i^T + A_i]\ (i = 1, 2, 3, \ldots r)$. Let $\lambda(\alpha) = \max_i\{\lambda_i\},\ 0 < \delta_j = \tau_j - \tau_{j-1} < \infty$ is an impulsive distance. If $\lambda(\alpha) \geq 0$ and there exists a constant scalar $\epsilon > 1$ and a semi-positive matrix P, such that:

$\ln(\varepsilon\beta_j) + \lambda(\alpha)\delta_j \leq 0$ (11)

where

$P = C^T C,\ \beta_j = maxC(I + K_{ij})$ (12)

then the system (9-10) is stable globally and asymptotically.

4.2 Theorem

Assume that λ_i is maximum eigen value of $[A_i^T + A_i]\ (i = 1, 2, 3, \ldots r)$. Let $\lambda(\alpha) = \max_i\{\lambda_i\},\ 0 < \delta_j = \tau_j - \tau_{j-1} < \infty$is an impulsive distance. If $\lambda(\alpha) < 0$ and thereexists a constant scalar $0 \leq \epsilon > -\lambda(\alpha)$, such that

$\ln(\beta) - \varepsilon\delta_j \leq 0$ (13)

where

$PA_i = A_i P, P = C^T C,\ \beta_j = maxC(I + K_{ij})$ (14)

then the system (9-10) is stable globally and exponentially.

5 Numerical Simulation

Since most of the biological systems are complex, they should be expressed by applying a fuzzy logical framework that includes expressive reports. The suggested impulsive T-S design model examines the predator-prey system with functional response and impulsive impacts.

By using fuzzy impulsive T-S design model on (6), the membership functions obtained as-

$$M_1 = \frac{z_1}{ed_1}, M_2 = \frac{ed_1 - z_1}{ed_1}, N_1 = \frac{z_2}{md_2}, N_2 = \frac{md_2 - z_2}{md_2}, K_1 = \frac{z_3}{\frac{nd_3}{A+d_1}}, K_2 = \frac{\frac{nd_3}{A+d_1} - z_3}{\frac{nd_3}{A+d_1}}, P_1 = \frac{z_4}{\beta d_3}, P_2 = \frac{\beta d_3 - z_4}{\beta d_3}$$

since we have 16 rules, the matrices $A_1, A_2, A_3, \ldots, A_{16}$are obtained as:

$$A_1 = \begin{bmatrix} r & 0 & 0 \\ 0 & (B - D) & 0 \\ 0 & 0 & -\gamma \end{bmatrix}, A_2 = \begin{bmatrix} r - ed_1 & 0 & 0 \\ 0 & (B - D) & 0 \\ 0 & 0 & -\gamma \end{bmatrix}, A_3 = \begin{bmatrix} r - md_2 & 0 & 0 \\ f_1 md_2 & (B - D) & 0 \\ 0 & 0 & -\gamma \end{bmatrix},$$

$$A_4=\begin{bmatrix} r-\frac{nd_3}{A+d_1} & 0 & 0 \\ 0 & (B-D) & 0 \\ \frac{f_2nd_3}{A+d_1} & 0 & -\gamma \end{bmatrix}, A_5=\begin{bmatrix} r & 0 & 0 \\ 0 & -\beta d_3+(B-D) & 0 \\ 0 & \beta d_3 & -\gamma \end{bmatrix},$$

$$A_6=\begin{bmatrix} r-ed_1-md_2 & 0 & 0 \\ f_1md_2 & (B-D) & 0 \\ 0 & 0 & -\gamma \end{bmatrix}, A_7=\begin{bmatrix} r-ed_1-\frac{nd_3}{A+d_1} & 0 & 0 \\ 0 & (B-D) & 0 \\ \frac{f_2nd_3}{A+d_1} & 0 & -\gamma \end{bmatrix},$$

$$A_8=\begin{bmatrix} r-ed_1 & 0 & 0 \\ 0 & -\beta d_3+(B-D) & 0 \\ 0 & \beta d_3 & -\gamma \end{bmatrix}, A_9=\begin{bmatrix} r-md_2-\frac{nd_3}{A+d_1} & 0 & 0 \\ f_1md_2 & (B-D) & 0 \\ \frac{f_2nd_3}{A+d_1} & 0 & -\gamma \end{bmatrix},$$

$$A_{10}=\begin{bmatrix} r-md_2 & 0 & 0 \\ f_1md_2 & -\beta d_3+(B-D) & 0 \\ 0 & \beta d_3 & -\gamma \end{bmatrix}, A_{11}=\begin{bmatrix} r-\frac{nd_3}{A+d_1} & 0 & 0 \\ 0 & -\beta d_3+(B-D) & 0 \\ \frac{f_2nd_3}{A+d_1} & \beta d_3 & -\gamma \end{bmatrix},$$

$$A_{12}=\begin{bmatrix} r-ed_1-md_2-\frac{nd_3}{A+d_1} & 0 & 0 \\ f_1md_2 & (B-D) & 0 \\ \frac{f_2nd_3}{A+d_1} & 0 & -\gamma \end{bmatrix}, A_{13}=\begin{bmatrix} r-ed_1-md_2 & 0 & 0 \\ f_1md_2 & -\beta d_3+(B-D) & 0 \\ 0 & \beta d_3 & -\gamma \end{bmatrix},$$

$$A_{14}=\begin{bmatrix} r-ed_1-\frac{nd_3}{A+d_1} & 0 & 0 \\ 0 & -\beta d_3+(B-D) & 0 \\ \frac{f_2nd_3}{A+d_1} & \beta d_3 & -\gamma \end{bmatrix}, A_{15}=\begin{bmatrix} r-md_2-\frac{nd_3}{A+d_1} & 0 & 0 \\ f_1md_2 & -\beta d_3+(B-D) & 0 \\ \frac{f_2nd_3}{A+d_1} & \beta d_3 & -\gamma \end{bmatrix},$$

$$A_{16}=\begin{bmatrix} r-ed_1-md_2-\frac{nd_3}{A+d_1} & 0 & 0 \\ f_1md_2 & -\beta d_3+(B-D) & 0 \\ \frac{f_2nd_3}{A+d_1} & \beta d_3 & -\gamma \end{bmatrix}$$

Therefore, Deffuzification is given as

$\dot{x}(t)=\sum_{i=1}^{r} h_i\big(z(t)\big)A_i(x(t))$ (15)

where

$h_1=M_1*N_1*K_1*P_1$, $h_2=M_2*N_1*K_1*P_1$, $h_3=M_1*N_2*K_1*P_1$, $h_4=M_1*N_1*K_2*P_1$,
$h_5=M_1*N_1*K_1*P_2$, $h_6=M_2*N_2*K_1*P_1$, $h_7=M_2*N_1*K_2*P_1$, $h_8=M_2*N_1*K_1*P_2$,
$h_9=M_1*N_2*K_2*P_1$, $h_{10}=M_1*N_2*K_1*P_2$, $h_{11}=M_1*N_1*K_2*P_2$,
$h_{12}=M_2*N_2*K_2*P_1$, $h_{13}=M_2*N_1*K_2*P_2$, $h_{14}=M_1*N_2*K_2*P_2$,
$h_{15}=M_2*N_2*K_1*P_2$, $h_{16}=M_2*N_2*K_2*P_2$

This Fuzzy model is a suitable representation of the non-linear system (6) in the region $[0,10]\times[0,10]\times[0,10]$.

6 Results and Discussion

In this section, the global stability of the considered intra-specific competition predator-prey model (6) is discussed. Because biological systems are complicated, nonlinear, and unpredictable, fuzzy logical methods

with linguistic descriptions should be used to represent them.

Table 1 Stability of the system at various parameters.

r	m	n	A	f_1	f_2	β	$(B-D)$	γ	e	d_1	d_2	d_3	$\max(\lambda_i)=\lambda(\alpha)$	$\ln(\varepsilon\beta_j)+\lambda(\alpha)\delta_j$	Conclu-sion
0.5	0.31	0.25	0.5	0.31	0.18	0.25	0.31	0.12	0.0005	10	10	10	0.87	-1.41	stable
0.1	0.5	0.5	0.5	0.2	0.2	0.5	0.45	0.2	25	30	30	30	0.95	-1.408	stable
3.8	0.5	2	0.3	1.25	1.5	0.15	2	0.3	3	20	20	20	50.34	-0.4202	stable
2.5	0.25	0.25	0.25	1.75	2.27	0.5	6	0.3	15	25	25	25	73.74	0.0478	unstable

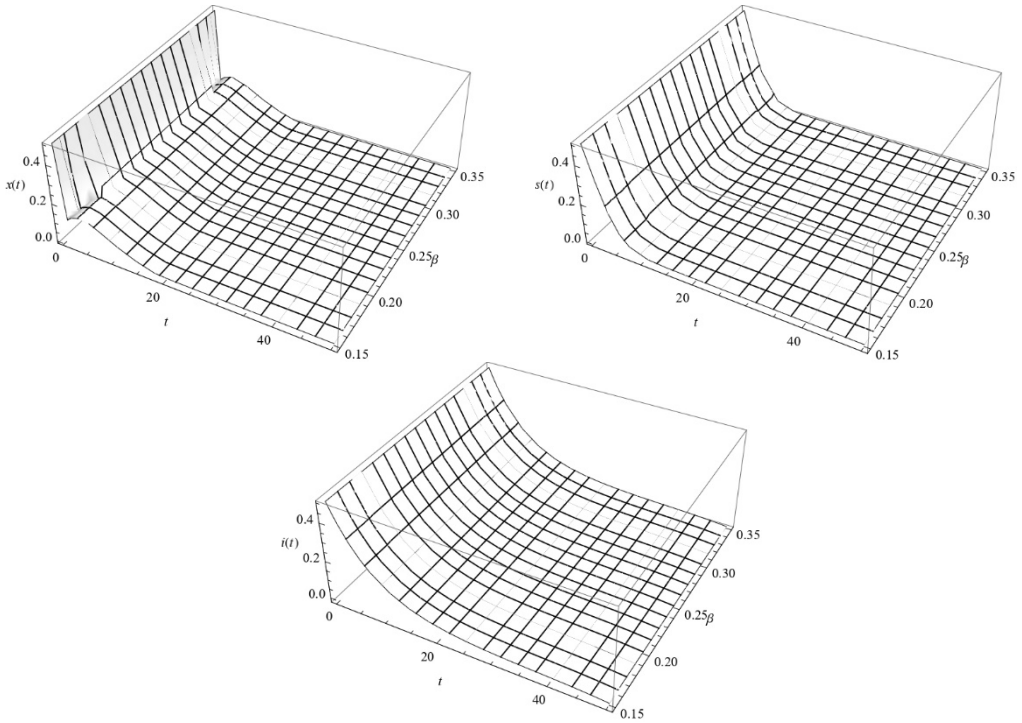


Fig. 1 Effect of infection transmission rate (β) on prey-predator system under impulsive control.

Calculations were carried by taking the values of the parameters at $r = 0.5,\ m = 0.31,\ n = 0.25,\ A = 0.5,\ f_1 = 0.31,\ f_2 = 0.18,\ (B-D) = 0.31,\ \beta = 0.25,\ \gamma = 0.12,\ e = 0.0005,\ d_1 = 10,\ d_2 = 10,\ d_3 = 10$ in 7 to get the eigen values of $[A_i^T + A_i](i = 1,2,3 \ldots r)$as explained in the theorems (4.1, 4.2). It is found that $\max(\lambda_i) = \lambda(\alpha) = 0.87$ then we have chosen $diag[-0.84, -0.84]$ as impulsive control matrix, such that$\beta_j = I + K = 0.16$. It is noted that the system 7 is stable globally (4.1) when $\varepsilon = 1.5, \delta = 0.02$ (at thoseabove values, $\ln(\varepsilon\beta_j) + \lambda(\alpha)\delta_j = -1.41<0$). Further, it is noted that the predator-preymodel is unstable (4.1) when $r = 2.5,\ m = 0.25,\ n = 0.25,\ A = 0.25,\ f_1 = 1.75,\ f_2 = 2.27, (B-D) = 6,\ \beta = 0.5,\ \gamma = 0.3,\ e = 15,\ d_1 = 25,\ d_2 = 25,\ d_3 = 25$, since $\max(\lambda_i) = \lambda(\alpha) = 73.74$, $\rightarrow \ln(\varepsilon\beta_j) + \lambda(\alpha)\delta_j = 0.0478 > 0$. Table. 1 presents the stability of the system at various values of the present study.

The impact of the various parameters on prey-predator system (3-5) with T-S fuzzy impulsive control model is presented in Figs. 1 - 10.

The effect of infection parameter (β)on prey-predator system is shown in Fig.1 at $r = 0.5,\ m = 0.31,\ n = 0.25,\ A = 0.5,\ f_1 = 0.31,\ f_2 = 0.18,\ (B-D) = 0.31,\ \gamma = 0.12,\ e = 0.0005,\ d_1 = 10,\ d_2 = 10,\ d_3 = 10$. This graph makes it abundantly evident that asinfection rates rise, so do the populations of

infected predators.

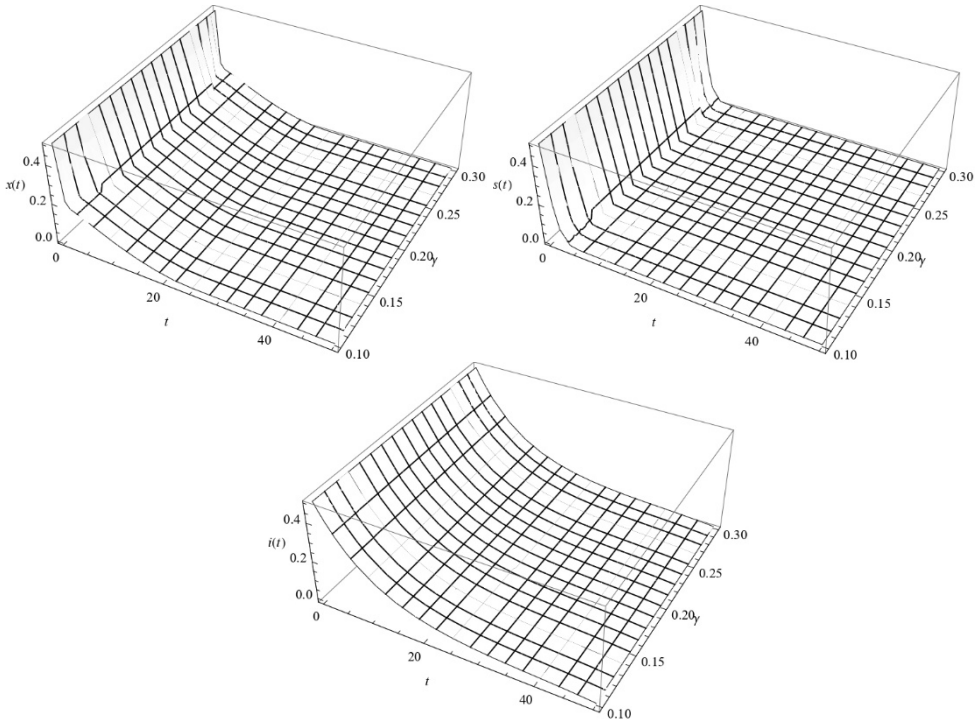
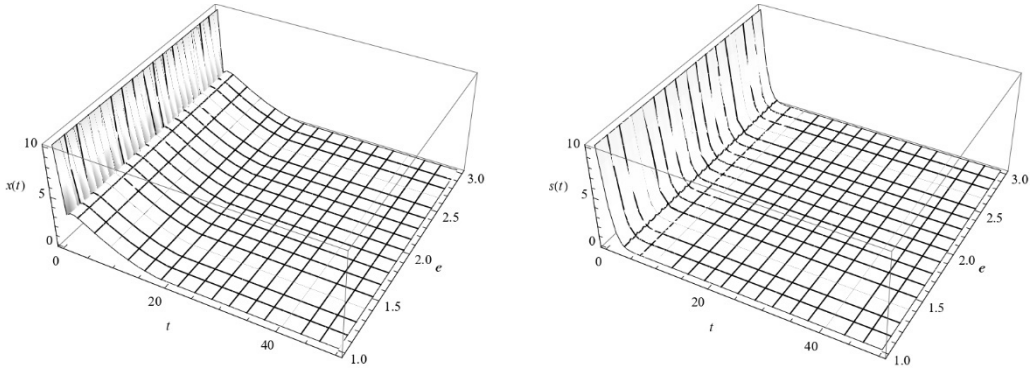


Fig. 2 Effect of disease mortality (γ) on prey-predator system under impulsive control.

The influence of disease mortality γ on prey-predator system is shown in Fig. 2 at $r = 0.5,\ m = 0.31,\ n = 0.25,\ A = 0.5,\ f_1 = 0.31,\ f_2 = 0.18,\ (B - D) = 0.31,\ \beta = 0.25,\ e = 0.0005,\ d_1 = 10,\ d_2 = 10,\ d_3 = 10$. This figure clearly exhibits that an increase in disease mortality leads increase in susceptible predator population decreases slowly whereas the infected predator population decreases faster.

The consequences of intra-specific competition (e) on prey-predator system is shown in Fig. 3 at $r = 0.5,\ m = 0.31,\ n = 0.25,\ A = 0.5,\ f_1 = 0.31,\ f_2 = 0.18,\ (B - D) = 0.31,\ \beta = 0.25,\ \gamma = 0.12,\ d_1 = 10,\ d_2 = 10,\ d_3 = 10$. This graph demonstrates how less intraspecificcompetition between prey and predator results in a rise in the population of prey.

The change on prey-predator system with growth rate of prey (r) is shown in Fig. 4 at $m = 0.31,\ n = 0.25,\ A = 0.5,\ f_1 = 0.31,\ f_2 = 0.18,\ (B - D) = 0.31,\ \beta = 0.25,\ \gamma = 0.12,\ e = 0.0005,\ d_1 = 10,\ d_2 = 10,\ d_3 = 10$. This figure clearly exhibits that increase in growth rate of prey leads to increase in the prey population and the population of the predator become stable.



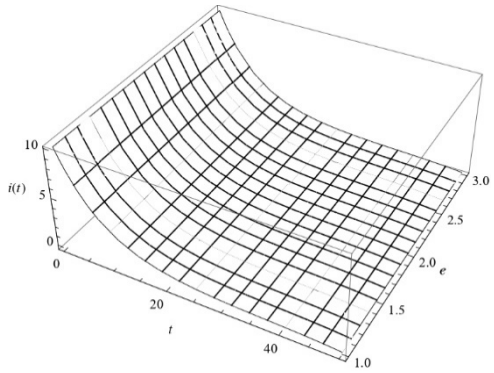


Fig. 3 Effect of inter-specific competition (e) on prey-predator system under impulsive control.

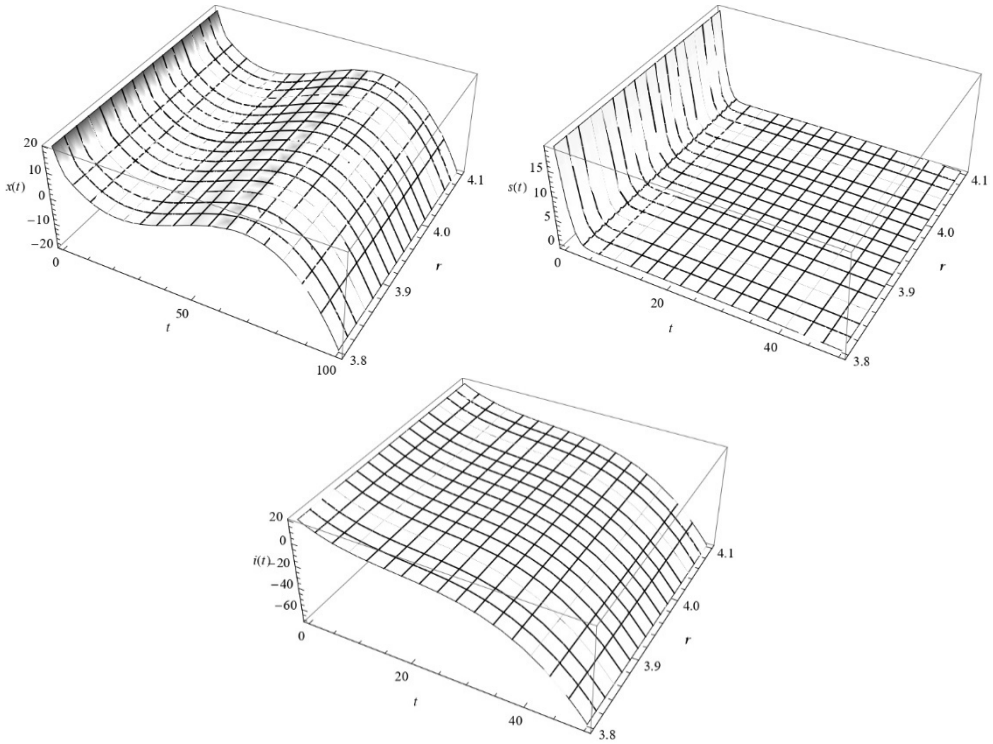
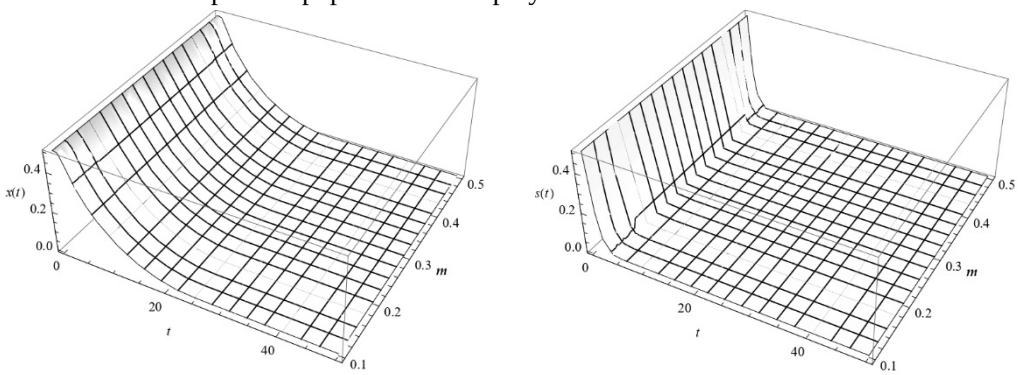


Fig. 4 Effect of growth rate of prey (r) on prey-predator system under impulsive control.

The outcome with varying predation rate of susceptible predator (m) on prey-predator system is shown in Fig. 5 at $r = 0.5,\ n = 0.25,\ A = 0.5,\ f_1 = 0.31,\ f_2 = 0.18,\ (B - D) = 0.31,\ \beta = 0.25,\ \gamma = 0.12,\ e = 0.0005,\ d_1 = 10,\ d_2 = 10,\ d_3 = 10$. This graph demonstrates unambiguously how an increase in a predator's predation rate causes a drop in the population of its prey.



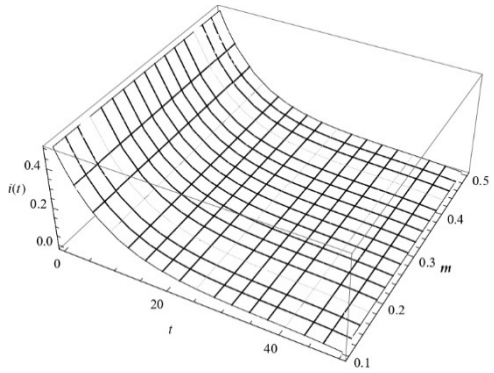


Fig. 5 Effect of predation rate of susceptible predator (m) on prey-predator system under impulsive control.

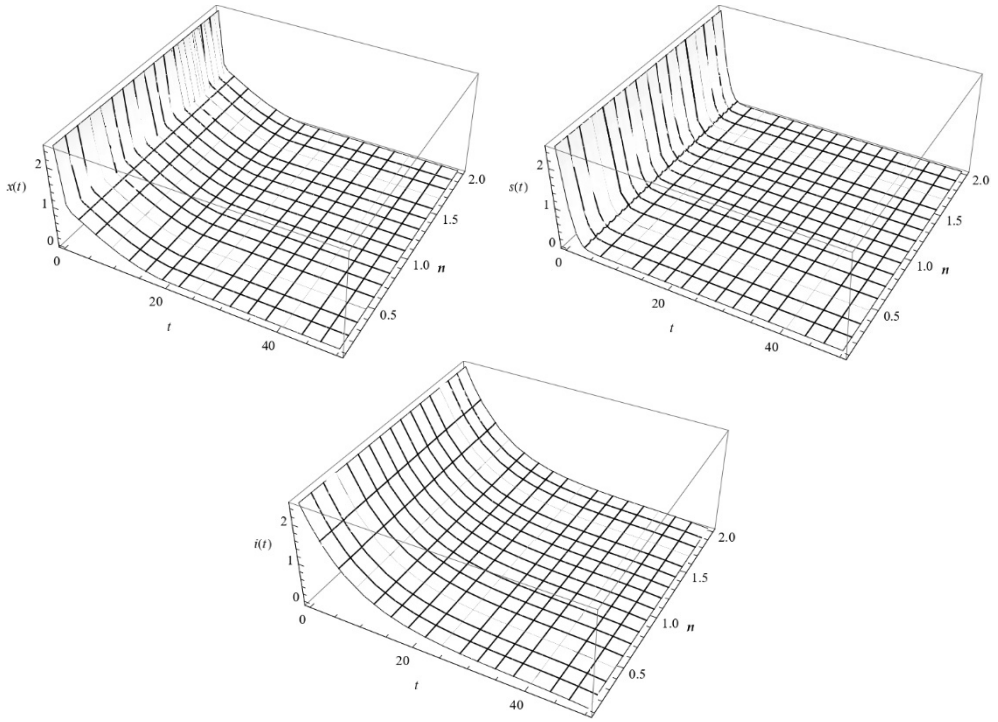
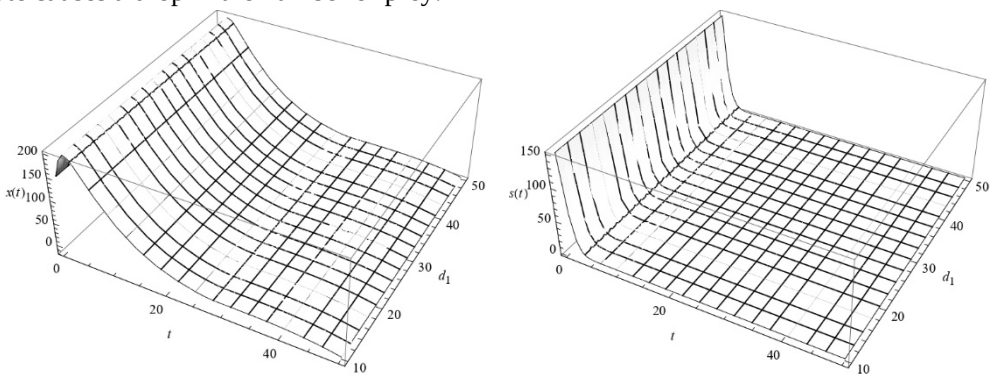


Fig. 6 Effect of predation rate of infected predator (n) on prey-predator system under impulsive control.

The impact of predation rate of infected predator (n) on prey-predator system is shown in Fig. 6 at $r = 0.5,\ m = 0.31,\ A = 0.5,\ f_1 = 0.31,\ f_2 = 0.18,\ (B - D) = 0.31,\ \beta = 0.25,\ \gamma = 0.12,\ e = 0.0005,\ d_1 = 10,\ d_2 = 10,\ d_3 = 10$. This graph illustrate clearly how an infected predator's increased predation rate causes a drop in the number of prey.



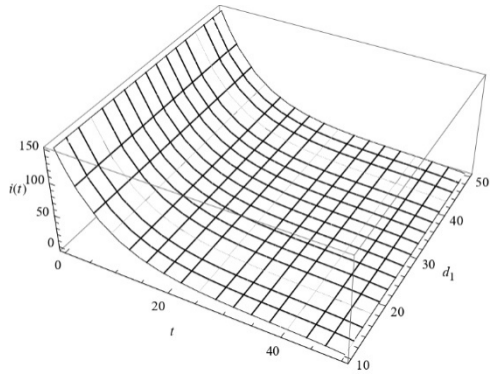


Fig. 7 Effect of max time (d_1) on prey-predator system under impulsive control.

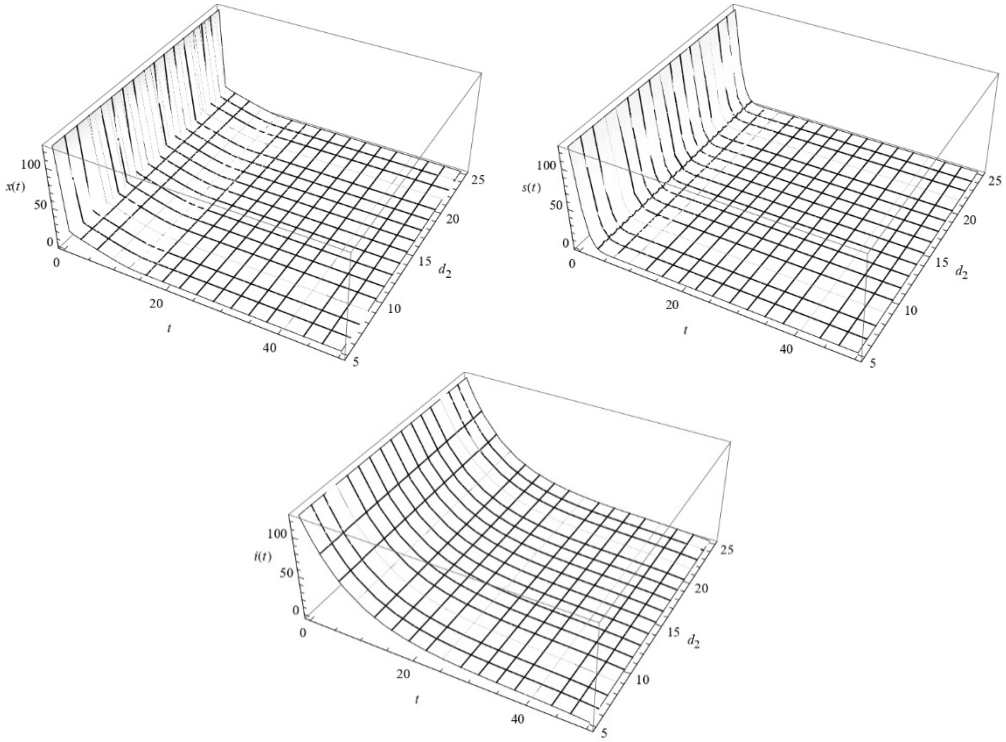


Fig. 8 Effect of max time (d_2) on prey-predator system under impulsive control.

The dynamical change on prey- predator population (x, s, i) by varying prey maxtime (d_1) parameter under fuzzy impulsive control can be noted in Fig. 7 at $r = 0.5,\ m = 0.31,\ n = 0.25,\ A = 0.5,\ f_1 = 0.31,\ f_2 = 0.18,\ (B - D) = 0.31,\ \beta = 0.25,\ \gamma = 0.12,\ e = 0.0005,\ d_2 = 10,\ d_3 = 10$. It is noticed from this figure that; the prey population increases as $d_1$decreases.

The effectiveness by varying susceptible predator max time (d_2) parameter of prey-predatorpopulation (x, s, i) under fuzzy impulsive control can be noted in Fig. 8 at $r = 0.5,\ m = 0.31,\ n = 0.25,\ A = 0.5,\ f_1 = 0.31,\ f_2 = 0.18,\ (B - D) = 0.31,\ \beta = 0.25,\ \gamma = 0.12,\ e = 0.0005,\ d_1 = 10,\ d_3 = 10$. It is noted from this figure that, the prey population increases as $d_2$decreases.

The vital pattern of prey- predator population $(x, s, i)$by varying infected predatormax time (d_3)parameter under fuzzy impulsive control can be noted in Fig. 9 at $r = 0.5,\ m = 0.31,\ n = 0.25,\ A = 0.5,\ f_1 = 0.31,\ f_2 = 0.18,\ (B - D) = 0.31,\ \beta = 0.25,\ \gamma = 0.12,\ e = 0.0005,\ d_1 = 10,\ d_2 = 10$. It is observed from this figure that, the prey population increases, and predator population decreases with an increase in (d_3).

Finally, the nature of two species (x, s, i)population (without impulsive control) is presented in Fig. 10 by fixing all the parameters obtained from T-S fuzzy model at $r = 0.5,\ m = 0.31,\ n = 0.25,\ A = 0.5,\ f_1 =$

$0.31,\ f_2 = 0.18,\ (B - D) = 0.31,\ \beta = 0.25,\ \gamma = 0.12,\ e = 0.0005,\ d_1 = 10,\ d_2 = 10,\ d_3 = 10$ and initial condition $x(0) = 5, s(0) = 5, i(0) = 5, t = 5$. The figure clearly shows how the prey- predator population reaches to stability whereas infected predator becomes unstable.

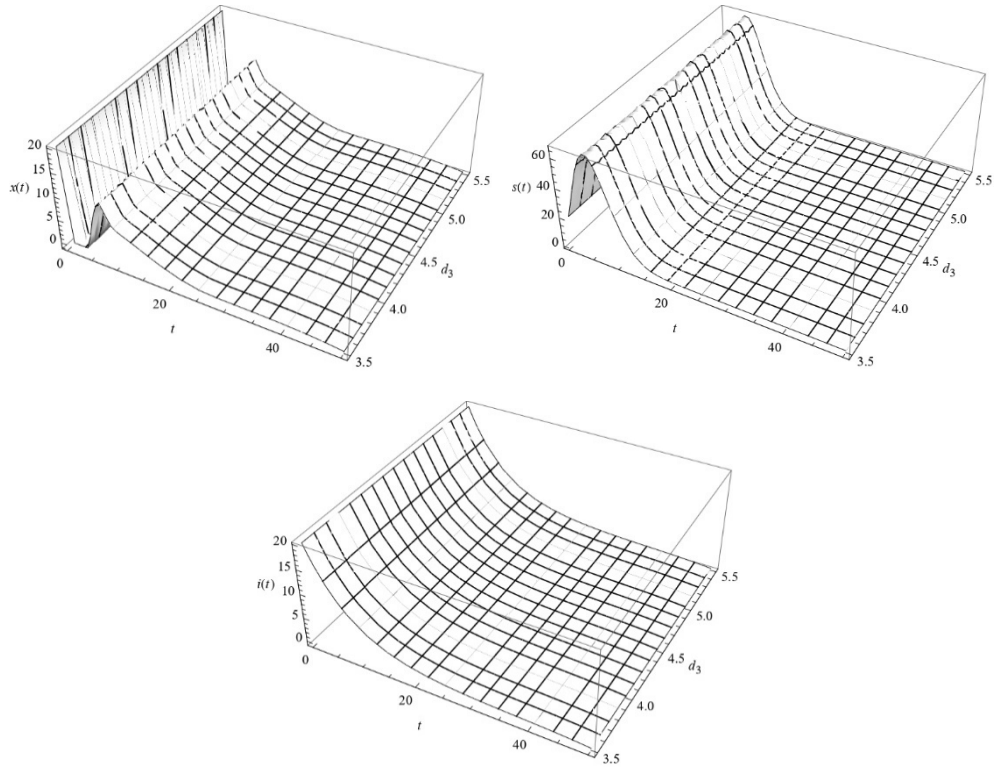


Fig. 9 Effect of max time (d_3) on prey-predator system under impulsive control.

7 Conclusions

In this paper we present stability analysis of a three species competition model with fuzzy impulsive control by T-S model. Firstly, a non-linear Lotka-Volterra predator-prey model with infection in predator based on the fuzzy impulsive control was analyzed. The impulsive control technique which was analyzed in the framework of the fuzzy systems based on T-S model, is found appropriate for very complex and non-linear system with impulsive effects. Then, the complete impulsive fuzzy system is obtained by combining each local linear impulsive system. Meantime, the asymptotic stability, and exponential stability of the impulsive fuzzy system are shown by various stability theorems. Finally, numerical examples for prey, susceptible predator and infected predator system with impulsive effects are given to illustrate the application of impulsive fuzzy control, and simulation results show the effectiveness of the proposed method. From this present study the main findings are listed as:

- We establish a predator–prey model in which predator population is infected.
- Less intra-specific competition between prey and predator results in a rise in the population of prey because of the infection in predators.
- A rise in the prey population results from an increase in the prey growth rate.
- The population of healthy predators decreases as the rate of disease transmission from diseased to susceptible predators rises because more predators will contract the disease.
- As the maximum period for prey diminishes, the prey population grows.
- Because predators will have less time for predation as the maximum period forsusceptible predators gets shorter, the number of preys rises.

- While the population of prey increases as the maximum time for an infected predator increase, the population of susceptible predators decreases because more predators will contract the infection.

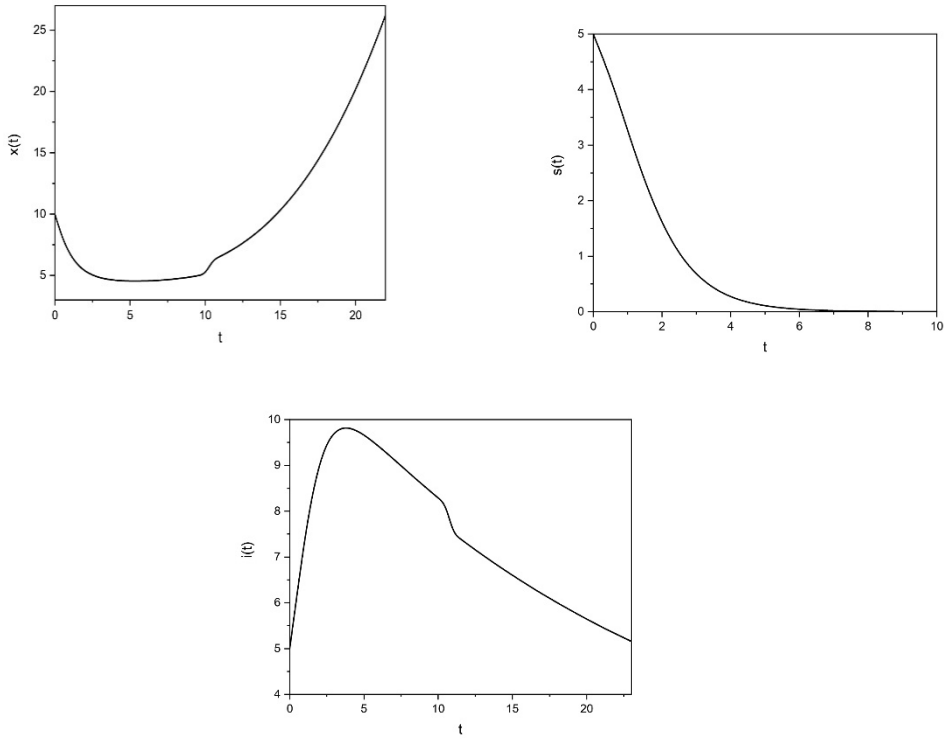


Fig. 10 Plot of predator-prey system without impulsive control.

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References

Anderson RM, May RM. 1986. The invasion, persistence and spread of infectious diseases within animal and plant communities. Philosophical transactions of the Royal Society of London. Series B, Biological Sciences, 314(1167): 533-570

Baek H. 2010. Dynamic complexities of a three-species Beddington-DeAngelis system with impulsive control strategy. Acta Applicandae Mathematicae, 110(1): 23-38

Elisa E, Grammauro M, Venturino E. 2013. Predator's alternative food sources do not support ecoepidemics with two strains-diseased prey. Network Biology, 3(1): 29-44

Ferrarini A. 2015. Evolutionary network control also holds for nonlinear networks: Ruling the Lotka-Volterra model. Network Biology, 5(1): 34-42

Greenhalgh D, Haque M. 2007. A predator-prey model with disease in the prey species only. Mathematical Methods in the Applied Sciences, 30(8): 911-29

Haque M. 2009. Ratio-dependent predator-prey models of interacting populations. Bulletin of Mathematical Biology, 71(2): 430-52

Haque M. 2011. A detailed study of the Beddington-DeAngelis predator-prey model. Mathematical

Biosciences, 234(1): 1-16

Haque M, Venturino E. 2006. The role of transmissible diseases in the Holling-Tanner predator-prey model. Theoretical Population Biology, 70(3): 273-288

Hu D, Zhang Y, Zheng Z, Liu M. 2022. Dynamics of a delayed predator-prey model with constant-yield prey harvesting. Journal of Applied Analysis and Computation, 12(1): 302-335

Kumari N, Mohan N. 2021. Coexistence states of a ratio-dependent predator-prey model with nonlinear diffusion. Acta Applicandae Mathematicae, 176(1): 11

Kermack WO, McKendrick AG. 1927. A contribution to the mathematical theory of epidemics. Proceedings of the Royal Society of London. Series A, Containing papers of a mathematical and physical character, 115(772): 700-721

Li YM, Zhu Y. 2009. The control and the reconfigurable control for prey-predator ecosystem with time delay. Applied Mathematical Modelling, 33(1): 148-160

Lin, YH, Chang WJ, Ku CC. 2022. Solving the formation and containment control problem of nonlinear multi-boiler systems based on interval type-2 Takagi–Sugeno fuzzy models. Processes, 10(6)

Liu R, Liu G. 2020. Dynamics of a stochastic three species prey-predator model with intraguild predation. Journal of Applied Analysis and Computation, 10(1): 81-103

Maiti A, Jana MM, Samanta GP. 2007. Deterministic and stochastic analysis of a ratio-dependent predator-prey system with delay. Nonlinear Analysis: Modelling and Control, 12(3): 383-398

Nandadulal B, Bhattacharya S, Auger P, Sarkar B. 2021. Bioeconomics fishery model in presence of infection: Sustainability and demand-price perspectives. Applied Mathematics and Computation, 405: 126225

Shakil M. 2015. The modeling of predator-prey interactions. Network Biology, 5(2): 71-81

Tanaka K, Hori T, Wang HO. 2001. A fuzzy Lyapunov approach to fuzzy control system design. Proceedings of the American Control Conference, 6: 4790-4795

Tseng CS, Chen BS, Uang HJ. 2001. Fuzzy tracking control design for nonlinear dynamic systems via T-S fuzzy model. IEEE Transactions on Fuzzy Systems, 9(3): 381-392

Volterra V. 1926. Variazioni e Fluttuazioni Del Numero d'individui in Specie Animali Conviventi. Memoria della regia accademia nazionale del lincei ser, 62: 31-113

Wang F, Kuang Y, Ding C, Zhang S. 2013. Stability and bifurcation of a stage-structured predator–prey model with both discrete and distributed delays. Chaos, Solitons and Fractals, 46: 19-27

Wang X. 2015. Robust stability of stochastic fuzzy delayed neural networks with impulsive time window. Neural Networks, 67: 84-91

Wang Y, Yu H, Zhang X, Li D. 2012. Stability analysis and design of time-varying nonlinear systems based on impulsive fuzzy model. Discrete Dynamics in Nature and Society, 2012

Zhang WJ, Liu GH. 2023. A dynamic model to simulate the development of insecticide resistance . Computational Ecology and Software, 13(3): 52-70

Zhao Y, Zhang WJ. 2013. Organizational theory: With its applications in biology and ecology. Network Biology, 3(1): 45–53