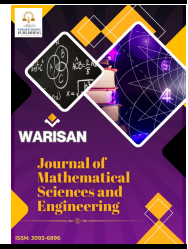




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Mathematical Study for The Dynamical Behavior of Toxoplasmosis Disease in A Population

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ABSTRACT

Mathematical modelling provides meaningful insight into infectious disease dynamics. Toxoplasmosis is a parasitic disease caused by *Toxoplasma gondii* which is present in cat's contamination, uncooked meat, polluted soil, water etc. which is risky for the people with weak immune system especially risky for the pregnant women. Due to its silent nature and persistency, it causes serious public health issues as well as in animals. The problems addressed in given study are the comprehensive understanding about the dynamics of the disease within animal by interaction with infected population and also contaminated environment. The aim of this research is to investigate the dynamical behavior of the toxoplasmosis disease in a cat population by mathematically using the compartmental model. The model is the extension of classical SIR framework introducing the compartments vaccinated, susceptible, seriously infected, mild infected and environmental contamination. This model uses various parameters which biologically reflect many rates like vaccination rate, immunity loss rate, oocysts shedding rate etc. By analyzing model mathematically, equilibrium points are derived which further introduced the threshold, basic reproductive number which tells about the behavior of disease either it will spread, persist or die out from population. With the help of analytical methods, stability analysis of the model is performed. Additionally, for the reliability of the results the different numerical schemes are tested using different initial conditions and step sizes for positivity, boundedness and dynamical consistency. Mathematical foundation is presented by the given model for comprehend the most influencing factors on the toxoplasmosis disease transmission. To check whether there is endemic or pandemic case, threshold conditions are identified and impact of each parameter on the system dynamics is observed. Detailed conclusions are drawn for the sensitivity analysis. In conclusion many results are drawn concerning vaccination as control strategies to completely die out the disease from the population.

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1. Introduction

Toxoplasmosis is a disease which is caused by a protozoan parasite, *Toxoplasma gondii*. It affects millions of people worldwide [1]. This disease is especially risky for the immunocompromised people and pregnant women. This disease has been increased risk of Schizophrenia, depression and behavioral changes. Some Researches indicate that infected individuals may take more risks, such as reckless driving. This parasite is mostly found in the infected animals and in the contaminated food and water. *Toxoplasma gondii* cysts typically have a diameter of 5–50 μm and it has crescent-shaped. In the brain, cysts are usually spherical, but in the heart and skeletal muscles, they are longer. Although they can be located in many different parts of the host's body, the brain, skeletal muscles, and heart muscles are where they are most frequently discovered [2]. Toxoplasmosis was firstly identified in the animals and then identified in the humans. In 1908, Nicolle and Manceaux made the initial discovery of *Toxoplasma gondii* in Tunisia while researching the *gundii* (*Ctenodactylus gundii*), a rodent species. In the same year, Splendore independently discovered the parasite in a rabbit in Brazil. A newly discovered protozoan species was identified as a result of these findings, and it was subsequently given the name *Toxoplasma gondii* because of its crescent-shaped (or "toxos" in Greek) form. Janku reported the first human case of toxoplasmosis in 1939, involving a newborn in Prague, Czech Republic. The baby showed symptoms such as calcium deposits in the brain and an enlarged head (hydrocephalus). Doctors found *Toxoplasma gondii* in the brain during examination, connecting the parasite to human congenital illness.

Toxoplasmosis spreads by consuming of cysts in uncooked meat of cattle, pigs or sheep, coming into contact with cat feces, transmission from an infected mother to the unborn child, extremely rare by contaminated blood transfusion (and organ transplant) etc. [3]. The majority of toxoplasmosis patients with healthy immune systems do not exhibit any symptoms. Flu-like symptoms, swollen lymph nodes, and muscle aches and pains are possible for those who are challenged. Toxoplasmosis damages the brain, eyes, and other organs [4]. According to WHO, toxoplasmosis is a dangerous disease for public health. It is especially harmful for pregnant women and who eat contaminated food. This can lead to serious health issues like brain damage, abortion etc. Basically, WHO estimated approximately 190,000 cases over the world but most of the cases were report in South America, and some Middle Eastern countries [5,6].

Mathematical modelling plays an essential role in epidemiology. The understanding of infectious disease dynamics and control methods depends heavily on mathematical modeling according to various research studies [7]. In epidemiology, mathematical modelling not only focus on the theoretical but also on the practical and essential tools which are helpful in saving life. Mathematical modelling solves the problem in case of infectious disease in systematic ways and to understand the dynamics of disease provide the quantitative analysis [8]. It helps in understanding disease dynamics, predicting future outbreaks, estimation of control strategies, in policy making, interdisciplinary insights and in many others ways which in return are helpful for researchers, public health officials and policy makers to control the disease. Without a doubt, the most well-known mathematical model for the spread of an infectious disease is the SIR model. The SIR model, created in 1927 by Kermack and McKendrick, categorizes a population into three groups: susceptible, infectious, and recovered. According to Kermack and McKendrick that model is essential for understanding how infectious diseases spread among communities. A useful framework for examining and forecasting how diseases spread within populations is provided by compartmental deterministic models, which are widely used to model the transmission of infectious diseases.

Symptoms of epidemic disease model by Cooke incorporated a discrete time delay symbolized by βSI and a bilinear incidence rate. The authors developed this model to study the transmission

patterns of infections that result from rat and cat populations [9]. The time delay feature indicates the amount of time required for infectious organisms to mature inside the vector until they can infect human beings [10]. The idea of time delay in epidemic modeling gained important biological significance according to past studies. The SIR epidemic model functions as an essential instrument to analyze and handle epidemic diseases. Public health professionals require this well-structured method to analyze disease transmission because it helps build effective measures which reduce epidemic related social harm. The SIR model stands as a vital instrument which epidemiologists and public health officials use to predict epidemic progress for developing strategic resource allocation and control measure implementations [11] while keeping in mind previous disease experiences.

In first section, literature and background of disease is discussed. Second section discusses the model formulation in which equilibrium points, stability and basic reproduction number are described. Numerical schemes Euler, RK_4 and NSFD are discussed in section three and section four is about impact of parameters on state variables and basic reproductive number. Sections five, six and seven are about sensitivity analysis, graphical representation and conclusion respectively.

2. Model Formulation

We will analyze the toxoplasmosis disease mathematically and find the different results using equilibrium points for stability analysis, numerical calculations, and simulations. Here is the research article [12] which is written by A. A. AYOADE, T. OYEDEPO, and S. AGUNBIADÉ. In this paper, the authors focus on the population of cats. The authors explain the transmission dynamics between the environment and the population of cats and used vaccination and sanitation as control strategies. They made the five compartments one for the environment and four for the cat population using the SIR frame work. The paper gives the conditions and results for disease-free equilibrium and for the endemic case.

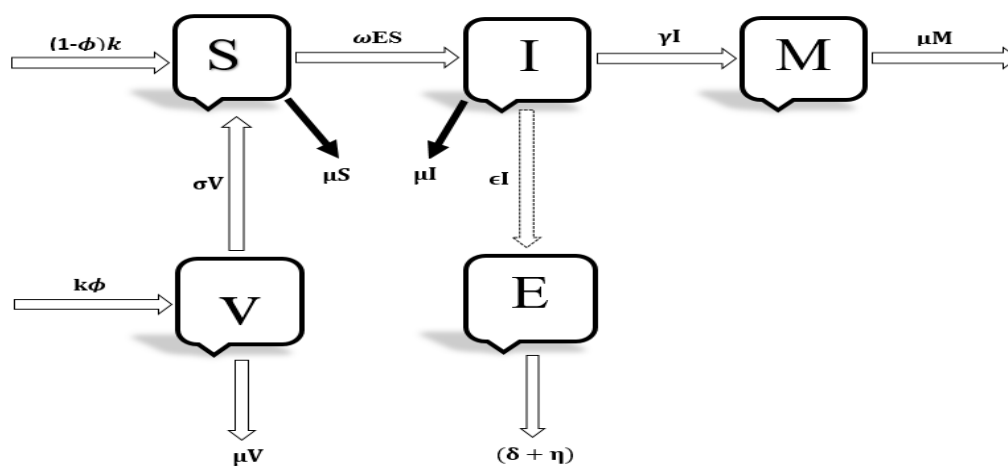


Fig. 1. Flow chart of model

Here, the model is divided into 5 compartments and these are $(S(t))$ for susceptible; $(V(t))$ for vaccinated; $(I(t))$ for seriously infected and $(M(t))$ for mildly infected. In the vaccinated compartment, $k\phi$ shows the number of cats that are temporarily immune, where μ shows natural death, and σ is the rate of those cats or oocysts that have lost immunity. In the susceptible compartment $(1-\phi)k$ is the recruitment rate. The ω is the rate at which susceptible are moving in the seriously infected compartment where μ is the rate of natural death. Here γ is the rate at which seriously infected are

moving in mildly infected compartment. ϵ is the rate at which the oocysts move from the seriously infected compartment to the environment.

2.1 Mathematical Model

The transmission of toxoplasmosis disease in cat population is given in the ODE(differential) form is:

$$\frac{dV}{dt} = \varphi k - (\sigma + \mu)V \quad (1)$$

$$\frac{dS}{dt} = (1 - \varphi)k + \sigma V - \omega ES - \mu S \quad (2)$$

$$\frac{dI}{dt} = \omega ES - (\gamma + \mu)I \quad (3)$$

$$\frac{dM}{dt} = \gamma I - \mu M \quad (4)$$

$$\frac{dE}{dt} = \epsilon I - (\delta + \eta)E \quad (5)$$

s.t initial conditions are

$$V(0) = V_o, S(0) = S_o, I(0) = I_o, M(0) = M_o, E(0) = E_o$$

The assumptions are as follows:

1. Mildly infected cats do not interfere with the environment.
2. Cats in the vaccinated compartment do not interact with the contaminated environment.

2.2 Equilibrium Points

Now we find the equilibrium points, so the derivative goes to zero.

$$V = \frac{\varphi k}{\sigma + \mu} \quad (A)$$

$$S = \frac{(1 - \varphi)k + \sigma(\frac{\varphi k}{\sigma + \mu})}{\omega E + \mu} \quad (B)$$

$$I = \frac{\omega ES}{\gamma + \mu} \quad (C)$$

$$M = \frac{\gamma I}{\mu} \quad (D)$$

$$E = \frac{\epsilon I}{\delta + \eta} \quad (E)$$

2.3 Disease Free Equilibrium

DFE is case when there is no disease in a population. All population is susceptible and vaccinated.

$$\therefore I = \frac{\epsilon I}{\delta + \eta}$$

$$I \left(1 - \frac{\omega \epsilon S}{(\delta + \eta)(\gamma + \mu)} \right) = 0$$

Here $I = 0$ is showing that there are no seriously infected cats so it is DFE.

$$S^o = \frac{(1 - \varphi)k(\sigma + \mu) + \varphi \sigma k}{(\sigma + \mu)\mu}$$

$$V^o = \frac{\varphi k}{\sigma + \mu}, I^o = 0, \therefore E = \frac{\epsilon I}{\delta + \eta}, E^o = 0$$

$$W_o = (S^o, V^o, I^o, E^o) = \left(\frac{(1 - \varphi)k(\sigma + \mu) + \varphi \sigma k}{(\sigma + \mu)\mu}, \frac{\varphi k}{\sigma + \mu}, 0, 0 \right)$$

2.4 Stability Analysis of Equilibrium Points

Consider the following equations and taking partial derivatives with respect to all state variables:

$$\begin{aligned}
 f_1 &= \varphi k - (\sigma + \mu)V, & f_2 &= (1 - \varphi)k + \sigma V - \omega ES - \mu S \\
 f_3 &= \omega ES - (\gamma + \mu)I, & f_4 &= \epsilon I - (\delta + \eta)E \\
 \frac{\partial f_1}{\partial V} &= -(\sigma + \mu), \frac{\partial f_1}{\partial S} = \frac{\partial f_1}{\partial I} = \frac{\partial f_1}{\partial E} = 0 \\
 \frac{\partial f_2}{\partial V} &= \sigma, \frac{\partial f_2}{\partial S} = -\mu - \omega E, \frac{\partial f_2}{\partial I} = 0, \frac{\partial f_2}{\partial E} = -\omega S \\
 \frac{\partial f_3}{\partial V} &= 0, \frac{\partial f_3}{\partial S} = \omega E, \frac{\partial f_3}{\partial I} = -(\gamma + \mu), \frac{\partial f_3}{\partial E} = \omega S \\
 \frac{\partial f_4}{\partial V} &= 0, \frac{\partial f_4}{\partial S} = 0, \frac{\partial f_4}{\partial I} = \epsilon, \frac{\partial f_4}{\partial E} = -(\delta + \eta)
 \end{aligned}$$

So, Jacobian matrix becomes:

$$J = \begin{bmatrix} -(\sigma + \mu) & 0 & 0 & 0 \\ \sigma & -\mu - \omega E & 0 & -\omega S \\ 0 & \omega E & -(\gamma + \mu) & \omega S \\ 0 & 0 & \epsilon & -(\delta + \eta) \end{bmatrix}$$

2.5 Stability for Disease Free Equilibrium

Given:

$$S = S^0, V = V^0, I = 0, E = E^0$$

$$\lambda_1 = -(\sigma + \mu) < 0, \lambda_2 = -\mu < 0$$

$$\lambda_3 = \begin{bmatrix} -(\gamma + \mu) & \omega S^0 \\ \epsilon & -(\delta + \eta) \end{bmatrix}$$

$$\text{Trace} = -(\gamma + \mu + \delta + \eta) < 0, D = (\gamma + \mu)(\delta + \eta) - \omega \epsilon S^0$$

$$(\gamma + \mu)(\delta + \eta) > \omega \epsilon S^0, 1 > \frac{\omega \epsilon S^0}{(\gamma + \mu)(\delta + \eta)}$$

$$R_z = \frac{\omega \epsilon S^0}{(\gamma + \mu)(\delta + \eta)}$$

$$R_z = \frac{\omega \epsilon [(\mu + \gamma)(1 - \varphi)k + \sigma \varphi k]}{\mu(\mu + \sigma)(\gamma + \mu)(\delta + \eta)}$$

$$R_z < 1$$

That was the stability analysis of the DFE. Therefore ,DFE is locally asymptotically stable if $R_z < 1$.

2.6 Endemic Equilibrium Points

$$I = \frac{\omega ES}{\gamma + \mu} \quad (F)$$

$$E = \frac{\epsilon I}{\delta + \eta}, S = \frac{(1 - \varphi)k + \frac{\sigma \varphi k}{\sigma + \mu}}{\omega E + \mu}$$

Putting value of E in S:

$$S = \frac{(1 - \varphi)k + \frac{\sigma \varphi k}{\sigma + \mu}}{\omega \frac{\epsilon I}{\delta + \eta} + \mu} \quad (G)$$

$$I^* = \frac{\omega \epsilon (1 - \varphi)k(\sigma + \mu) + \omega \epsilon (\sigma \varphi k) - \mu(\delta + \eta)(\gamma + \mu)(\sigma + \mu)}{\omega \epsilon (\sigma + \mu)(\gamma + \mu)}$$

$$S^* = \frac{[(1 - \varphi)k(\sigma + \mu) + \sigma \varphi k](\gamma + \mu)(\delta + \eta)}{\omega \epsilon (1 - \varphi)k(\sigma + \mu) + \omega \epsilon \varphi k}$$

$$\therefore E = \frac{\epsilon I}{\delta + \eta}$$

$$E^* = \frac{\epsilon[\epsilon\omega(1-\varphi)k(\sigma+\mu) + \omega\epsilon(\sigma\varphi k) - \mu(\delta+\eta)(\gamma+\mu)(\sigma+\mu)]}{\omega\epsilon(\sigma+\mu)(\gamma+\mu)(\delta+\eta)}$$

$$V^* = \frac{\varphi k}{(\sigma+\mu)}$$

$$W_* = (V^*, S^*, I^*, E^*)$$

2.7 Stability of Endemic Equilibrium

Here we will check the stability of endemic equilibrium points by using Jacobian matrix.

$$J = \begin{bmatrix} -(\sigma+\mu) & 0 & 0 & 0 \\ \sigma & -\mu-\omega E^* & 0 & -\omega S^* \\ 0 & \omega E^* & -(\gamma+\mu) & \omega S^* \\ 0 & 0 & \epsilon & -(\delta+\eta) \end{bmatrix}$$

$$\text{Trace} = -[4\mu + \sigma + \gamma + \delta + \omega E^*] < 0$$

For determinant we have to use the cofactor expansion method.

$$M_{11} = \begin{bmatrix} -\mu-\omega E^* & 0 & -\omega S^* \\ \omega E^* & -(\gamma+\mu) & \omega S^* \\ 0 & \epsilon & -(\delta+\mu) \end{bmatrix}$$

$$\det(J) = -(\sigma+\mu) * \det(M_{11})$$

$$\det(J) = -(\sigma+\mu) * [(-\mu-\omega E^*)[(\gamma+\mu)(\delta+\eta) - \omega\epsilon S^*] - \omega^2\epsilon S^* E^{*2}]$$

$$-(\sigma+\mu) * [(-\mu-\omega E^*)[(\gamma+\mu)(\delta+\eta) - \omega\epsilon S^*] - \omega^2\epsilon S^* E^{*2}] > 0$$

$$(\gamma+\mu)(\delta+\eta) - \omega\epsilon S^* - \frac{\omega^2\epsilon S^* E^{*2}}{(-\mu-\omega E^*)} > 0 \quad (H)$$

$$\therefore S^* = \frac{S^0}{R_z}$$

$$R_z^2 > R_z$$

$$R_z = \frac{\omega\epsilon S^0}{(\gamma+\mu)(\delta+\eta)} \quad , \quad R_z > 1$$

Hence we proved that basic reproductive number is greater than one which is showing that Endemic Equilibrium is locally stable.

2.8 Basic Reproductive Number

Now we are finding basic reproductive number R_z which is the average number of infection caused by individual. We are taking seriously infected (I) and environment (E) compartments because these compartments are responsible for the transmission of infection. So

$$\frac{dI}{dt} = \omega ES - (\gamma+\mu)I$$

$$\frac{dE}{dt} = \epsilon I - (\delta+\eta)E$$

$$\begin{bmatrix} \frac{dI}{dt} \\ \frac{dE}{dt} \end{bmatrix} = \begin{bmatrix} \omega ES^0 \\ \epsilon I \end{bmatrix} - \begin{bmatrix} (\gamma+\mu)I \\ (\delta+\eta)E \end{bmatrix}$$

$$F^* = \begin{bmatrix} \omega ES^0 \\ \epsilon I \end{bmatrix} = \begin{bmatrix} F_1^* \\ F_2^* \end{bmatrix}$$

$$T^* = \begin{bmatrix} (\gamma+\mu)I \\ (\delta+\eta)E \end{bmatrix} = \begin{bmatrix} T_1^* \\ T_2^* \end{bmatrix}$$

Taking partial derivative:

$$F^* = \begin{bmatrix} 0 & \omega S^0 \\ \epsilon & 0 \end{bmatrix}$$

$$T^* = \begin{bmatrix} \gamma + \mu & 0 \\ 0 & \delta + \eta \end{bmatrix}$$

The next generation method is given as:

$$L = F^* * T^{*-}$$

$$L = \begin{bmatrix} 0 & \frac{\omega S^0}{\delta + \eta} \\ \frac{\epsilon I}{\gamma + \mu} & 0 \end{bmatrix}$$

$$\det \left\{ \begin{bmatrix} 0 & \frac{\omega S^0}{\delta + \eta} \\ \frac{\epsilon I}{\gamma + \mu} & 0 \end{bmatrix} - \lambda \begin{bmatrix} 1 & 0 \\ 0 & 1 \end{bmatrix} \right\} = 0$$

$$\lambda = \pm \sqrt{\frac{\omega \epsilon S^0}{(\gamma + \mu)(\delta + \eta)}}$$

$$R_z = \max | \text{Eigenvalues}(F^* * T^{*-}) | = \sqrt{\frac{\omega \epsilon S^0}{(\gamma + \mu)(\delta + \eta)}}$$

$$\therefore S^0 = \frac{(1 - \varphi)k(\sigma + \mu) + \varphi \sigma k}{(\sigma + \mu)\mu}$$

$$R_z = \frac{\omega \epsilon [(\mu + \sigma)(1 - \varphi)k + \sigma \varphi k]}{\mu(\mu + \sigma)(\gamma + \mu)(\delta + \eta)}$$

2.9 Examples

Here is the graphical representation of above calculated study state solutions of the model.

Example1: For $\omega = 0.011, \epsilon = 0.045, \mu = 0.003, \sigma = 0.02, \varphi = 0.08, k = 1.5, \gamma = 0.8, \delta = 0.07, \eta = 0.3$, then ($R_z = 0.8 < 1$), hence the T. gondii-free equilibrium (W_0) is locally asymptotically stable where initial conditions are $V(0) = 200, S(0) = 500, I(0) = 55, M(0) = 150, E(0) = 250$.

Example 2: For $\omega = 0.019, \epsilon = 0.055, \mu = 0.003, \sigma = 0.02, \varphi = 0.079, K = 1.5, \gamma = \delta = 0.07, \eta = 0.29$ then ($R_z = 1.8 > 1$), hence the T. gondii-endemic equilibrium (W^*) is locally stable where initial conditions are $V(0) = 200, S(0) = 600, I(0) = 15, M(0) = 150, E(0) = 400$. Results are shown in figures (2,3).

3. Numerical Analysis

In previous sections we calculated the equilibrium points and check their stability. We checked the behavior of R_z by simulation by using built in function in Matlab. Sometimes when we change the initial conditions or change the parameters then the given built in tools give the false results in form of unboundedness or divergence or in other ways. In this case we need numerical techniques for reliable results. After finding the study state solutions and plotting their results, this chapter will give us the numerical analysis of toxoplasma transmission and effect of each parameters which we have used, on state variables and basic reproductive number R_z . This will give us which parameter is more sensitive in spreading the disease. As some techniques or tools are not reliable for nonlinear model because these model have some assumptions like sum of all population should be positive, population should be bounded and dynamical system should be consistence. Also non-linear models do not give exact solutions always. In this case we need numerical technique in which we see the behavior of system numerically. Researchers have made many numerical techniques like Euler, Non Standard Finite Difference (NSFD), RK4, Back Difference Formulae (BDF), Implicit-Explicit (IMEX),

Stochastic Simulation Algorithms etc. [13]. For our numerical calculations we will use the three schemes which will be Euler, RK4 and NSFD. Using these techniques, we will plot the all our required graphs which will tell us the specific information about disease behavior viewing each state variables and each parameter.

3.1 Euler Scheme

Leonhard Euler was the first mathematician who developed the Euler Scheme between the era of 1768 to 1770. Basically this scheme was developed for solving ordinary differential equations. In case of disease dynamics, this is the fundamental and simplest numerical scheme which tells us about spread of diseases and their interaction with population [14]. In numerical method we make the iterative system and chose the step sizes. Euler scheme shows us negative results when we increase the step sizes and it is biologically unrealistic. It has stability and accuracy problems. This shows that this scheme is not reliable for numerical method. The model of this project is given as:

Conversion of Model Using Euler Method

$$\begin{aligned} V^{n+1} &= h[\varphi k - (\sigma + \mu)V^n] + V^n, & S^{n+1} &= h[(1 - \varphi)k + \sigma V^n - \omega E^n - \mu S^n] + S^n \\ I^{n+1} &= h[\omega E^n S^n - (\gamma + \mu)I^n] + I^n, & M^{n+1} &= h[\gamma I^n - \mu M^n] + M^n \\ E^{n+1} &= h[\epsilon I^n - (\delta + \eta)E^n] + E^n \end{aligned}$$

Results are illustrated in figures (4,5).

3.2 Runge-Kutta RK_4 Method

After successfully observing results from Euler which is numerical techniques, we move towards the other numerical technique which is Runge-Kutta RK_4 . This fourth-order method was developed by the German mathematicians Carl Runge and Wilhelm Kutta about 1900. This technique was developed to approximate solutions to ordinary differential equations and are family of iterative techniques [15]. Given method is the extension of Euler method and initially introduced by Carl Runge in 1895. In this section we will see all the results using RK_4 technique and will compare with all the results from previous technique and will see that which technique is more reliable and authentic for given mathematical model. We have seen from the results (6,7) that at some specific step sizes RK_4 scheme is convergent otherwise divergent. So, we can say that it is conditionally convergent which behaves like Euler.

3.3 NSFD Scheme

The advance scheme NSFD, which was firstly designed by Ronald E. Mickens in the last decade of 20th century, is the best technique which gives the result in better way no matter what are the step sizes. It maintains the boundedness of solution and also maintain the positivity of population at any step size. Also, the model remains consistence and is conditionally convergent. Due to having these properties this scheme is superior on Euler scheme and others [16].

Conversion of Model Using NSFD Method

We convert our differential equations of given model into NSFD using the numerical method NSFD.

$$V^{n+1} = \frac{V^n + h\varphi k}{1 + h(\sigma + \mu)}, \quad S^{n+1} = \frac{S^n + h(1 - \varphi)k + h\varphi V^n}{1 + h\omega E^n + h\mu}$$

$$I^{n+1} = \frac{I^n + h\omega E^n S^n}{1+h(\gamma+\mu)} \quad , \quad M^{n+1} = \frac{M^n + h\gamma I^n}{1+h\mu} \quad , \quad E^{n+1} = \frac{E^n + h\epsilon I^n}{1+h(\delta+\eta)}$$

Results are shown in figures (8,9).

3.4 Comparison between Euler, RK_4 & NSFD

Here are the comparisons between Euler, RK_4 and NSFD schemes presented through individual graphs for a specific step size. These comparisons highlight the qualitative behaviour of each numerical scheme in modelling the dynamics of the population. In particular, one method preserves the positivity of the population over time (a biologically essential property), while the other fails to do so, resulting in negative values which are unrealistic in epidemiological modelling. RK_4 and Euler have the same behaviour as both schemes are conditional convergent. At small steps these show convergence but when we increase the step size a bit these become divergent and show unboundedness. This significant contrast emphasizes the superiority of the positivity-preserving method. The outcomes of these comparisons are illustrated in Figures, showcasing how each compartment such as vaccinated, susceptible, infected, and environmental is influenced under three schemes. Results are shown in figures (10,13).

4. Impact of Parameters on State Variables

We used different parameters for different four step sizes in our model which represent different scenarios according to state variables. In these graphs (14,17) we have seen the parameters which have greater impact on given five state variables. We can see that by increasing step size some results are increasing and some decreasing.

4.1 Impact of ϕ on R_z

The basic motive of our model is to keep the value of basic reproductive number $R_z < 1$ so that our infected population becomes minimum or becomes zero. This will happen by adjusting the values of parameters. Here we see the impact of ϕ on $R_z < 1$ which show that when we increase the value of ϕ our $R_z < 1$ decrease and eventually approaches to zero which will show that disease will completely die out from population shown in figure (18).

5. Sensitivity Analysis of R_z

Basic Reproductive Number plays an important role in infectious diseases, telling us that either the disease will die completely, persist, or spread in the population depends on its values (Nakul Chitnis, 22 February 2008). Here we will check the impact of each parameter on the R_z and will see that which parameter is more sensitive to R_z . All the parameters and their values are given as For $\omega = 0.019$, $\epsilon = 0.055$, $\mu = 0.003$, $\sigma = 0.02$, $\phi = 0.079$, $K = 1.5$, $\gamma = \delta = 0.07$, $\eta = 0.29$ then ($R_z = 1.8 > 1$).

Symbols	Formulas	Parameters Description's	Sensitive Index
μ	$A_{\mu}^{R_z} = \frac{\partial R_z}{\partial \mu} * \frac{\mu}{R_z}$	Natural death rate	≈ -1.01
k	$A_k^{R_z} = \frac{\partial R_z}{\partial k} * \frac{k}{R_z}$	Recruitment rate by birth and immigration	$= 1$
ω	$A_{\omega}^{R_z} = \frac{\partial R_z}{\partial \omega} * \frac{\omega}{R_z}$	Effective contact rate	$= 1$
φ	$A_{\varphi}^{R_z} = \frac{\partial R_z}{\partial \varphi} * \frac{\varphi}{R_z}$	Fraction of cats vaccinated at birth	≈ -0.0104
γ	$A_{\gamma}^{R_z} = \frac{\partial R_z}{\partial \gamma} * \frac{\gamma}{R_z}$	Termination rate of shedding of oocysts	≈ -0.995
σ	$A_{\sigma}^{R_z} = \frac{\partial R_z}{\partial \sigma} * \frac{\sigma}{R_z}$	Loss rate of immunity	≈ 0.0076
ϵ	$A_{\epsilon}^{R_z} = \frac{\partial R_z}{\partial \epsilon} * \frac{\epsilon}{R_z}$	Shedding rate of oocysts	$= 1$
δ	$A_{\delta}^{R_z} = \frac{\partial R_z}{\partial \delta} * \frac{\delta}{R_z}$	Removal rate of oocysts (non-sanitation)	≈ -0.194
η	$A_{\eta}^{R_z} = \frac{\partial R_z}{\partial \eta} * \frac{\eta}{R_z}$	Removal rate of oocysts (due to sanitation)	≈ -0.805

From the above study we observe that the basic reproduction number R_z is very sensitive to changes in Recruitment rate by birth and immigration k , Effective contact rate ω and Shedding rate of oocysts ϵ . If these increase R_z will also increase in the same way and if these decrease R_z will also decrease because R_z and these are directly proportional to R_z . All the other parameters are inversely proportional to R_z . We can say that our basic reproductive number is more sensitive to ω and σ as if we decrease, the contact rate of our R_z decreases and we can approach disease free equilibrium case. The same happens by decreasing the shedding rate of oocysts R_z to less than zero. It is clear from the results that when infected cats decrease the shedding oocysts in the environment, then automatically the environment becomes less contaminated. Same, the rate of contact from susceptible to infected cats and contaminated environment decreases. Thus, these two parameters are more effective in controlling the disease.

6. Graphical Representation

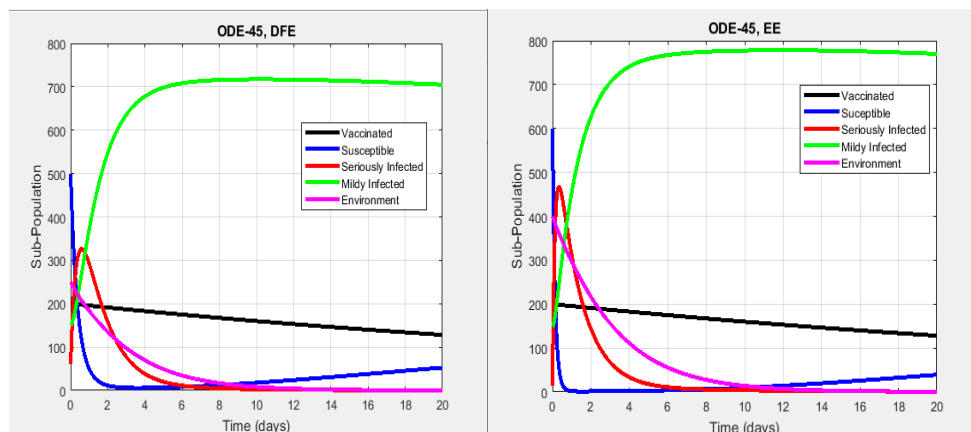


Fig. 2 and Fig. 3. Results from ODE-45

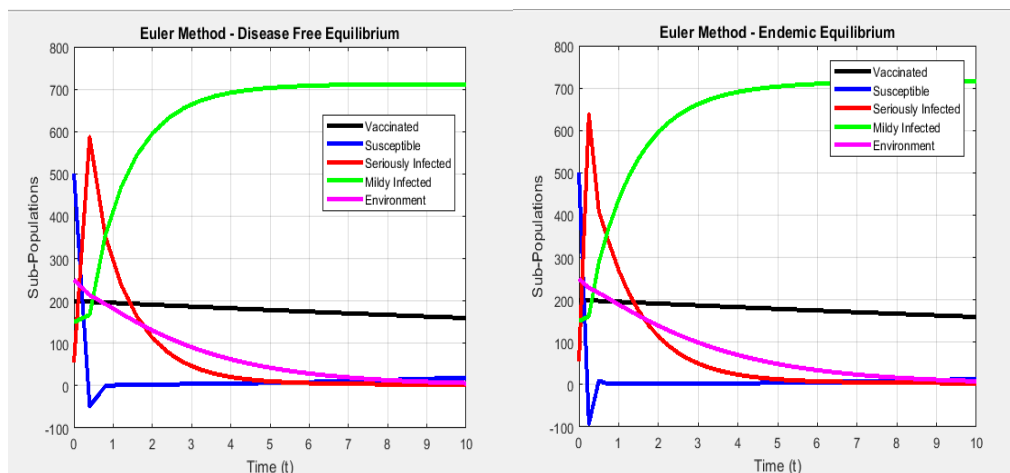


Fig. 4 and Fig. 5. Results using Euler Scheme

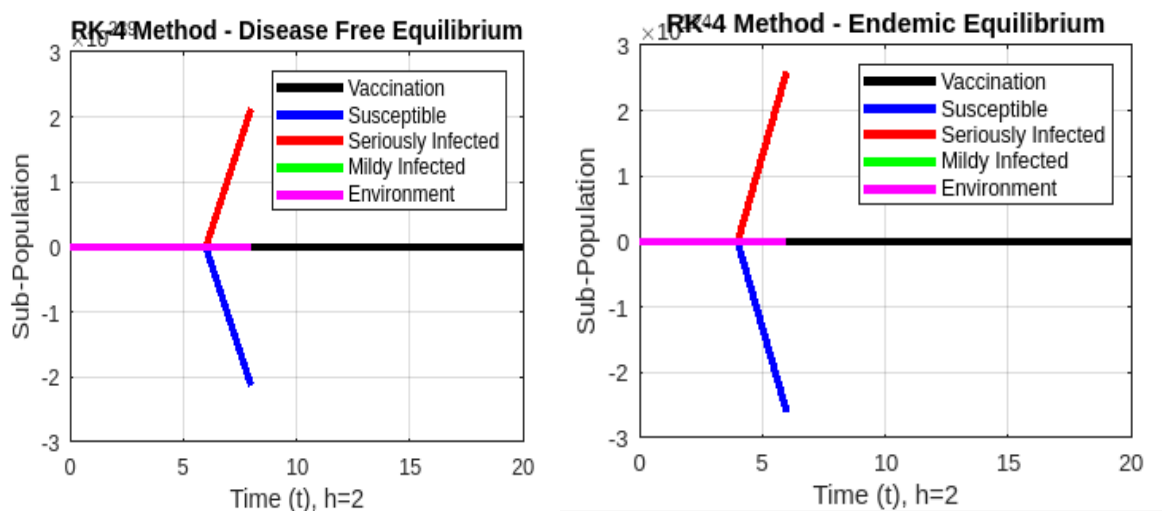


Fig. 6 and Fig. 7. Results using RK_4 scheme

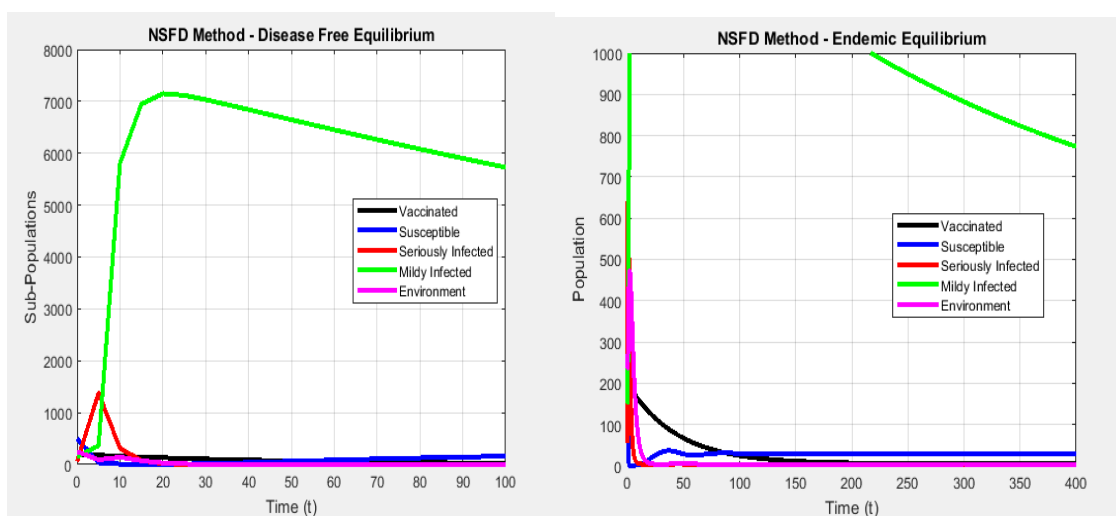


Fig. 8 and Fig. 9. Results using NSFD scheme

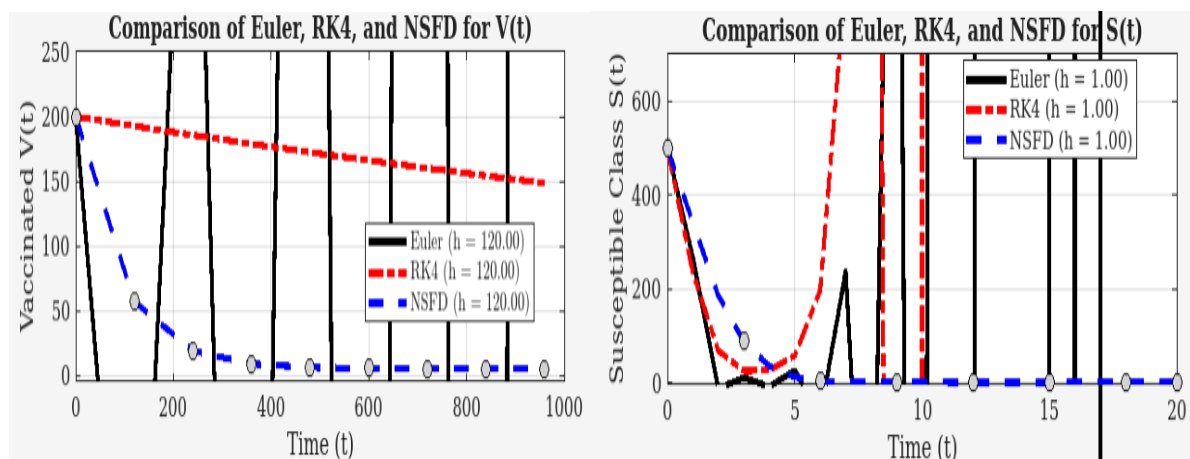


Fig. 10 and Fig. 11. Results from comparisons

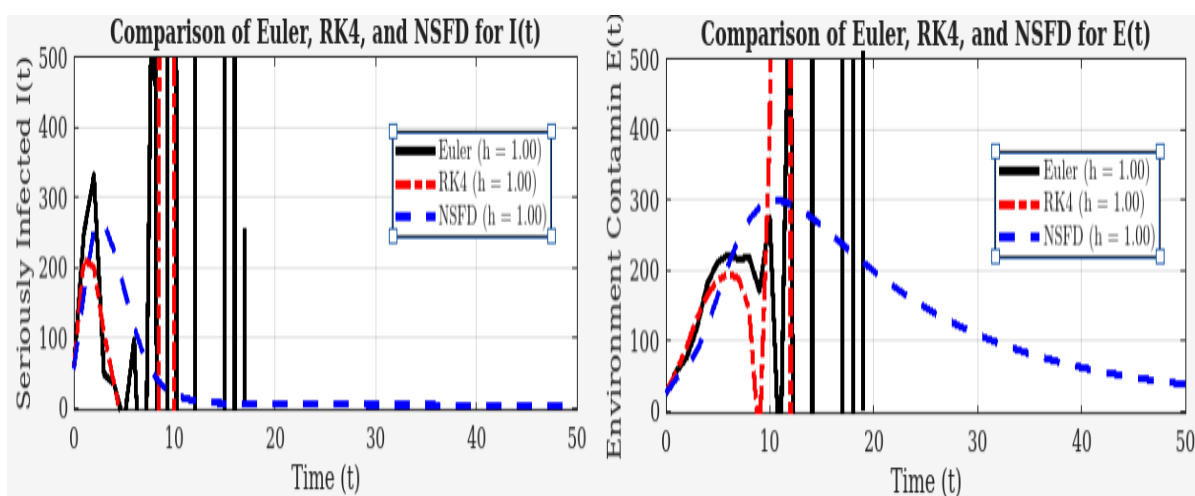


Fig. 12 and Fig. 13. Results from comparisons

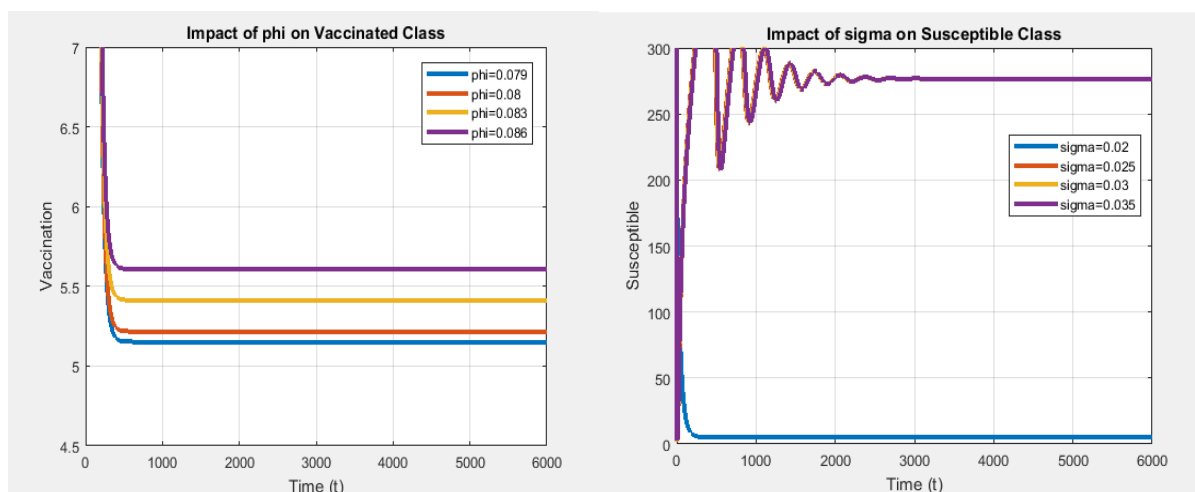


Fig. 14 and Fig. 15. Impact of parameters on S.V

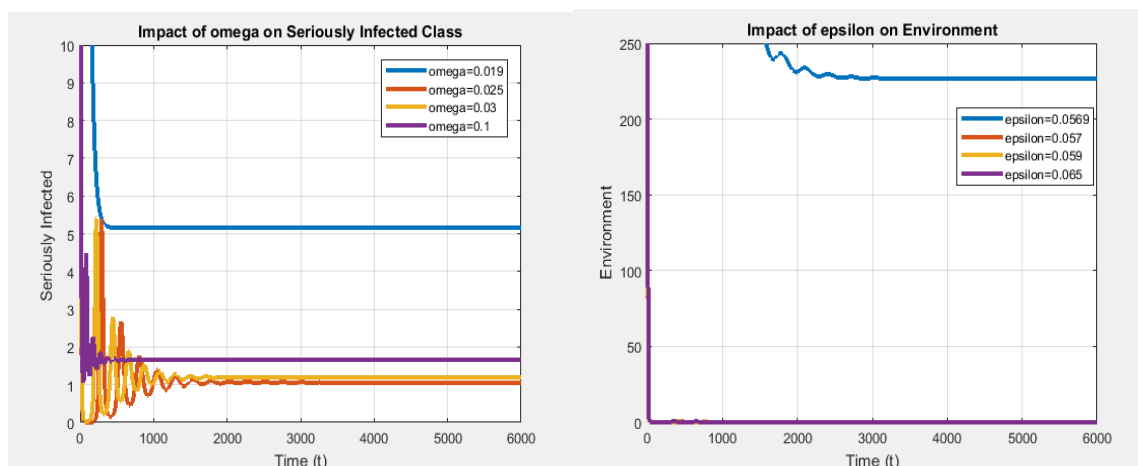


Fig. 16 and Fig. 17. Impact of parameters on S.V

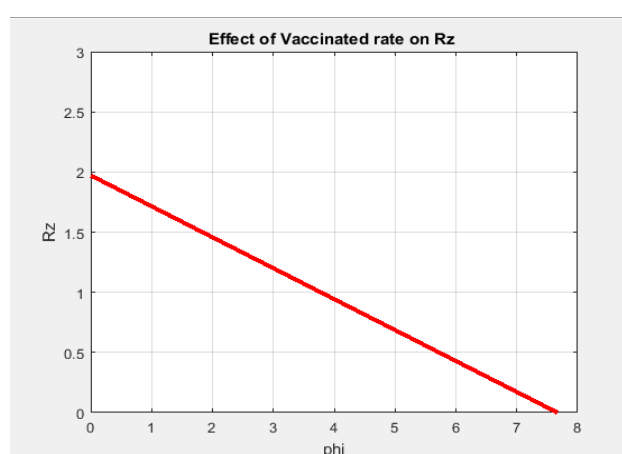


Fig. 8. Impact of φ on R_z

7. Conclusion

In recent years, the world has witnessed the devastating impacts of infectious diseases, both pandemic (like COVID-19) and endemic (such as malaria and HIV). These diseases not only challenge public health systems but also affect global economies and social structures. Introducing "Vaccination" in our model, we examine how these this effect the proportion of *T. gondii* in cat population. We deeply analyze the model to validate our model and calculate the basic reproductive number R_z which tells us about disease. We examined the conditions to exist the global stability for disease free equilibrium and also for endemic equilibrium by calculating the threshold quantity R_z . The result shows the effective contact rate ω , shedding rate of oocysts ϵ , recruitment rate of cats k , removal rate of oocysts due to sanitation η and vaccination rate φ as most sensitive parameters to R_z . We performed simulations based on theoretical results to analyze the effect of these parameters on spread and managing *T.gondii*. After successfully calculating the results from study state solution of the model, we checked the numerical techniques (Euler, RK4 & NSFD) from which we saw all the results of Euler, RK4 and NSFD schemes. We saw that for long-term behavior of disease, Euler and RK4 schemes have shown negative results and are inconsistent dynamically from which we say that these are not reliable schemes. On the other hand, we also plotted the results of NSFD scheme, from which we saw that no matter what the step size is, this scheme gives the reliable results and keeps the population positive. From given experiment NSFD scheme is a reliable tool from which we can

check our long-term behavior of disease. We checked the impact of parameters on each state variable from which we saw that which parameter is more effecting the specific variables. Basic Reproductive Number R_z which plays a crucial role in this model for elimination of disease which depends on its value by using the parameters. φ is the vaccination rate in cat population and from the results, we have seen that it has a great impact on R_z . By increasing its value, R_z decreased, on the other hand infected population also decreased. It was the basic motive of our project that by increasing the vaccination rate in the population, we can control the disease. At the last, we saw the sensitivity of parameters on the R_z which showed that contact rate and shedding rate of oocysts are more sensitive to R_z . Their increment or decrement have the direct impact on the R_z . The results say that safe environment form oocysts is the best control strategy which can help in controlling the disease and may be helpful for the healthcare centers in the world.

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